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VOLUME 20
1938

PUBLISHERS
AMERICAN MEDICAL ASSOCIATION
CHICAGO, ILL.

CONTENTS OF VOLUME 20

JULY 1938 NUMBER 1

	PAGE
Cataract Operations in the Prehistoric Age Kartic Chunder Dutt, L M S, Calcutta, India	1
Problem of Rickettsias in Trachoma Phillips Thygeson, M D, New York	16
Conjunctivitis Associated with Infection by Streptococcus Viridans Clinical and Bacteriologic Observations in an Epidemic Adolph R Berger, M D, David H Goldstein, M D, Currier McEwen, M D, and Rose C Alexander, M S, New York	19
Influence of Vitamins and Dinitrophenol on the Production of Experimental Cataract M L Tainter, M D, and W E Borley, M D, San Francisco	30
Absorption of Visible Light by the Refractive Media of the Human Eye Elek Ludvig, Ph D, and E F McCarthy, A M, Boston	37
Association of an Annular Band of Pigment on Posterior Capsule of Lens with a Krukenberg Spindle William Zentmayer, M D, Philadelphia	52
Testing Fitness for Night Flying Visual Acuity C E Ferree, Ph D, and G Rand, Ph D, Baltimore	58
Biochemistry of the Lens XI Effect of Galactose on Permeability of the Capsule of the Lens John Bellows, M D, and Lawrence Rosner, Ph D, Chicago	80
Clinical Notes	
Dacryocystitis in Lymphatic Leukemia William H Stokes, M D, Omaha	85
Oculoglandular Tularemia Irving I Cramer, M D, Cleveland	88
Accidental Vaccination of the Conjunctiva F Necdet Sezer, M D, Malatya, Turkey	89
A Modified Corneal Clamp to Facilitate the Insertion of Stitches T L Terry, M D, Boston	91
Ophthalmologic Review	
Vertical Prism Values in Commonly Used Bifocal Lenses Sidney L Olsho, M D, Philadelphia	95
Correspondence	
Ocular Dominance Lloyd Mills, M D, Los Angeles	105
Surgical Treatment of Strabismus Otis Wolfe, M D, Marshalltown, Iowa	105
Abstracts from Current Literature	107
Society Transactions	
French Ophthalmologic Society	122
International Congress of Ophthalmology	135
New York Academy of Medicine, Section of Ophthalmology	148
College of Physicians of Philadelphia, Section on Ophthalmology	152
Book Reviews	159
Directory of Ophthalmologic Societies	165

AUGUST 1938 NUMBER 2

Disturbances of the Vertical Motor Muscles of the Eyes A Bielschowsky, M D, Hanover, N H	175
Perimetric Studies in Syphilitic Optic Neuropathies Louise L Sloan, Ph D, and Alan C Woods, M D, Baltimore	201
Cancer of the Eyelids, Conjunctiva and Cornea II Squamous Cell Epithelioma Henry L Birge, M D, Rochester, Minn	254
Treatment of Staphylococcal Conjunctivitis with Staphylococcus Toxoid Preliminary Note Phillips Thygeson, M D, New York	271
Mannitol Fermentation as an Indicator of Conjunctival Pathogenicity of Staphylococci Phillips Thygeson, M D, New York	274
Recurrent Retinal and Vitreous Hemorrhages in the Young—Eales' Disease Report of Two Cases R Townley Paton, M D, New York	276

CONTENTS OF VOLUME 20

AUGUST—Continued

	PAGE
An Ophthalmic Carriage John N Evans, M D, Brooklyn	286
Topography and Frequency of Complications of Uveal Sarcoma Bernard Kronenberg, M D, New York	290
Inositol in the Ocular Tissues Arlington C Krause, M D, Ph D, and Roger Weckers, M D, Chicago	299
Clinical Notes	
A Specific Treatment for Herpes Zoster J R Walker, M D, and B F Walker, M D, Fresno, Calif	304
Ophthalmologic Review	
Detachment of the Retina Summary of the Modern Opinions Thomas D Allen, M D, Chicago	307
Correspondence	
A New Method for Rebuilding a Lower Lid Ferris Smith, M D, Grand Rapids, Mich, and Wendell L Hughes, M D, Hempstead, L I	315
Simplification of the O'Connor Cinch Operation Roderic O'Connor, M D, San Francisco	315
News and Notes	317
Obituaries	
James A Spalding, M D	318
Abstracts from Current Literature	319
Society Transactions	
College of Physicians of Philadelphia, Section on Ophthalmology	334
New York Academy of Medicine, Section of Ophthalmology	337
Book Reviews	344
Directory of Ophthalmologic Societies	349

SEPTEMBER 1938 NUMBER 3

Intraocular Tension in Cases of Sarcoma of the Choroid and Ciliary Body John H Dunnington, M D, New York	359
Clinical Significance of the Retinal Changes in Leukemia Glen G Gibson, M D, Philadelphia	364
Bilateral Congenital Ectopia Lentis with Arachnodactyly (Marfan's Syndrome) Joseph Laval, M D, New York	371
Nature and Management of the Heterophorias J T Maxwell, M D, Omaha	375
Optochiasmic Arachnoiditis Importance of a Mixed Type of Atrophy of the Optic Nerve as a Diagnostic Sign Derrick Vail, M D, Cincinnati	384
Research Studies of the Eye Some Technical and Practical Notes Archimede Busacca, M D, São Paulo, Brazil	395
Advantages of Use of Coagulants in Ocular Operations, Especially in Extraction of Cataract and in Plastic Operations Archimede Busacca, M D, São Paulo, Brazil	406
Rotation of the Cheek in Ophthalmology J F S Esser, M D, Monaco, France	410
Biochemistry of the Lens XII Studies on Glutathione in the Crystalline Lens Lawrence Rosner, C J Farmer and John Bellows, Chicago	417
Retinitis Proliferans Clinical and Histologic Studies Bertha A Khen, M D, Chicago	427
Studies of the Visual Fields in Cases of Verified Tumor of the Brain Daniel Kravitz, M D, Brooklyn	437
Clinical Notes	
Retinitis Pigmentosa with "Hole" in the Macula Report of a Case Charles A Perera, M D, New York	471
Hypersensitivity to Larocaine Frederick H Theodore, M D, New York	474
Ophthalmologic Review	
Binocular Vision and Orthoptic Procedure Dorothy J Shaad, Ph D, New York	477
Correspondence	
Simplification of the O'Connor Cinch Operation M E Smukler, M D, Philadelphia	502
A New Model Gonioscope John M McLean, M D, Baltimore	502
News and Notes	504
Abstracts from Current Literature	506

CONTENTS OF VOLUME 20

SEPTEMBER—Continued

Society Transactions	PAGE
New York Academy of Medicine, Section of Ophthalmology	520
Book Reviews	528
Directory of Ophthalmologic Societies	531

OCTOBER 1938 NUMBER 4

	PAGE
Physiologic and Clinical Ophthalmologic Problems in Relation to Individual Variability (To Be Continued) A Bruckner, M D, Basel, Switzerland	541
Nature of the Filtrable Agent of Trachoma Phillips Thygeson, M D, New York, and Polk Richards, M D, Albuquerque, N Mex	569
An Evaluation of Homatropine-Benzedrine Cycloplegia Herbert F Sudranski, M D, Indianapolis	585
A Cataract Operation to Reduce the Incidence of Prolapse of the Iris Frank C Parker, M D, Norristown, Pa	597
Induced Size Effect I A New Phenomenon in Binocular Space Perception Associated with the Relative Sizes of the Images of the Two Eyes Kenneth N Ogle, Ph D, Hanover, N H	604
Experimental Iontophoresis of Rabbits' Corneas Report of Two Cases of Corneal Dystrophy with Treatment by Ionic Medication Stephen G Seech, M D, and William LeGrande Cooper, M D, Los Angeles	624
Inferior Iridotomy in Operations for Cataract on Eyes with Posterior Synechiae or Pupillary Membrane Value of Operation Paul A Chandler, M D, Boston	641
Results of Autotransplantation of Cornea into Anterior Chamber Their Significance Regarding Corneal Nutrition Trygve Gundersen, M D, Boston	645
An Intern's Experiences with the Verhoeff Method of Cataract Extraction Paul H Case, M D, Phoenix, Ariz	651
Clinical Notes	
A Cobalt Blue Filter for Observation of the Fit of Contact Lenses Theodore E Obrig, A B, New York	657
Blepharitis Comb James W Smith, M D, New York	658
Abstracts from Current Literature	659
Society Transactions	
New York Academy of Medicine, Section of Ophthalmology	680
Book Reviews	694
News and Notes	698
Directory of Ophthalmologic Societies	699

NOVEMBER 1938 NUMBER 5

Implantation of Hollow Grooved Body into Orbit for Filling Late After Enucleation of Eyeball John M Wheeler, M D, New York	709
Determination and Significance of the Scotopic Retinal Visibility Curve Elek Ludvigh, Ph D, Boston	713
Physiologic and Clinical Ophthalmologic Problems in Relation to Individual Variability (To Be Continued) A Bruckner, M D, Basel, Switzerland	726
Congenital Buphthalmos Complicated by Dislocation of Lens and Hemorrhage into Vitreous with Complete Recovery of Central Vision William John Holmes, M D, Warren, Ohio	757
Circulation of the Aqueous VII A Mechanism of Secretion of the Intraocular Fluid Jonas S Friedenwald, M D, and Robert D Stiehler, Ph D, Baltimore	761
Coloboma of the Optic Nerve and of the Macula A Microscopic Study David Wexler, M D, and Murray Last, M D, New York	787
Acute Alcoholic Amaurosis Frank D Carroll, M D, and Robert Goodhart, M D, New York	797
Panophthalmitis and Sympathetic Ophthalmia Bernard Samuels, M D, New York	804
Experimental Hypertension VIII Vascular Changes in the Eyes John E L Keyes, M D, and Harry Goldblatt, M D, C M, Cleveland	812

CONTENTS OF VOLUME 20

NOVEMBER—*Continued*

	PAGE
Reading Difficulties in Children George E Berner, M D, and Dorothy E Berner, B S (Educ), Philadelphia	829
Ophthalmologic Review	
Carbon Disulfide Poisoning Robb McDonald, M D, Philadelphia	839
Clinical Notes	
Homonymous Hemianopic Paracentral Scotoma Report of a Case T D Allen, M D, and H F Carman Jr, M D, Chicago	846
Abstracts from Current Literature	850
Society Transactions	
American Medical Association, Section on Ophthalmology	867
News and Notes	883
Obituaries	
John Martin Wheeler, M D	885
Book Reviews	892
Directory of Ophthalmologic Societies	897

DECEMBER 1938 NUMBER 6

Aniseikonia Walter B Lancaster, M D, Boston	907
Physiologic and Clinical Ophthalmologic Problems in Relation to Individual Variability (Concluded) A Bruckner, M D, Basel, Switzerland	913
Simple Dacryocystorhinostomy Loren Guy, M D, New York	954
Angiomatosis Retinae Report of a Case with a Pathologic Study of the Eucleated Eye Robb McDonald, M D, and Stuart W Lippincott, M D, Philadelphia	958
Lesion of the Optic Tract Probably the Result of Infected Sphenoid Sinuses Albert N Lemoine, M D, Kansas City, Mo	966
Events of Vascularization and Devascularization Seen in Corneas P F Swindle, Ph D, Milwaukee	974
Orbital Hyperostosis Its Occurrence in Two Cases of Meningioma of the Skull Arnold Knapp, M D, New York	996
Intraocular Nematode Worms Report of a Case and Review of the Literature Lester T Jones, M D, Lewis W Jordan, M D, and Nicholas P Sullivan, M S, Portland, Ore	1006
Scleromalacia Perforans Report of a Case in Which the Eye Was Examined Microscopically F H Verhoeff, M D, and Merrill J King, M D, Boston	1013
Use of Sorbitol in Glaucoma John Bellows, M D, Irving Punttenney, M D, and Jack Cowen, M D, Chicago	1036
Clinical Notes	
A New Refractor Suspension S Maisler, M D, San Francisco	1044
A New Capsule-Grasping Forceps M M Cullom, M D, Nashville, Tenn	1045
Ophthalmologic Review	
Etiology of Retinal Separation Considered from the Standpoint of Surgical Correction Edmund B Spaeth, M D, Philadelphia	1046
News and Notes	1071
Obituaries	
George Edmund de Schweinitz, M D	1074
Ladislav de Blaskovics	1081
Abstracts from Current Literature	1082
Society Transactions	
American Ophthalmological Society	1099
Book Reviews	1113
Directory of Ophthalmologic Societies	1119
General Index	1129

ARCHIVES OF OPHTHALMOLOGY

VOLUME 20 (old series Volume 77) JULY 1938

NUMBER 1

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CATARACT OPERATIONS IN THE PREHISTORIC AGE

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The early history of operations for cataract is shrouded in mystery. Many an ophthalmologist has attempted to trace it, but so far the subject has remained unsolved and the account incomplete and unconnected. Consequently, the chapter on the history of operations for cataract is uninteresting and unconvincing. Nor has the date of the inauguration of such surgical procedures been traced much earlier than the era of Christ. Permit me, then, to unveil the records of the prehistoric age.

Before 1745 A D, when Daviel devised the capsulotomy method of cataract extraction, couching was practiced throughout Europe and Asia. Indeed, the history of operations for cataract before 1745 A D is equivalent to the history of couching.

Col Henry Smith, in his famous book entitled "The Treatment of Cataract,"¹ began the chapter on couching with admirable insight. His views are worth quoting. Smith wrote

Lens couching has been practiced in the East from time immemorial. It is the parent of all modern operations on cataract, and, alone deserves the name "classical." History no more relates its origin than that of the practice which was the parent of all modern vaccination, the inoculation of a healthy individual with the juice from the pustule of a smallpox patient, with the object of provoking a mild attack of the disease, which would give subsequent immunity to it. Both were important observations in their time. Both were given by the East to the West.

But is it really true that couching had its origin in the East? If so, in what country of the East? If history does not relate its origin, how near to its origin does it come? Is there any authentic record of its origin or of what may be reasonably taken as such? Yes.

The first authentic record of an operation for cataract can be traced to about 1000 B C. This record has been handed down in complete

¹ Smith, H. Treatment of Cataract, ed 2, Calcutta Butterworth & Co., Ltd, 1928.

form in "Susruta Sanhita,"² which is a treatise on surgery, written by Susruta, a great surgeon of ancient India. It has been ascertained that Susruta practiced and taught his method for the surgical treatment of cataract sometime between 750 and 1500 B. C. As there is a difference of opinion as to the exact date that Susruta first performed his operation, I may be excused if I agree with those historians who put it about 1000 B. C. In chapter 17 of volume 7 of "Susruta Sanhita," Susruta beautifully described the varieties of cataract and the technic of the depression method of couching by the anterior route. Susruta's description of the postoperative treatment is admirable. I have no doubt that my translation of this description will be read with pleasure by modern ophthalmologists.

The next question that will naturally be asked is "Why has 'Susruta Sanhita' so long remained a sealed book to modern ophthalmologists and research workers?" The reason is that it is written in Sanskrit, a difficult classical language to study and a more difficult language to translate properly.

Another question that arises is whether Susruta was the originator of couching. In the absence of any record earlier than "Susruta Sanhita," I should call Susruta "The Father of Cataract Operations." It is possible that he had forefathers at the dawn of civilization in Hindustan, but I have not yet discovered any authentic record of them.

I shall present an English translation, rendered by myself, from "Susruta Sanhita," of the description of Susruta's original operation, the depression method of couching by the anterior route. I hope that it will be of interest not only to antiquarians and research workers but to all inquisitive ophthalmologists. Susruta's original writings are not to be relegated to the curios of by-gone ages, because they are instructive in more ways than one.

Indeed, it will be found that Susruta was a potential force in the development of operation for cataract and that even to this day surgeons are subconsciously influenced by him. It will be found that many a Susrutian custom which is unwittingly followed today, but which has lost its significance, should be discontinued, on the other hand, many a time-honored precaution of Susruta which is neglected may be profitably followed even now.

It is well known that in the preanesthetic and preantiseptic days open operations for cataract such as are practiced at present were followed by disastrous results. Hence couching was the only method practicable and in the Susrutian age it gave the best results. One should never look down on the great ophthalmic surgeon who was the first to institute surgical treatment for cataract as a coucher. Indeed, it will be evident from his writings that he was fully aware of the after-

² Susruta Sanhita (dated 1000 B. C.), vol 7, chap 17, verses 57 to 73

effects and shortcomings of couching, but he was handicapped for want of anesthetics and antiseptics. Nor can the results of Suśruta's couching operation be evaluated by the results of couching at the hands of the quack couchers of Europe, known as charlatans, and those of India, known as jowals.

Would it be justifiable to look down on Smith or on Barraquer or on Knapp because of the bad results obtained at the hands of quacks with Smith's pressure method or with Barraquer's suction method or with Knapp's method *i. e.*, traction by forceps combined with the pressure method? Can the quacks succeed any better with any of these methods than with Suśruta's method? Certainly not. Would any one expect a quack to score a percentage of success with any of these methods that approaches the percentage of success attained by the originator with his own method? Certainly not. The results of operation by Smith's method in the hands of Smith, of Barraquer's method in the hands of Barraquer or of Knapp's method in the hands of Knapp are admirable indeed. To condemn these pioneers on the basis of bad results with their operative technics in the hands of a quack is a proposition which is self-condemned. Indeed, those who invent new methods are geniuses and great masters of their art. Similarly, Suśruta was a surgical genius and a great master who initiated and taught an operation for cataract at the dawn of civilization. His abilities must not be gaged by the abilities of quacks. That would be most unjustifiable, would it not?

One should, then, judge Suśruta's observations on cataract, his operation and his postoperative treatment with an open mind and with judiciousness necessary to arrive at an unbiased judgment. It will be found that Suśruta's observations will stand the acid test of modern science to the very letter. If that is so, then one should give to Caesar what is Caesar's.

Suśruta began his chapter on cataract with the cause and varieties of cataract. He advocated no medicinal treatment. His treatment consisted in couching the cataractous lens when it was mature. Unripe cataracts, according to him, are unfit for couching. Suśruta described the technic of couching in detail and formulated the routine postoperative treatment. He concluded by noting the contraindications for couching.

The cause of cataract formation, according to Suśruta, is opacity of the lens, consequent on the derangement of intraocular lymph.

Suśruta divided cataracts into several varieties. He wrote

The cataract may be semilunar or crescent shaped. It may look like a drop of sweat or muddy water through the pupil.

The cataract may be stationary. It may be hard and shaped like the "masur" lentil. [*Ervum lens* (Linn) *leguminosæ* is a biconvex lens-shaped lentil of the size of a mature hard cataract. It is reddish and has a capsule-like epicarp.] It may

also be irregular in shape. The opacity may be centrally located, or it may be at the cortex, with a clear center.

The cataract may be of various colors. It may be white and pearl shaped and shining or glistening.

The cataract may be of a greenish or bluish color or of a reddish or brownish color.

The technic of Susruta's depression method of couching by the anterior route, translated by me from the original Sanskrit,³ follows.

The operation of couching should be performed in a season which is neither very hot nor very cold.

On the days previous to the operation, the patient should take nonstimulating food and drink. On the day of the operation he should have a cool temper and an undisturbed mind.

The patient should sit on an especially constructed chair with his head and limbs fixed by a special contrivance. He should look equably at his nose, without moving the eye in any other direction, and continue to look uniformly in the same way during the operation.

The surgeon should open the patient's eye completely and keep the lids in the same condition during the operation. He should select the point of puncture just outside the junction of the black and white outlines of the eye. The point should be in the line dividing the white portion equally into an upper and a lower segment. It should be a little on the temporal side but not much outside the junction of the black and white outlines. The point of puncture should not be over the plexus of blood vessels. The Jabamukhi Salaka [literally, the curved barley-tipped needle], *i. e.*, a curved needle, the tip of which is shaped like a barley corn tapering in a point, is the only instrument required for the operation [Mookerji, diagram of Jabamukhi Salaka⁴].

The Jabamukhi Salaka, *i. e.*, the curved barley-tipped needle, should be held between the thumb and index and middle fingers of the surgeon. He should hold the curved barley-tipped needle in his right hand if the left eye is to be operated on and in his left hand if the right eye is to be operated on.

The needle should be held carefully, but confidently, with the fingers contracted and steady. The puncture should be made in the right place, already indicated, and in a horizontal direction. The hand should be steadied by placing it in a position of rest while the needle penetrates the coats of the eye. As soon as the puncture is made, there is a sound. A drop of aqueous humor may come through the point of the puncture. After the puncture, the milk of a nursing mother should be sprinkled into the conjunctival sac.

The action of mother's milk has been fully discussed in an article entitled "Melaena Neonatorum"⁵. It points out that mother's milk is an excellent hemostatic in cases of this condition. In vitro, the acceleration of the coagulation time from five to ten minutes to from one-half to one minute can be easily demonstrated. In vivo, the clotting of blood occurs by direct contact of the coagulating substances present in the

³ Susruta Samhita,² verses 59 to 73.

⁴ Mookerji, G. Surgical Instruments of the Hindus, Calcutta, Calcutta University, 1914.

⁵ Melaena neonatorum, Klin Wchnschr 1936.

cream of mother's milk with the bleeding tissue Solo has arrested hemorrhage following the extraction of teeth and in persons with hemophilia and with thrombopemia by the application of pieces of gauze soaked in mother's milk

Melena neonatorum has been successfully arrested by feeding the newborn baby with the milk of some other woman than its mother who has been nursing a child for more than a fortnight The hemostatic properties of the milk of the baby's own mother do not develop until after the first fortnight of suckling⁶

Today epinephrine is used for cataract operations in place of the mother's milk of the pre-epinephrine days

Susruta continued

The Jabamukhi Salaka i.e. the curved barley-tipped needle, should then be carefully steered into the natural aperture of the pupil, neither upward, downward nor sideways The point of the needle should be properly placed on the cataract The cataract should then be scribed and scribbled by the point of the curved barley-tipped needle The attachment of the cataract is thus loosened as much as is required



Susruta's barley-tipped needle, the Jabamukhi Salaká

The nose of the patient is now closed The patient is asked to sniff and snore and draw in phlegm from the nasal sinuses into his throat, while his nose is held and the nostrils are thus kept closed During this time the surgeon lances the cataract and pushes it toward the nose as well as downward with the tip of the needle until the cataract is dislocated and slipped out of the field of vision This maneuver should be continued with gentle care and without violence till the area of vision is cleared of the cataractous obstruction

Then the needle is slowly and carefully withdrawn The patient at once regains clear eyesight, as the sun shines after the sky is clear of the clouds When objects are seen in their true colors, the eye should be besmeared with boiled butter and bandaged with a cloth all around the head

Then the patient should be laid on his bed, calmly and comfortably, in a room without disturbance He should remain absolutely quiet in his bed and should not cough, sneeze, spit, eructate, move or sit up for a few days He should make it a rule to drink fresh milk for some days regularly, which will be good for his eyes

Three days after the operation the eye should be washed with a lotion of boiled myrobalan and should be rebandaged after boiled butter is applied The eye should be regularly dressed every three days as before

The patient should continue to rest and to take milk for ten days The bandages should be removed after ten days Soft rice meal and semisolid food should then be taken for a few days with fresh milk and other liquids

⁶ Rao, T D H Melaena Neonatorum Checked by Mother's Milk, Burma M Times 1720 (Feb) 1937

Susruta, in emphasizing caution and carefulness, wrote

The surgeon should not operate on a patient who had previously suffered from some inflammatory disease of the eye or on a patient who is unfit for venesection owing to hemorrhagic tendency or diseased blood vessels

The surgeon should not puncture the ciliary plexus of blood vessels, nor should he puncture the blue iris. During the operation if the ciliary blood vessels or the ciliary body or the iris is punctured, the eye will be filled with blood. In such a case the milk of a nursing mother should be sprinkled into the conjunctiva and boiled butter applied, and the eye should be bandaged.

After the operation the patient who is cured actually gets new eyesight, and consequently he or she should not look toward strong light, blazing and powerful light, focused or reflected light or the sun for some weeks.

If the surgeon operates on an unripe cataract, it may not be dislocated, because a solid obstruction is not encountered with the tip of the needle. Even if the cataract is scribbled or scarified or scratched by the point of the needle, the cataract may not be properly dislocated and removed. The cataract may only be punctured by the point of the needle and partially moved or subluxated. In such a case the cataract forms again and becomes more thick and opaque afterward and obstructs eyesight as before. Moreover, the after-cataract may even become red, inflamed and painful and therefore worse than before. Hence, the couching of a cataract should be done when it is mature. The most suitable ones are the pearl-shaped shining white cataracts.

As regards operation for hard cataract, when the cataract has become very hard, like a "masur" lentil, and its capsule very thick and so large as to cover the whole field of vision, the surgeon will be successful. The cataract can be dislocated and removed from the field of vision by the tip of the needle, as a cloud is removed by the wind. It may be couched by pushing with the tip of the needle, by turning the tip of the needle and by moving the cataract from side to side with the point of the needle. But after a long time the cataractous mass may float again and obstruct sight.

These observations of Susruta are correct. Couching is unsuitable for the unripe cataract but most suitable for the soft mature and the hypermature morgagnian cataract—the pearl-shaped shining or glistening variety of Susruta.

Though the immediate results of couching in cases of hard mature and of hypermature cataract with a large nucleus are successful, after a few years the cataract floats forward into the aqueous and obstructs sight.

In this connection, the observations of Smith, who made a special study of the results of couching and followed up hundreds of patients for many years on whom couching was done by quack couchers of the Punjab, will testify to the accuracy of Susruta's remarks.

Smith observed that soon after the couching of a mature soft cataract with a small nucleus and much cortical matter or of a morgagnian cataract the soft fluid content of the lens was rapidly absorbed by the vitreous and only the small nucleus within the shrunken capsule remained embedded in the vitreous. Good vision remained for as long as seven

years with a diminished visual field. When a hard cataract with a large nucleus was subjected to couching there was seldom useful vision for more than three years. Simple progressive atrophy of the optic nerve spread from the periphery toward the macula, or the large lens some time after floated up and obstructed vision.⁷

The foregoing translations describe the depression method of couching by the anterior route as recorded in "Susruta Samhita." Suśruta invented and taught couching sometime about 1000 B. C.

Enough material can be found to trace the path of the progress of operations for cataract through Arabia, Bagdad, Egypt, Alexandria, Babylon, Jerusalem, Athens and Rome to other countries of Europe. As is usual every expert surgeon modified the original technic of Susruta according to his skill, knowledge and experience from time to time.

Philoxenes, a renowned Egyptian surgeon of Alexandria, who lived and practiced couching about 300 B. C., is referred to by Celsus.⁸ There were also surgeons in Babylon, Arabia, Jerusalem and Athens who practiced couching at that time.

Then Celsus, of Rome, in 10 A. D., made a radical change by introducing the reclination method of couching by the posterior route.

The following description is that of the operative procedure of couching as introduced by Celsus.⁹

The patient should be placed on a light and nonstimulating diet. When the operation is to be performed he is seated in front of the operator, facing the light. The surgeon sits on a higher seat, while an assistant fixes the head of the patient. The sound eye should be bandaged with a woolen bandage. The couching needle is held with the right hand for operation on the left eye and with the left hand for operation on the right eye.

The needle punctures the sclera on the temporal side at a distance from the corneal circumference. The needle is passed through the vitreous toward the middle of the cataract till it comes in contact with or punctures the cataract on its posterior side. Then the needle is turned so as to move the cataract till the zonule is ruptured. After rupture of the zonule, the point of the needle is directed downward, so as to remove the cataract below the region of the pupil. The cataract is pressed deep into the lower part of the orbit. After the operation the eye is bandaged with a woolen bandage.

⁷ Smith,¹ p. 205.

⁸ Celsus, A. C. *Of Medicine*, translated by J. Greive, London, D. Wilson and T. Durham, 1756.

⁹ Celsus, cited by Adams, W. *A Practical Inquiry into the Causes of the Frequent Failure of the Operations of Depression and of the Extraction of Cataract*, London, Baldwin, Cradock & Joy, 1817, footnote 8.

The Grecian surgeon, Galen (second century A D), also modified the technic of couching as he found convenient

MacKenzie,¹⁰ in his "Practical Treatise on Diseases of the Eye," described the technics of both the depression and the reclination method of couching, as performed by charlatans, rowals, mals, sattyas or vaidyas Elliot,¹¹ Maynard,¹² Smith¹³ and Drake-Brockman¹⁴ have also recorded the methods and statistics of the results of couching by these quack couchers, who were ignorant of the anatomy of the eye and of surgical technic and had never heard of the names of Suśruta and Celsus The technic of the two great pioneers in the field of the operative treatment of cataract have degenerated in the hands of these quacks Moreover, they had no idea of cleanliness and of the postoperative treatment formulated by Suśruta Consequently, the results of couching in the hands of these quacks were not only miserable but often disastrous MacKenzie, Elliot, Maynard, Smith and Drake-Brockman and other great surgeons have done the greatest service to suffering humanity by exposing these quacks and by declaring a crusade against quackery in operations for cataract I think that all ophthalmologists should join in such a crusade today

With Celsus' reclination method of couching by the posterior route the needle is inserted into the temporal side of the sclerotic, beyond and behind the danger zone of the ciliary body, i e., about from 8 to 10 mm outside the limbus The needle is carefully pushed through the vitreous gel till it reaches and punctures the cataractous lens at its posterior aspect, and it is then skilfully turned so as to dislocate the cataract The dislocated cataract is gently pushed downward and toward the nasal side, away from the field of vision, deep into the vitreous If it is pushed forward, the lens will slip into the anterior chamber This is to be carefully avoided

With this method, the vitreous gel is unavoidably ploughed and damaged by the turns and maneuvers of the needle which are done to dislocate the lens and to lodge the cataract deep into the lower nasal region of the vitreous chamber Consequently, the results of couching by the reclination method were not as good as those of couching by the depression method

10 MacKenzie, W A Practical Treatise on the Diseases of the Eye, Philadelphia, Blanchard & Lea, 1855

11 Elliot, R H The Operation of Couching as Practiced in Southern India, *Ophth Rev* **31** 259 1912, The Indian Operation of Couching for Cataract, London, H K Lewis & Co, Ltd, 1917

12 Maynard, F P After-Results of Sixty-Three Operations for Depression of the Lens Performed by Indian Cataract Pickers, *Ophth Rev* **22** 91, 1903

13 Smith, H Cataract Couching, *Tr Ophth Soc U Kingdom* **24** 264, 1904

14 Drake-Brockman, H E The Indian Oculist and His Instruments, *Tr Ophth Soc U Kingdom* **15** 249, 1895

Bagbhat (225-300 A D), professor of surgery in Taxila University in ancient India, therefore revived the depression method by the anterior route with some modifications. He¹⁵ recorded his method of couching in his book on surgery entitled "Astanga Hrīday". A literal translation, made by me from the original Sanskrit, will be of interest to the readers.

The technic of Bagbhat's couching method follows

In good season in the morning after sunrise and after regulated proper diet on the previous days and proper cleansing of the patient, of the surgeon also, the operation of couching of the cataractous lens should be performed. The patient should be seated conveniently and immovably before the surgeon. The surgeon should take his place on a seat as high as his knee. The patient's head should be properly held, and his eyelids should be retracted by the thumb and fingers of an assistant. The eye should be warmed by the breath of the mouth. The patient should look at his own nose, while his head is held immovably.

The needle should be held by the surgeon between the thumb and the middle and index fingers of the right hand in case of operation on the left eye and of the left hand in case of operation on the right eye.

The needle should puncture the sclerotic on the temporal side, just outside the black border of the eye. It should then be steered from the side into the natural aperture of the pupil. The cataract is moved by the needle and dislocated and gradually pushed aside by the tip of the needle.

When the puncture of the coats of the eye is properly done, there is a sound. Mother's milk should be put into the conjunctiva, in order to soothe the eye and prevent bleeding.

When the needle is properly passed through the natural aperture of the pupil there is no obstruction. When the cataract is reached, an obstruction will be felt. The cataract should be scribbled and stretched round and round with the point of the needle till it is dislocated, and then it should be gently pushed toward the nose. Mother's milk is sprinkled into the eye until the cataract is dislocated and dislodged out of the field of vision. The barley-tipped needle should then be gently taken out of the eye. The vision becomes clear at once.

The eye should be properly bandaged, after being covered by a piece of cotton lint soaked in boiled butter.

If one eye is operated on, the patient should be made to lie on the opposite side. If both of the eyes are operated on at one sitting, the patient should be made to lie on his back. The patient should lie absolutely calm and quiet and should not move his head. He should fast for one day. He should not sneeze, cough, eructate, spit or drink anything for one day.

The patient should not lie face downward and should not bathe, brush his teeth or eat solid food for seven days. He should drink only bland liquids and enough fresh milk for seven days.

The eye should be opened after three days and washed with a lotion of boiled antiphlogistic drugs.

If all goes well, the bandages can be removed after seven days. However, although the eye is kept open, the patient should not look toward fine things or look at a glare or strong light suddenly but should wait till the normal condition of the eye returns.

15 Bagbhat. Astanga Hrīday, Calcutta, B. L. Sen, 300 A D, verses 9 to 17.

But if the patient has misbehaved during the operation or showed temper or rubbed his eye or has misbehaved in any other way during the postoperative period of one week, or if there has been any rough handling during the operation or an incorrect puncture has been done, or if the condition of the eye was not favorable for the operation, then the patient must undergo a longer period of restraint, and the eye should be kept properly bandaged during this period. After the period of restraint, the use of milk and nonstimulating diet should be continued for some time.

Bagbhat divided cataracts into the following varieties

A cataract may be white, black or shining white, like bellmetal. It may be of a greenish yellow hue, like the color of a parrot, or of a brownish or a reddish color. It may be crescent shaped, semilunar or full moon shaped. The contents may be like milk or butter or sugar candy. A cataract may be hard and its capsule thick.

Concerning the cataracts fit for operation, he stated

An uncomplicated ripe cataract which is not associated with inflammation of the surrounding tissues, pain or redness or pus formation in the eyes is fit for couching.

Bagbhat gave the following contraindications for couching

Couching of a complicated cataract will cause intense pain, and vision will be ultimately lost. The eye will soon be filled with pus, and there will be other difficulties. Therefore, do not operate on patients with otitis or otorrhea or on patients with pain and inflammation in the eye. Do not operate on patients who are very nervous or mentally deficient, who suffer from dyspepsia and vomiting, who have very irregular habits, who suffer from cough and diseases of the nasopharyngeal and respiratory tracts or who are unfit for venesection.

The Caliphs of Bagdad ordered that "Susruta Samhita" and "Astanga Hridaya" be translated into Arabic. Learned men of Arabia and India were called to Bagdad to consult and prepare correct translations of these great works. It took many years to complete them, and it was in the beginning of the eighth century A. D. before the translations were ready. The translated works were named "Ketab-ā-Susrud," i. e., "Book of Susruta," and "Sindhī-Saī," i. e., "Surgery by Sindhī." Bagbhat's surname was Sindhī, as he was an inhabitant of the province of Sind, in India.

The translation of these works gave a great impetus to the depression method of couching by the anterior route in Arabia as well as in the neighboring countries. Many Arabian surgeons won great renown in the field of cataract operation. Arabian surgeons of the eighth century A. D. practiced couching extensively and introduced certain modifications of Susruta's technic. They made a small incision at about the limbus with a knife and used a blunt needle for dislocation of the cataract if it was hard. But if the cataract was of the soft variety, they

introduced a small tube through the incision into the anterior chamber and then into the cataract and sucked out the fluid cortical matter

Rhazes (850-932 A D) an Arabian surgeon who won great renown in couching in his writings quoted from Ketab-ā-Susiud as authority

Avicenna (980-1037 A D), the renowned Arabian surgeon of the tenth century A D , introduced a modification in the technic of couching and used a double-edged knife for incision of the cornea and a blunt needle for dislocation of the lens

Benevenutus,¹⁶ of Jerusalem, an expert surgeon in the field of cataract operations during the eleventh century A D , practiced couching extensively His writings have been translated by Casey Wood, who stated that they were the most popular ophthalmic manuals of the Middle Ages Benevenutus introduced certain modifications in the technic of couching He gave an initial purgative compounded by himself, called "Jerusalem pills " He made the patient sit on a bench, as on horseback He used a silver or a gold needle for the operation and egg albumin for dressing the eye, he finally opened the bandages after eight days He stated that his technic was based on the teachings of the ancients, modified in accordance with his own wide experience, gained by practice in various parts of the world

Then came the Dark Ages, during which no renowned surgeon took up the subject of the surgical treatment of cataract The practice of couching was consequently captured by quacks The techniques of Susiuta and Celsus degenerated in the hands of these quacks, who were ignorant of the anatomy of the eye and of the operative techniques and postoperative rules framed by Susiuta They wandered from place to place to ply their trade and were called charlatans in Europe and lowals, mals, sattyas and vaidyas in different provinces of India No wonder that the results of couching in their hands were bad in the majority of cases

This state of things continued till 1745 A D , when Daviel, of France, devised the technic of capsulotomy The discovery of local anesthetics and antiseptics has added to the ease and safety of the open methods of operation Intracapsular methods are being devised to obviate after-cataracts and the necessity of further needling

The open methods are superior to couching in that with capsulotomy the nucleus of the lens is extracted and with the intracapsular procedure the whole of the lens in the capsule is extracted Hence, no difficulty arises later by the lens floating in the aqueous chamber and thus obstructing vision With this exception, couching is free from

¹⁶ Grassus, B De oculis, translated from the first printed edition by C A Wood, Stanford University, Calif, Stanford University Press, 1929

danger if practiced by an ophthalmic surgeon and even may be practiced successfully on a person who would be a bad risk if operated on by any of the open methods

Smith truly observed that if antiseptic precautions are taken in couching, complications can be avoided. In cases in which there are no complications and the operation is successful, the cosmetic result is perfect, it is seldom equaled and is never surpassed by any modern method of cataract operation. But the results of couching are not lasting. In cases of soft cataract useful vision lasts for seven years with a diminished visual field. In cases of hard cataract useful vision seldom lasts for more than three years, simple progressive atrophy of the optic nerve takes place, and the cataractous lens may float up into the aqueous chamber and obstruct vision.¹⁷

Jonathan Hutchinson, who in his younger days saw couching performed at Moorfields, in London, was of the opinion that had the good results of couching been permanent the operation would never have been given up, even after the advent of listerism, which made the open methods of operation practicable.

Hirschberg¹⁸ (1894) formed a favorable opinion as to the successful results of couching.

Johnson¹⁹ has published an article in the ARCHIVES entitled "A Plea for Reviving the Operation for Couching."

Powers²⁰ published an article in the *British Medical Journal* in which he made a plea for the occasional performance of the operation of depression on cataracts which are inoperable by the open methods.

Is it not possible to rise above prejudice and to consider dispassionately the plea for couching raised by the foregoing ophthalmologists? Can one not pause and ponder over the pros and cons of couching in cases in which open methods of operation are dangerous before declaring a cataract to be inoperable?

There can be no doubt that under modern conditions of asepsis, anti-sepsis, mydriasis and local anesthesia and with modern instruments, viz, the eye speculum and the fixation forceps, in addition to Susruta's barley-tipped needle, couching under a conjunctival flap is much less risky than any of the open methods of operation. But, according to Smith, good vision may not last for more than seven years in cases of soft cataract or for more than three years in cases of hard cataract.

¹⁷ Smith,¹ p 204

¹⁸ Hirschberg, J. Ueber den Star-stick der Indian, Centralbl f prakt Augenh **18** 48, 1894

¹⁹ Johnson, G. L. A Plea for Reviving the Operation of Couching, Arch Ophth **54** 466 (Sept) 1925

²⁰ Powers, H. A Plea for the Occasional Performance of the Operation of Depression in Cases of Cataract, Brit M J **2** 1200, 1901

However, good vision for from three to seven years is something for the blind, and the adage goes that "something is better than nothing" Is this period of good vision not enough of a lease on new sight, at least for the aged and debilitated patient, whose span of life may not extend longer than from three to seven years? Under the circumstances, who can deny that Suśruta's depression method may still prove to be a boon to persons whose condition is declared hopeless and past all benefit from modern surgical measures?

It is not generally known how much credit is due Suśruta for cataract operations. One should not be misled by criticisms on couching in the hands of quacks. Suśruta's influences on modern operations for cataract may be scrutinized with benefit even today. There are many points in Suśruta's technic which deserve the best attention of the modern ophthalmic surgeon and may be adaptable to modern conditions, with or without some modifications. These are many and should be selected by the prudent without prejudice and without derision for "The Father of Cataract Operations."

There are other points in the technic of Suśruta which are unknowingly followed today but which have no significance. These should be looked on as unprofitable legacy and should be discarded by those who follow them.

May I cite a few good points and also a few old relics from the technic of Suśruta as examples?

The directions of Suśruta which should be discarded by those surgeons who unknowingly follow them will be considered first.

1 It is no longer necessary to change hands for operations on different eyes. A change of hands was unavoidable with Suśruta's technic, because couching was done after the patient was seated in front of the surgeon. Now that an operation for cataract is performed after the patient is placed on the operating table, this Suśrutian practice of changing hands has no significance and is worse than useless. The surgeon can now hold the cataract knife in his best hand, the right hand in the case of a right-handed surgeon and the left hand in the case of a left-handed surgeon, and can operate on either eye if he simply changes his place of standing, either by the side of the patient or toward the head of the patient. Thus, a right-handed surgeon, holding the cataract knife in his right hand, can conveniently operate on the left eye if he stands on the left side of the patient and on the right eye if he stands at the head of the patient. Similarly, a left-handed surgeon, holding the cataract knife in his left hand, can conveniently operate on the right eye if he stands by the right side of the patient and on the left eye if he stands at the head of the table. This change of position will be most convenient for the surgeon, because there will be no necessity to practice

ambidexterity It will be most beneficial to the patient, who will surely get the benefit of the best hand of the surgeon Moreover, to acquire dexterity of the unused hand in operations for cataract the surgeon has to operate first in 100, or at least 50, cases of cataract In these 50 or 100 cases the surgeon will have to be satisfied with a less expert operation than he can perform with the practiced hand Again, the patient also has to be satisfied with less satisfactory results Is this justifiable?

Moreover, ambidexterity, however perfected by practice, cannot equalize the dexterity of the two hands used together, especially in operations for cataract An operation for cataract is a delicate procedure An eye is a precious thing The patient has every right to the benefit of the surgeon's best hand in the operation

Ambidexterity is a relic of the prehistoric age and should not be blindly advocated now This Susrutism should be discarded as unwarranted and condemnable

2 The direction of Susruta to the patient to look equably at his own nose is another relic of antiquity This direction was necessary in order to expose the upper outer portion of the eyeball and to prevent the patient from moving and rolling his eye during the operation It was indispensable, because Susruta did not use an eye speculum or fixation forceps

But now that modern methods include the use of a speculum to keep the eyelids wide open, anesthetization of the eye and the use of fixation forceps to fix the eye, this Susrutism is unnecessary The issuing of such orders during a period of great anxiety and suspense to the patient is irksome and makes him more nervous He should be allowed to keep his eye in its normal position of rest Moreover, as Susruta advised, "the patient should have a cool temper and undisturbed mind" and one should not unbalance it unnecessarily With the aid of fixation forceps the surgeon should be competent to turn the eyeball and to fix it in the position convenient for the operation

I shall next cite a few good points from Susruta's instructions for the consideration of my readers

1 Susruta noted that the point of puncture should be on the horizontal diameter of the eyeball, just outside the limbus on its temporal side This point should be free from blood vessels, so that unnecessary annoyance by oozing of blood may be avoided This advice still holds good

2 As a routine, Susruta's advice of ten days' rest in bed with dressings and of change of dressings every three days will even now be found most suitable during the postoperative period Any deviation should be made in special cases only Unnecessary daily interference for washing the eye predisposes to prolapse of the nris and does more harm than

good. With the host of antiseptics at one's disposal, there is no reason why the wound cannot be kept aseptic for three days at a time. Suśruta managed this in the preantiseptic days with the aid of the natural lysozymes of the lacrimal secretion and the phagocytic action of the conjunctival cells and the use of a mild antiseptic and astringent lotion of boiled myrobalan and the application of boiled butter.

To the advice of Suśruta to open the bandages after ten days, I should like to add that if it is done in the evening the darkness of the night will be better tolerated when new sight is regained. At least in a tropical country the glare of the dazzling sun can be avoided.

3. The special stress laid by Suśruta on the use of a milk diet after the operation and the addition of fresh milk to the diet of the patient in sufficient quantity for some time after cannot be overlooked. This, explained according to modern scientific knowledge, means that fresh milk, which contains natural and easily assimilable vitamin A and other vitamins, should be added to the dietary of the patient to insure an adequate supply of vitamins necessary for the nutrition of the incised corneal flap and rapid healing of the operative wound. To this I may add that the addition of some cod liver oil in a suitable form and fresh eggs will further increase the vitamin content of the diet. The efficacy of such a diet is proved by experiment and experience.

The foregoing review is the ABC of the surgical treatment of cataract as it was taught in the University of Taxila and the University of Nalanda in ancient India and much earlier in the Asram schools of the surgeon-sages of Hindustan at least three thousand years ago, if not earlier. No knowledge concerning operations for cataract can be called complete without some knowledge about "The Father of Cataract Operations" and his original mode of operation, as recorded in "Suśruta Sanhita." I have spared no pains to translate this work for presentation to the readers. If this is found interesting, then my labors will be fully rewarded. India is the land of cataracts. Necessity is the mother of invention. No wonder that the first operation for cataract was devised and performed in India.

PROBLEM OF RICKETTSIAS IN TRACHOMA

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In 1933 Busacca¹ reported the finding of minute bodies which he believed were rickettsias in conjunctival epithelial cells and in the mononuclear cells of trachoma follicles. Similar findings were later reported by Cuénod and Nataf,² Poleff³ and Foley and Parrot,⁴ but unlike Busacca, these investigators identified the bodies with the elementary and initial bodies of the epithelial cell inclusion (Halberstadter and Prowazek). Complicating this interpretation, however, was the fact that they described the bodies as occurring abundantly in the follicles (both free and in the cytoplasm of mononuclear cells), a site at which the inclusion bodies have never been known to occur.

In the course of my studies on the epithelial cell inclusion body of trachoma, a large number of preparations from epithelial scrapings stained by Giemsa's method and expressed follicular material were examined for micro-organisms of the Rickettsia group. Except for the elementary and initial bodies, which undeniably bear a resemblance to certain Rickettsia (particularly Rickettsia ruminantium of Heartwater⁵), no rickettsia-like bodies could be found. The elementary and

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1 Busacca, A. Sulla presenza di germi simili a Rickettsie nei tessuti trachomatosi, *Klin Monatsbl f Augenh* **91** 277, 1933, Ueber das Verhändensein von Rickettsien-ähnlichen Körperchen in den trachomatösen Geweben und über das Vorkommen von spezifischen Veränderungen in Organen von mit Trachom-Virus geimpften Tieren, *Arch f Ophth* **133** 41, 1934, Un germe aux caracteres de rickettsies (Rickettsia trachomae) dans les tissus trachomateux, *Arch d'opht* **52** 567, 1935, Is Trachoma a Rickettsial Disease? *Arch Ophth* **17** 117 (Jan) 1937.

2 Cuénod, A. Note préliminaire sur la présence d'éléments infra-microbiens dans les follicules trachomateux, *Arch d'opht* **52** 145, 1935. Cuénod, A., and Nataf, R. Deuxieme note sur la présence d'éléments infra-microbiens dans les follicules trachomateux, *ibid* **52** 573, 1935, Troisieme note sur l'agent bactérien du trachome, *ibid* **53** 218, 1936.

3 Poleff, L. Culture des Rickettsias du trachome in vitro, *Arch d'opht* **53** 882, 1936.

4 Foley, H., and Parrot, L. Sur la Rickettsia du trachome, *Compt rend Soc de biol* **124** 230, 1937.

5 Jackson, C. The Microscopic Diagnosis of Heartwater. A Preliminary Note on the Value of Intima Smears, in Seventeenth Report of the Director of Veterinary Services and Animal Industry, Union of South Africa, August 1931, p 161.

initial bodies were seen only in epithelial scrapings, never in follicular material, and were difficult to demonstrate outside the cells except in cases of acute trachoma. It seemed evident that they could not be what either Busacca or Cuénod and Nataf had described.

In response to letters stating that I had been unable to demonstrate rickettsias in my trachomatous material, Cuénod and Nataf forwarded stained specimens, and Busacca, both stained and unstained material.

The Tunisian slides furnished by Cuénod and Nataf consisted of expressed follicular material stained by the method of Giemsa and differed in no way from similar preparations of trachomatous material obtained in the United States. Elementary and initial bodies either were absent or were present in too small numbers to be demonstrated, and I was unable to find any bodies similar to the rickettsias of Rocky Mountain spotted fever or endemic typhus fever or to the conjunctival rickettsias, such as are found in sheep, goats and cattle with conjunctivitis (Coles⁶), specimens of which were at hand for comparison. There was a considerable amount of the usual punctate debris, which is derived in major part from the damaged cytoplasm of the large mononuclear cells of the follicles, and it was probably these formations which had been considered rickettsias by Cuénod and Nataf.⁷

The material from Brazil provided by Busacca consisted of impression smears made from excised trachoma follicles and had been stained heavily by the Giemsa method. No free bodies corresponding to known rickettsias were seen, but reddish granules, irregular in size and not sharply outlined, were noted inside some of the large mononuclear cells. These, apparently, were the bodies described by Busacca as rickettsias, in my opinion, they were probably cell granules accentuated by prolonged staining. Similar formations were observed in my own material when it was subjected to prolonged staining and could also be produced in trachomatous and nontrachomatous epithelial cells.

In order to have the opinion of an investigator familiar with rickettsias, I forwarded Busacca's preparations to Dr. Ida A. Bengtson, of the National Institute of Health, Washington, D. C., who was working currently with the rickettsias of Rocky Mountain spotted fever and endemic typhus and had also had experience with trachoma. She reported that she was unable to find any rickettsia-like bodies.

6 Coles, J. D. W. A. A Rickettsia-Like Organism in the Conjunctiva of Sheep, in Seventeenth Report of the Director of Veterinary Services and Animal Industry, Union of South Africa, 1931, pp. 175-186, An Unknown Intracellular Organism of the Conjunctival Epithelium of Sheep, *ibid.*, pp. 187-189, J. South Africa Vet. M. A. 7 4, 1936.

7 In a personal communication, L. Parrot stated that in his opinion Cuénod and Nataf had confused with Rickettsia simple alterations of the cytoplasm of cells of the trachomatous follicles.

Busacca⁸ suggested victoria blue as a differential stain, but with his recommended technic I was still unable to demonstrate formations which I could consider rickettsias, either in the unstained material provided by him or in trachomatous material obtained here in the United States

CONCLUSIONS

Trachomatous materials from Tunis, Brazil and the United States were examined for the rickettsia-like bodies described by Busacca and Cuénod and Nataf. No minute parasitic bodies other than the elementary and initial bodies of the epithelial cell inclusions of trachoma could be demonstrated. It is believed that the formations which these observers described as occurring in large numbers in the trachoma follicles are not parasitic but in all probability cell granules and cytoplasmic débris.

⁸ Busacca, A. Methode simple et rapide au bleu victoria pour la demonstration des rickettsies du trachome, *Folia clin et biol* 7 253, 1935

CONJUNCTIVITIS ASSOCIATED WITH INFECTION BY STREPTOCOCCUS VIRIDANS

CLINICAL AND BACTERIOLOGIC OBSERVATIONS IN AN EPIDEMIC

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During the summer of 1936 we had an opportunity to study an epidemic of acute conjunctivitis in a boys' camp. The clinical appearance of the patients and the results of routine smears of material from the conjunctivas suggested that a pneumococcus was the causative organism. Cultural studies, however, resulted in the isolation of *Streptococcus viridans* from the inflamed conjunctivas. In view of the rarity with which this micro-organism has been thought to be of etiologic significance in conjunctivitis, it was considered worth while to report the observations made during this epidemic.

Relatively little importance has been given to *Str. viridans* as a normal inhabitant of the conjunctival sac or as a causative agent of acute conjunctivitis. In the older literature, moreover, this type of organism has often been referred to by the all-inclusive term "streptococcus," with little or no attempt to differentiate between hemolytic, green and indifferent species. Though Axenfeld¹ and Fuchs² stated that streptococci are rarely found in the normal conjunctiva, recent observations indicate that these micro-organisms may be present in the normal conjunctiva more frequently than has been previously suspected. Keilty³ found nonhemolytic streptococci in 11 of 91 cases (12 per cent). Schmelzer and Eckstein⁴ reported that streptococci were

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1 Axenfeld, T. *The Bacteriology of the Eye*, translated by Angus McNab, London, Bailliere, Tindall & Cox, 1908.

2 Fuchs, E. *Textbook of Ophthalmology*, translated by Alexander Duane, Philadelphia, J. B. Lippincott Company, 1911.

3 Keilty, R. A. *The Bacterial Flora of the Normal Conjunctiva with Comparative Nasal Culture Study*, *Am J Ophth* **13** 876, 1930.

4 Schmelzer, H., and Eckstein, E. *Pneumokokken und Streptokokken am Auge*, *Ber u d Versamml d deutsch ophth Gesellsch* **49** 376, 1932.

present in 74 of a series of 275 cases, *Sti viridans* being present in 64 and an indifferent type of organism in 3

Concerning the type of conjunctival disease produced by the various types of streptococcus, in the older standard texts (Fuchs,² Weeks,⁵ de Schweinitz,⁶ Parsons⁷ and Axenfeld¹) it has been almost universally held to be pseudomembranous conjunctivitis. Axenfeld expressed the belief that simple primary streptococcic conjunctivitis is extremely rare and stated that he could find no record of a true epidemic due to streptococcic infection. Until the papers of Schmelzer and Eckstein appeared, the various epidemics of "pink eye" and acute conjunctivitis studied were reported to be due to pneumococci or to the Koch-Weeks or the Morax-Axenfeld bacillus. In 1932, however, Schmelzer and Eckstein⁴ reported the bacteriologic investigation of an epidemic of so-called pneumococcic conjunctivitis in their clinic at Erlangen, Germany. Of 20 cases, pneumococci were causative in only 7, while *Sti viridans* was the causative organism in 13. In a further paper in 1934 these authors⁸ reported a series of 155 cases of acute conjunctivitis, in 64 of which the condition was due to *Str viridans* and in 3 to an indifferent streptococcus. Michaelson⁹ presented a clinical and bacteriologic study of 272 cases of conjunctivitis in Glasgow, Scotland, in 13 of which nonhemolytic streptococci were causative. Kleuver¹⁰ recovered streptococci in 7 of 448 cases of conjunctivitis, and in 2 of these 7, 1 of primary and 1 of secondary conjunctivitis the organism was *Str viridans*. It is worthy of note that, with the exception of the local epidemic reported by Schmelzer and Eckstein, the data recorded here were derived from analyses of the statistics of large clinics covering years and not from studies of epidemic conjunctivitis.

MATERIAL AND METHODS OF STUDY

The material on which this study was based was obtained from 35 patients who had acute conjunctivitis in close succession in a boys' camp in the Pocono Mountains of eastern Pennsylvania. Except for the usual influx of guests for week-ends, the boys in the camp were comparatively isolated from outside con-

5 Weeks, J. E. A Treatise on Diseases of the Eye, Philadelphia, Lea & Febiger, 1910

6 de Schweinitz, G. E. Diseases of the Eye, Philadelphia, W. B. Saunders Company, 1892

7 Parsons, J. H. The Pathology of the Eye, London, Hodder & Stoughton, 1904-1906

8 Schmelzer, H., and Eckstein, E. Die augenpathogene Bedeutung der Streptokokken und Pneumokokken (547 Untersuchungen), Arch f Ophth **132** 24, 1934

9 Michaelson, I. C. Conjunctivitis. A Clinical and Bacteriological Investigation, Glasgow M. J. **123** 185, 1935

10 Kleuver, H. C. Streptococci in Inflammations of the Eye, Am J Ophth **18** 805, 1935

tacts. The permanent population numbered about 180, including 135 campers, whose ages ranged from 4 to 17 years, and 34 counselors, all over 18, the kitchen help comprised the remainder. One case in which conjunctivitis was contracted during an epidemic in a camp in Vermont is included.

Direct smears were made of material from the inflamed conjunctival sac in all 35 cases, and in 7 instances the conjunctival exudate was cultured on blood agar. The strains thus recovered were studied culturally and serologically.

CLINICAL DESCRIPTION OF THE DISEASE

The disease was of acute purulent type and of varying severity. The majority of the patients presented themselves at the infirmary because of a discharge from the eye. In many instances it was possible to elicit a history of itching, burning and a sensation of a foreign body in the eye for several hours prior to the onset of the discharge. In the younger children, sticking of the eyelids at the time of arising was sometimes the initial symptom. In the typical case, in from two to four hours after the onset of discomfort in the eye a slight mucopurulent discharge appeared. This rapidly became profuse, reaching its peak in about twelve hours, with intense engorgement of the conjunctival vessels (predominantly in the fornices), some edema of the lids and variable chemosis of the bulbar conjunctiva. Marked discharge continued for from twenty-four to forty-eight hours, then diminished gradually and usually ceased on the fourth or the fifth day. Conjunctival hyperemia rapidly subsided until the time of "clinical cure," which took an average of seven and four-tenths days.

Marked bulbar chemosis and edema of the lids were present in only 2 instances. In no case was corneal ulceration noted. Petechiae were never observed. With the exception of mild dacryocystitis in 1 case and recurrence in 5 cases, there were no complications.

Direct smears made during the stage of discharge revealed innumerable pus cells, occasional epithelial cells and many diplococci in every instance. In 2 instances smears made during the prodromal period, when the clinical appearance of the eyes did not warrant a definite diagnosis, revealed moderate numbers of pus cells and typical micro-organisms. Within the next few hours profuse purulent discharge appeared in both cases.

ANALYSIS OF MATERIAL

Acute conjunctivitis developed in 35 cases during the epidemic (table 1). It was distributed throughout the camp, though in the two oldest groups, senior (aged 14 to 17) and counselor (over 18), the incidence was lowest. The average duration of the first attack in each group is indicated in the table, that for the entire 35 cases was seven and four-tenths days. There was marked variation in duration in the individual cases, the time ranging from two to sixteen days. The groups were too small, and the variation too great, for the mode to be of

statistical significance. There were 5 recurrences, representing reinfection of the eye originally involved as well as new infection in the other eye. The average duration of the recurrences was slightly greater than that of the original attack.

Both eyes were involved (in either the first attack or by reinfection) in 23 cases. Except for the difference in duration, the disease was essentially similar in all groups. In no case at the time of acute symptoms was there an obvious infection of the nose, throat or sinuses.

The control referred to in table 1 was one of us who was inoculated by swab from the conjunctiva of a patient with symptoms of mild discomfort but no objective changes. Within three hours after the inoculation, typical purulent conjunctivitis developed in the patient. In the control, prodromal symptoms appeared twenty hours after inoculation and were followed in two and one-half hours by a profuse purulent

TABLE 1—*Classification of Cases*

Group	Age	No of Persons	No of Cases	Average Duration of First Attack, Days	Range of Duration of First Attack, Days	No of Recurrences	Duration of Recurrences, Days	Both Eyes Involved (1 or Both Attacks)
Midget	1-5	18	8	6.6	2-12	0	—	5
Freshman	5-7	25	6	7.6	2-15	1	9	4
Sophomore	7-10	29	6	9.3	3-14	2	3, 5	6
Junior	10-11	1	7	7.1	3-16	0	—	5
Senior	11-17	22	4	6.7	5-9	0	—	1
Counselor	18	31	3	6.6	5-9	1	5	1
Control	28	1	1	7	7	1	14	1

discharge. The experiment with this subject confirmed the impression that the period of incubation of the disease ranged between sixteen and twenty-four hours. The subject of the experimental inoculation was removed to New York city, where the course of the disease was in every way typical. For three days after "clinical cure" cultures of the material from the conjunctiva showed no organisms, but at the end of this period, twenty-four hours after the subject had been swimming in the ocean, he had a recurrence in both eyes, associated with (and apparently attributable to) mild dacryocystitis on the side of the original infection.

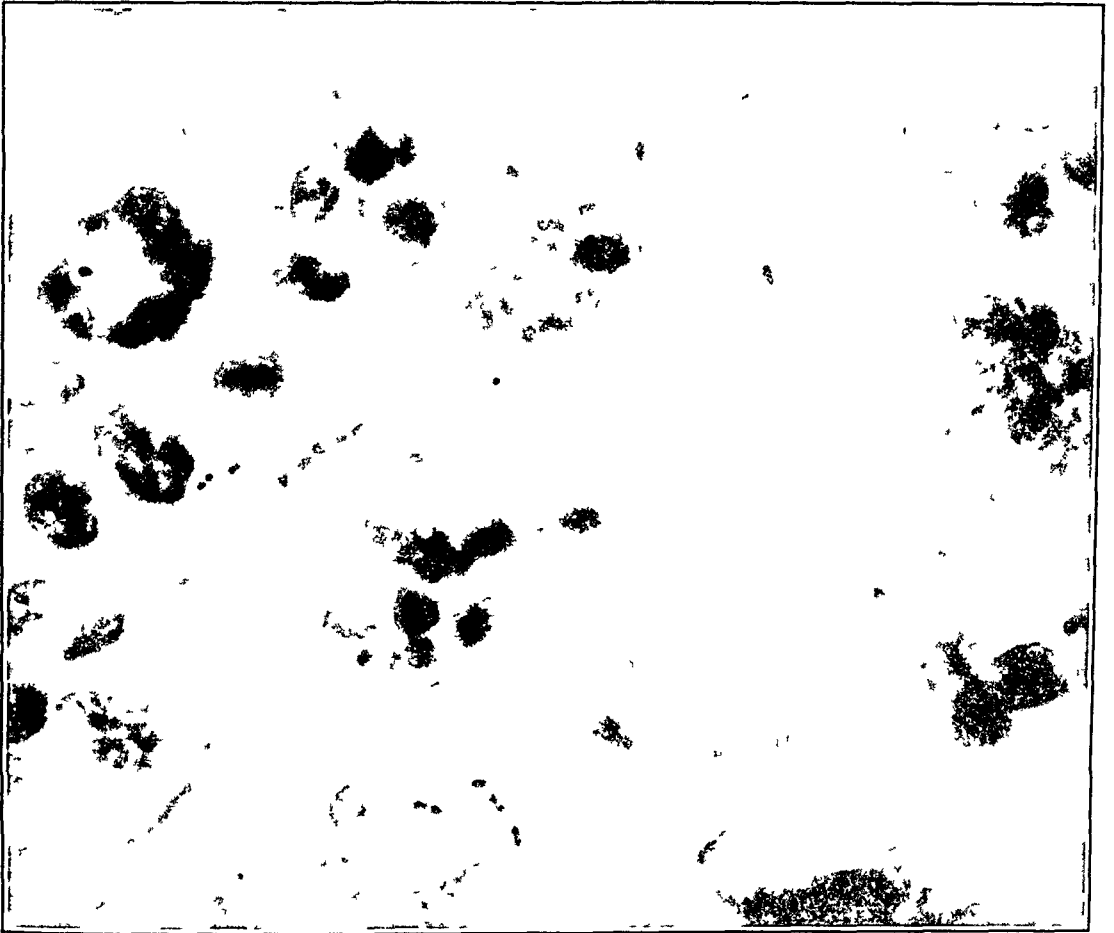
TREATMENT

In every instance treatment was preceded by a cleansing irrigation of the conjunctiva. The patients affected early in the epidemic were treated with instillations of 20 per cent mild protein silver, followed by irrigations with solution of boric acid and medication with a 0.5 per cent solution of zinc sulfate (3 drops to each eye). In the latter half of the epidemic the cases were divided into two groups. One group received irrigations and instillations of a 1 per cent solution of ethylhydrocupreine hydrochloride, the other, simple irrigations with solu-

tion of boric acid. The three types of therapy appeared equally ineffective in altering what we felt was the natural course of the disease.

BACTERIOLOGIC OBSERVATIONS

The material for the bacteriologic study was obtained from 7 patients affected during the epidemic in Pennsylvania and 1 who had the disease during an epidemic in a Vermont camp. In direct smears the organisms appeared as gram-positive diplococci, which were fre-



Photomicrograph of direct smear of material from the infected conjunctiva of a patient with a typical form of conjunctivitis at the height of inflammation, showing intracellular and extracellular lancet-shaped diplococci (Methylene blue stain, $\times 1,000$).

quently lancet shaped and commonly extracellular though occasionally intracellular (fig). Rarely, short chains were noted. In the methylene blue preparations, the diplococci were often surrounded by a clear refractive zone suggestive of a capsule. However, Hiss's copper sulfate stain of direct smears showed no capsules.

From material from each of these 8 subjects a pure culture of a strain of *Str. viridans* was recovered. On blood agar, after eighteen hours, these micro-organisms produced small colonies (0.5 to 1 mm), which were smooth and surrounded by a narrow green zone. Dextrose-

phosphate broth yielded a luxuriant growth with a relatively clear supernatant fluid and a fine flocculent deposit. Smears from broth cultures showed gram-positive diplococci and short chains. Capsules were not demonstrated. All strains were insoluble in bile, and none fermented inulin. The Neufeld¹¹ test for capsular swelling was applied to 4 of the freshly isolated strains, antiserum for thirty-two types of pneumococci being used, with negative results in every instance. The strains were not pathogenic for mice, despite repeated mouse to mouse transfer. With mucin,¹² however, it was possible to increase the virulence of strain DG by serial passage among mice so that the sediment of 3 cc of culture would kill a mouse in twelve hours, as compared with 100 cc required at the start of the experiment. Even after repeated passage through mice, the micro-organisms in any amount did not kill unless mucin was present. Inoculation of strain DG into the conjunctivas of rabbits and monkeys after augmentation of virulence failed to provoke any detectable reaction.

Vaccines were prepared from eighteen hour broth cultures of Pennsylvania strains DG and SE and Vermont strain X. The centrifuged micro-organisms, washed and resuspended in one-tenth the volume of physiologic solution of sodium chloride originally used, were killed by heating to 60 C for two hours. Rabbits were immunized over a period of five or six weeks by intravenous injection of these vaccines, with doses increasing from 0.1 to 0.8 cc. Agglutination tests were done according to the usual technic, 0.5 cc of living culture was added to an equal amount of serial dilutions of immune serum, and tests were read after two hours' incubation at 56 C and refrigeration overnight. As shown in table 2, all the specimens of serum agglutinated all the strains to a high titer.

To investigate further the relation between the strains suggested by simple agglutination, agglutinin absorption tests on specimens of X and of DG serum were carried out by the method of Krumwiede.¹³ The absorbing dose factors were 1:15 and 1:20, serum and antigen were incubated at 37 C for one hour and left in the ice box for eighteen hours. After absorption, the specimens of serum were cleared by centrifugation and agglutination tests again made (table 3). The absorption of agglutinins from serum DG by strain X and from serum X by strain DG were shown clearly. Such reciprocal absorption of agglutinins establishes the identity of these 2 strains, which were recovered from patients in widely separated localities.

11 Neufeld, F. Ueber die Agglutination der Pneumokokken und über die Theorien der Agglutination, *Ztschr f Hyg u Infektionskr* 40:54, 1902.

12 Nungester, W. J., Jourdonais, L. F., and Wolf, A. H. The Effect of Mucin on Infections by Bacteria, *J Infect Dis* 59:11, 1936.

13 Krumwiede, C., Cooper, G., and Provost, D. J. Agglutinin Absorption. *J Immunol* 10:55, 1925.

The soluble specific substance (S fraction) was prepared for strains X and DG by the method of Lancefield¹⁴ Tubes were set up with 0.2 cc of serum each and their contents brought to a total volume of 0.6 cc with varying dilutions of extract and saline solution They were

TABLE 2—Results of Agglutination Tests

Anti serum	Strain	1 20	1 40	1 80	1 160	1 320	1 640	1 1,280	1 2,560	1 5,120	1 10,240	1 20,480	Anti-gen*
SE	SE	+	++	+++	+++	+++	+++	+++	++	±	—	—	—
	DG	+	++	+++	++	+++	+++	+++	++	±	—	—	—
DG	DG	+	++	++	+++	+++	+++	+++	+++	+++	++	++	—
	SE	—	+	++	+++	+++	+++	+++	++	++	±	±	—
X	X	+	++++	+++	++++	+++	++++	++++	++++	++++	+++	+++	—
	DG	±	+	++	++	+++	++++	+++	+++	++	++	+++	—
	SE	±	+	+	++++	+++	+++	++++	+++	+++	+++	+++	—

* Control

TABLE 3—Results of Reciprocal Agglutinin Absorption Tests with Strains DG and X

Anti serum	Absorbed with Strain	Strain Used in Agglutination Test	Agglutinin Titer Before Absorption	Agglutinin Titer After Absorption					
				1 40	1 80	1 160	1 320	1 640	1 1,280
DG	DG	DG	20,480 +++++	—	—	—	—	—	—
	X	DG		—	—	—	—	—	—
X	{	{	20,480 +	1 30	1 60	1 120	1 240	1 480	1 960
				±	±	—	—	—	—
				±	—	—	—	—	—
				±	—	—	—	—	—

TABLE 4—Results of Precipitin Tests With Antiserums and Hydrochloric Acid Extracts of Representative Strains

Antiserum	Hydrochloric Acid Extract of Strain	Amount of Extract, Cc			
		0.4	0.01	0.025	0.001
DG	X	+++	++++	+++	+++
	DG	++++	++++	+++	+
SE	X	+++	+++	+++	+++
	DG	+++	+++	+++	+++
X	X	++++	++++	+++	++
	DG	++++	++++	+++	+++

Controls of serum and of extracts negative

++++ indicates a precipitate composed of disks, +++, one composed of disks which were easily broken up, ++, one composed of large flakes, and +, one composed of small flakes

examined after incubation for two hours at 37 C and refrigeration for eighteen hours The results shown in table 4 again indicate the serologic identity of the 3 strains

14 Lancefield, R. C. The Antigenic Complex of *Streptococcus Haemolyticus* II. Chemical and Immunological Properties of the Protein Fractions, *J. Exper. Med.* **47**: 469, 1928, III. Chemical and Immunological Properties of the Species-Specific Substance, *ibid.* **47**: 481, 1928

In the experimental case an attempt was made to determine whether any systemic immunity was produced by the conjunctival infection. The patient's serum, withdrawn four weeks after the initial infection, failed to demonstrate agglutinins for homologous or heterologous strains.

EPIDEMIOLOGIC STUDY

One aspect of the epidemiologic study dealt with the spread of conjunctivitis in the local outbreak. The housing facilities consisted of eighteen bungalows, arranged in a semicircle, with about 20 feet (6 meters) between adjacent buildings. Each bungalow had a toilet and a washbasin with running water (from an artesian well) and accommodated 6 or 7 campers. The windows were unscreened.

Because the infectiousness of conjunctivitis was recognized, methods of isolation were introduced and subsequently modified twice. During the first period, from July 11 to 27, inclusive (I, table 5), the patients were segregated during the day but permitted to return to their own bunks at night. They were cautioned to avoid contacts. In the second period, from July 28 to August 2, inclusive (II, table 5), all patients were kept day and night in an isolation bungalow, which, unfortunately, was located in the middle of the row of bungalows. During both these periods the boys ate at a separate table in the common mess hall. Undoubtedly, numerous breaks in isolation occurred during these periods. The third period, from August 3 to 31, inclusive (III, table 5), was one of strict isolation, and all patients were held incommunicado in a strictly isolated bungalow. Meals were served in the bungalow, separate dishes were used, and no external contacts were permitted.

Even with strict isolation of every known patient, new cases appeared at irregular intervals. When, however, each new case was charted in relation to the other cases, there was such scattering that it was impossible to determine the source of infection. While there were several instances in which infections appeared in a group in the same bungalow, these were a small minority of the series. The appearance of the disease in these new cases made us suspect that an unknown person or persons harbored the infecting agent in a focus other than the eye—possibly in the paranasal sinuses, the nose or the throat.

It does not seem likely that bathing water played a significant role, since conjunctivitis developed in none of a large group of employees using the lake. Moreover, a boy who never went swimming was infected. In the presence of many insect vectors and without adequate screening, transmission by insects could not be excluded.

To determine the geographic distribution of this outbreak, a questionnaire was submitted to the directors of camps throughout New England, New York and Pennsylvania. Five camps within a radius of 50 miles (80 kilometers) from the camp studied reported cases in vary-

TABLE 5.—Distribution of Cases Throughout Epidemic

[illegible]

Letters indicate initials of patient, numbers, duration in days, and parentheses around initials, recurrence

ing numbers. No path of spread could be demonstrated between the camp in Pennsylvania and the camp in Vermont from which a case has been included in this report.

COMMENT

Previous reports have indicated that green streptococci may be present in the normal conjunctiva and may be associated with acute conjunctivitis of either sporadic or epidemic nature. Our experience, which we believe to be the first of its kind reported in this country, lends further support to the idea that *Str. viridans* may be one of the causes of epidemic conjunctivitis.

Although one of Koch's postulates has not been met—i. e., pure cultures of the micro-organisms isolated did not cause conjunctivitis in experimental animals—the presence of the cocci in pure culture in the eyes of all infected persons and their absence in 15 normal controls is strong evidence that they played some causal role. Unfortunately, experiments with filtered exudates could not be performed at the camp, therefore, the possibility that the primary agent was a virus could not be excluded. However, the short period of incubation is against such a possibility, and there seems little doubt that the streptococci played an important part and acted at least as symbiotic if not as sole agents.

Whether or not a virus was involved, the demonstration of the cocci was of practical interest, because their resemblance to pneumococci in direct smears makes apparent the danger of an incorrect etiologic diagnosis. Prior to cultural differentiation we falsely considered the conjunctivitis in our cases to be due to pneumococci. Similarly, the patient who contracted the disease during the epidemic in Vermont and from whom cultured material yielded an identical streptococcus was thought to have a pneumococcic infection and was so treated. It would seem entirely possible that in many cases so-called pneumococcic conjunctivitis has been due, in reality, to *Str. viridans*—a belief which has been expressed by Schmelzer and Eckstein.

Finally, the serologic identity of the strains from the epidemic in Pennsylvania and that in Vermont warrants comment. So far as we could determine, there was no human contact between the two camps. If there was none, the identity of these strains in such a heterogeneous group as the green streptococci is most remarkable and raises the possibility that this particular serologic type is especially apt to cause or to be associated with epidemic conjunctivitis.

SUMMARY

An epidemic of 35 cases of acute conjunctivitis in a boys' camp is described. The infection apparently was caused by *Str. viridans*.

The disease was of purulent type and of varying severity and had a period of incubation of from sixteen to twenty-four hours and an average duration of seven days

No method of chemotherapy employed appeared to be of any value in altering the course of the disease

There was no significant complication

Direct smears of material from the conjunctiva in every case revealed pus cells and diplococci morphologically similar to pneumococci. In the 7 cases in which cultural study was performed, *St. viridans* was recovered. A similar organism was isolated from material from a patient who had the disease during an epidemic in Vermont.

Immunologic identity was demonstrated between 2 strains recovered from the epidemic in Pennsylvania and 1 from the epidemic in Vermont.

The possible etiologic and epidemiologic significance of the microorganisms isolated is discussed.

INFLUENCE OF VITAMINS AND DINITROPHENOL ON THE PRODUCTION OF EXPERIMENTAL CATARACT

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In recent years investigations of the causation of cataract have largely turned from the clinical and the anatomic to the experimental methods of physiologic and biochemical investigation as applied to the lens. With the invention of the slit lamp in 1911 by Gullstrand, the anatomic field for the exploration of cataract was opened, and the extensive and detailed reports which have followed in regard to opacities of the lens of all types form a fairly useful classification of cataract on a morphologic basis. The experimental approach to the study of cataract has been more or less hampered, however, by an inability to produce cataract in animals with any degree of constancy. Naphthalene has been used to produce cataract, but it is inconstant in its action on the lens, and it is well known that other ocular tissues show changes after the administration of this chemical compound. Fortunately, a more satisfactory method for the production of cataract in laboratory animals has been evolved through the work of Mitchell and Dodge and of Yudkin and Arnold.

The present investigation was undertaken in an effort to reproduce, if possible, cataracts such as those reported in a group of obese patients who had taken dinitrophenol for reducing purposes. Horner, Jones and Boardman¹ first reported the association of cataract and dinitrophenol in 1935, and since that time other similar reports have appeared. In a more recent paper Horner² gave an excellent review of the subject from the clinical point of view. In a paper by H. Barkan and his co-workers³

Supported, in part, by grants from the Rockefeller Fluid Research Fund of the School of Medicine, Stanford University

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1 Horner, W D, Jones, R B, and Boardman, W W. Cataracts Following the Use of Dinitrophenol. Preliminary Report of Three Cases, *J A M A* **105** 108 (July 13) 1935

2 Horner, W D. Cataract Following Di-Nitrophenol Treatment for Obesity, *Arch Ophth* **16** 447 (Sept) 1936

3 Barkan, H, Borley, W E, Fine, M, and Bettman, J. *California & West Med* **44** 360, 1936

is included a description of the clinical operative procedures and results in a series of 20 cases of cataract which developed coincident with dinitrophenol therapy. From clinical observation, these patients were known to have capsular and subcapsular changes in the lenses, with differences in refractive errors tending toward increased hyperopia. This suggested the possibility of changes in the permeability of the capsule. We therefore tested the influence of dinitrophenol on the permeability of the capsules of beef lenses *in vitro* and of rat lenses *in vivo*, using variable concentrations of the drug, but our results were entirely negative.⁴ Consequently, simple changes in the permeability of the capsule appeared unpromising as an explanation of the clinical cataracts.

In view of the fact that the patients with cataract were obese, dietary restriction was considered as a possible factor, activated in some way by the administration of dinitrophenol. It is well known that the incidence of cataract in these patients did not parallel the dose of dinitrophenol or the reduction in body weight. It is conceivable that they may have suffered from various degrees of vitamin deficiency, which might have predisposed them to the production of cataract by the dinitrophenol. The work of Day, Langston and O'Brien⁵ and of many others on the production of cataract in albino rats on a diet free from vitamin B₂ (G) has proved the close relation of nutrition and changes in the lens.

Therefore, the possibility that vitamin deficiency combined with the administration of dinitrophenol could lead to cataractous changes was tested experimentally in white rats and guinea pigs, under conditions designed to show any cataract-producing propensities of dinitrophenol, if such existed. The results are presented in this paper. In a subsequent paper the influence of dinitrophenol on the cataractous processes produced by diets high in lactose or carbohydrates will be discussed.

In view of the absence of changes in the lens in experimental animals receiving dinitrophenol during their entire life span,⁶ as shown by early researches with this drug, it was found necessary to attack the problem from a somewhat different standpoint than is ordinarily followed in testing the toxic effects of a drug.

In essence, the procedure used consisted in creating in experimental animals by means of diet vitamin A, B₂ or C deficiency, conditions which have been associated with the formation of cataracts or other

4 Borley, W. E., and Tainter, M. L. Effects of Dinitrophenol on the Permeability of the Capsule of the Lens, *Arch. Ophth.* **18** 908 (Dec.) 1937.

5 Day, P. L., Langston, W. C., and O'Brien, C. S. *Am. J. Ophth.* **14** 1005, 1931.

6 Tainter, M. L. *J. Pharmacol. & Exper. Therap.* **63** 51, 1938.

lenticular changes, and then determining whether or not the addition of dinitrophenol to the diet led to a greater tendency to the production of cataractous changes. The details of the methods are given in connection with a discussion of the results with each type of vitamin deficiency.

VITAMIN A DEFICIENCY

Young albino rats ranging in weight from 32 to 46 Gm, with an average weight of 36 Gm, were used. They had been reared on the following normal laboratory diet:

	Parts
Corn meal	292
Linseed oil cake meal	40
Ground alfalfa	8
Crude casein	40
Cod liver oil	13
Bone ash	6
Sodium chloride	2

In the first group 4 litter mate pairs were placed in two cages, so as to have one cage of 4 rats as a control on the other 4 rats which received dinitrophenol. In a second group, studied at a different time for confirmation, the same arrangement was adhered to, so that a total of 16 rats were used for this phase of the work. All the rats were then fed the special vitamin A-free diet of Yudkin,⁷ special casein freed of its vitamin A content by the method of Sherman and Smith⁸ being used. This diet had the following composition:

	Parts
Casein (free from vitamin A)	18
Hydrogenated oil	15
Osborne and Mendel's salt mixture	4
Dried irradiated brewers' yeast	10
Corn starch	53

The vitamin D requirements were met by using irradiated yeast rather than by giving supplemental viosterol. Alphadinitrophenol (1, 2, 4) in a concentration of 0.1 per cent was added to this diet, which was fed to the rats of one of the cages of each group. This has been demonstrated to be about the maximum concentration of dinitrophenol tolerated by rats in chronic feeding experiments⁶ and represents a considerably greater amount proportionately than that ever used clinically. The rats were fed these diets ad libitum, from special glass feeders previously

⁷ Yudkin, A. M. Ocular Disturbances Produced in Experimental Animals by Dietary Changes. Clinical Implications, J. A. M. A. **101** 921 (Sept. 16) 1933.

⁸ Sherman, H. C., and Smith, S. L. The Vitamins, ed. 2, New York, Chemical Catalog Company, Inc., 1931, p. 275.

described,⁹ and were weighed three times weekly. Since the results for both groups were identical, they will be described together.

The 8 control rats continued to gain in body weight at a decreasing rate for about forty days, when they began to lose weight. They died in an average of ninety-three days with definite signs and symptoms of photophobia, xerophthalmia, loss of hair and emaciation. The typical condition of chronic vitamin A deficiency was produced. The 8 rats receiving dinitrophenol grew much more slowly than their controls and reached an average maximum weight of only about 60 Gm. before they began to lose weight and died. This was undoubtedly due to the high intake of dinitrophenol. They died in an average of fifty-eight days and showed the same characteristic symptoms of vitamin A deficiency as the controls.

Careful examination of the eyes of all these rats every second day throughout the experiment with the routine use of the ophthalmoscope supplemented with the slit lamp failed to show any deviations from normal in the lenses. Therefore, dinitrophenol failed to produce any demonstrable damage in the lens of these rats under conditions of fatal vitamin A deficiency.

VITAMIN B₂ (G) DEFICIENCY

Eight young albino rats with body weights ranging from 30 to 38 Gm. were placed in two cages and put on a diet deficient in vitamin B₂ (G). To the diets of the rats in one cage, 0.1 per cent dinitrophenol was added. At the termination of the experiment with this group, a second similar group of 5 rats in each cage was carried through the same procedure. Thus 9 rats were used as controls, and 9 rats received dinitrophenol added to their diet; the diet for the entire group was deficient in vitamin B₂. The diet was prepared by the usual method of using casein free from vitamins and supplying the B₁ fraction from rice polish extract. The casein was freed of all vitamin B portions, including the B₂ or G, by repeated extraction with 60 and 90 per cent alcohol, according to the methods of Sherman and Spohn¹⁰. The B₁ portion was then replaced by rice polish extract made according to the method of Day and Langston¹¹. The complete diet as prepared consisted of the following:

	Parts
Casein (free from vitamin B ₂ [G])	18
Osborne and Mendel's salt mixture	4
Butterfat	8
Cod liver oil	2
Rice polish extract (free from vitamin B ₂ [G])	5
Corn starch	63

⁹ Tainter, M. L. *Proc. Soc. Exper. Biol. & Med.* **30**:1234, 1933.

¹⁰ Sherman, H. C., and Spohn, A. *J. Am. Chem. Soc.* **45**:2719, 1923.

¹¹ Day, P. L., and Langston, W. C. *J. Nutrition* **7**:97, 1934.

Since the responses of both groups were similar, the results were combined for purposes of description. The control group of rats survived to a median of sixty-eight days, with an average rate of gain in body weight of 0.15 Gm daily for the entire period. One rat of this control group was alive at the end of one hundred and seventy-three days, when the experiment was stopped. The 9 rats receiving 0.1 per cent dinitrophenol in the diet survived to a median of seventy-seven days, 1 rat being alive on the one hundred and seventy-third day, when the experiment was stopped. In the latter rats there was an average loss of weight of 0.05 Gm per day. The diet deficient in vitamin content, therefore, stopped the growth of the animals almost completely and finally killed them in a little over two months. Careful examinations of the eyes of all these rats were made, as in the series fed the diet free from vitamin A. There were no observable lenticular opacities in any of these rats, with or without the administration of dinitrophenol, and the lenses of the rats surviving one hundred and seventy-three days remained clear. In other words, a maximum tolerated daily intake of dinitrophenol produced no detectable changes in the lenses of these rats during fatal vitamin B₂ deficiency.

VITAMIN C DEFICIENCY

Sixteen guinea pigs ranging in body weight from 320 to 424 Gm were used to study the possibility of dinitrophenol affecting the lens in the absence of adequate supplies of vitamin C. These were used in two different groups of 8 guinea pigs each. In each experiment the 8 guinea pigs were divided into a group of 4 which served as controls and a group of 4 which received 0.2 per cent dinitrophenol in the diet. The greater concentration of the drug was selected for these scorbutic animals because of the greater resistance of guinea pigs to dinitrophenol. They were all placed on a diet free from vitamin C, prepared from autoclaved soy bean meal after the method of Koch and Smith¹². The composition of the diet was as follows:

	Parts
Soy bean meal (autoclaved)	86
Dried yeast	3
Sodium chloride	3
Calcium lactate	3
Cod liver oil	5

Both groups of guinea pigs responded similarly and are discussed together. All guinea pigs in the control group died within a period of from twenty-one to thirty days with well marked signs of scurvy. All

¹² Koch, M. L., and Smith, A. H. *Proc Soc Exper Biol & Med* **21** 366, 1924.

guinea pigs in the group receiving dinitrophenol showed similar lesions and died between the twenty-first and the thirty-seventh day. Careful study of their eyes during life, as in the other experiments, and at autopsy failed to show any evidence of lenticular changes. It was therefore concluded that dinitrophenol failed to produce any demonstrable changes in the lenses of guinea pigs during fatal vitamin C deficiency.

DINITROCHLOROBENZENE AND TOLUQUINONE

Another possibility to be considered was that there was a trace of some impurity in the dinitrophenol taken by the patients in whom cataracts developed which was responsible for the changes in the lens rather than the dinitrophenol. The only chemical impurity which would have much chance of being present was the intermediate product, dinitrochlorobenzene. Chemical studies of the important commercial brands of dinitrophenol revealed only traces of a chloride compound. Nevertheless, dinitrochlorobenzene was added to the diet of a group of rats fed on the aforementioned normal diet to determine its possible effects on the eyes.

Concentrations of 1 per cent dinitrochlorobenzene in the diet were found to be irritating to the tongue and caused the death of 5 rats in twenty-two days. Lower concentrations appeared to be innocuous. None of the rats receiving this compound showed any changes in the lenses. Therefore, there is little or no likelihood that any minute trace of this chemical, such as might be present in a clinical dose of dinitrophenol, could have any deleterious effect.

Almost all patients in whom cataract developed after the use of dinitrophenol were women. Women are particularly exposed to the drugs contained in contraceptives. One patient was seen with a clinical picture of cataract indistinguishable from that of cataract associated with the use of dinitrophenol, yet she had never taken dinitrophenol or any other medication for many years. The only drugs to which she was exposed were those contained in a popular proprietary contraceptive product. Investigation revealed that the active ingredient of this product was toluquinone, a spermicide. Therefore, the possible effects of this drug on the eyes was investigated experimentally.

Two different groups of 6 rats each were fed 0.1 and 0.5 per cent toluquinone in the stock diet. When the lower concentration was used all the rats gained steadily in body weight until the experiment was terminated in one hundred and thirty-five days. When the higher concentration was used 5 rats died in sixteen days, while 1 rat survived until the experiment was terminated in one hundred and seventy-three days. In none of these 12 rats did repeated ophthalmoscopic examinations reveal any alterations in the eye or lens. Therefore, no evidence was found which would indicate any causative role for toluquinone in the production of experimental cataract.

SUMMARY AND CONCLUSIONS

Rats fed diets deficient in vitamin A and vitamin B₂ (G) and containing dinitrophenol in 0.1 per cent concentration failed to show any changes in the lens which could be associated with the production of clinical cataract.

Guinea pigs fed a diet deficient in vitamin C and containing dinitrophenol in a 0.2 per cent concentration similarly showed no detectable changes in the lenses.

Accordingly, no experimental evidence was found that dinitrophenol could produce cataractous changes in the lens when the drug was given in maximum tolerated concentrations to animals suffering from severe or fatal deficiencies of vitamin A, B₂ or C.

Dinitrochlorobenzene, a theoretically possible contaminant of commercial dinitrophenol, and toluquinone, the sole compound to which 1 patient with cataract was exposed, were both fed to rats without producing demonstrable lenticular changes.

Therefore, there was a uniform failure to produce by experimental means in animals cataracts similar to those reported as due to medication with dinitrophenol in human beings.

ABSORPTION OF VISIBLE LIGHT BY THE REFRACTIVE MEDIA OF THE HUMAN EYE

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Quantitative data on the absorption of visible light by the refractive media of the human eye¹ would seem to be important from many points of view. Thus the data may be used for (1) the correction of the photopic ocular visibility curve to a photopic retinal visibility curve, (2) the correction of a scotopic ocular visibility curve to a scotopic retinal visibility curve, (3) a comparison of the absorption spectrum of visual purple with the sensitivity of the eye and (4) the evaluation of various facts and theories concerning vision and color vision. Since an examination of the literature indicated that there were no adequate data available on the absorption spectrum of the ocular media for light of visible wavelengths, we decided that these data should be obtained. Accordingly, about three years ago we set up apparatus, with the aid of which we have determined the combined absorption characteristics in the visible spectrum from 400 to 820 millimicrons of the refractive media of 4 whole human eyes while the media were in situ in the eyes. These eyes were normal from the point of view of their optical characteristics. We have determined separately the absorption characteristics of the various component parts of these eyes (the cornea, aqueous, vitreous and lens) and also the absorption characteristics of 11 normal and 42 cataractous lenses. The purpose of the present paper is to present a description of the apparatus and the method of procedure and to give the results of our measurements on the absorption of visible light by the ocular media.

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1 By this phrase we mean any difference of total luminous flux arising between the external surface of the cornea and the internal surface of that portion of the retina on which the image of the source would normally be formed. In addition to true absorption, this difference may be due to irregular refraction, regular and irregular reflection, fluorescence and diffraction. By visible light we mean radiant energy in the wavelength range between 400 and 820 millimicrons. By refractive media we mean the cornea, aqueous, vitreous and lens.

Many investigations have been made of the absorption of infra-red and ultraviolet rays by the human ocular media, probably because of the possible pathogenic significance of light of such wavelengths. Numerous references to these investigations may be found in the bibliography of the article by Verhoeff, Bell and Walker² on the pathologic effects of radiant energy on the eye. As far as the absorption of rays in the visible range of the spectrum is concerned, however, adequate data are lacking. Nevertheless, it appears to have been generally assumed that the absorption of the human ocular media for this wavelength range is equivalent to that of an equal thickness of water. It is interesting to trace the origin of this assumption. It is evidently based on the work of Aschkinass,³ published in 1895, which we shall now briefly consider. Aschkinass determined the absorption spectrum of water in the extreme red end and in the infra-red portion of the spectrum. He then made similar investigations concerning the absorption of the ocular media of cattle. The cattle had been slaughtered about twenty hours before the necessary measurements were made. The absorption spectrums of the cornea, aqueous, vitreous and lens were determined separately. The transmission values for the combined media in situ were not determined. He observed that the aqueous transmitted less than an equal thickness of water but noted that the aqueous was cloudy, he accordingly assumed that if it had been clear its absorption spectrum would have been identical with that of water. Aschkinass' energy-measuring device was a thermopile, and he made no measurements on the ocular media below wavelengths shorter than 670 millimicrons. Since his measurements even in the extreme red end of the spectrum, beyond 670 millimicrons, do not agree with ours, we shall discuss his method somewhat.

As Aschkinass pointed out, he rather consistently obtained transmission values of above 100 per cent for the crystalline lens of cattle. Although Aschkinass is not completely clear in his explanation for the cause of the phenomenon it appears, as he stated himself, that he "obtained up to the angle of deflection $\Delta = 3^{\circ} 15'$ less deflection [of the galvanometer] in the direct radiation than after insertion of the lens." In other words, his results show negative absorption on the part of the lens or transmission values of greater than 100 per cent for some portions of the spectrum. He attributed this to the fact that the crystalline lens was pressed between two glass plates and stated that "on

2 Verhoeff, F. H., Bell, L., and Walker, C. B. The Pathological Effects of Radiant Energy on the Eye, *Proc. Am. Acad. Arts & Sc.* **51** 630, 1916.

3 Aschkinass, E. Ueber das Absorptionsspectrum des flüssigen Wassers und über die Durchlässigkeit der Augenmedien für rote und ultrarote Strahlen, *Ann. d. Phys. u. Chem.* **55** 401, 1895.

account of the characteristic structure of the lens, the concentrating effect of it was not completely kept." Two possibilities appear (1) that forcing the lens between the plates resulted in diffusion and that "loss of concentrating effect" means diffusion, or (2) that placing the lens between the plates appeared, as would be expected from the structure of the lens, not totally to eliminate its dioptric power, so that the size of the beam hitting the bolometer was smaller than it would have been if the lens had not been placed between the plates. As to the first possibility, it is difficult to see how this could result in the apparent transmission of the lens being greater than 100 per cent. The second possibility, however, could result in an apparent transmission of over 100 per cent under two possible conditions (1) if the rays of light when the lens was not in the beam were cut off by a diaphragm stop somewhere between the bolometer and the lens receptacle, or (2) if the galvanometer deflection was a function not only of the total energy of radiation but of the area of the bolometer exposed to the radiation. In any case, the existence of transmission values greater than 100 per cent is physically impossible, as Aschkinass realized. He therefore attempted to correct the data in the following fashion. He noted that the transmission values of the crystalline lens, including those over 100 per cent, were similar to those of a layer of water of equal thickness, except that transmission values for the lens were consistently higher. What he did, essentially, was to select two transmission values of less than 100 per cent, one, determined for water, and a second, for the crystalline lens. He then divided the percentage transmission of water at this wavelength by the apparent percentage transmission of the crystalline lens at this wavelength and obtained a correction factor, m , with a value less than unity. He multiplied all the values for the transmission of the lens by the fraction m , thus reducing the transmission values above 100 per cent to 100 per cent or less. In the absence of any showing as to what the phenomenon of observed transmission above 100 per cent is due, this mode of correction is clearly solely of an empiric nature, and, as Aschkinass himself stated, "There is, to be sure, a certain arbitrariness in the method."

Aschkinass next devoted a paragraph to his investigations on the human eye. Whether or not the eyes he used were obtained post mortem is not stated. There is no mention of the condition of the eyes or of the degree of freshness of the material. Aschkinass' paper contains no experimental data on these eyes, but he stated that his results are in complete agreement with his measurements on the eyes of cattle. If this complete agreement means that Aschkinass encountered the same difficulties due to cloudy media and transmission values above 100 per cent in human eyes that he did in the eyes of cattle, the significance of

his measurements on the former is dubious. In a subsequent paper by Aschkinass⁴ there is a graph showing the calculated absorption of a layer of water 2.28 cm thick, the axial length of the human eye. The graph does not extend below 670 millimicrons. To summarize the situation, the absorption values given are for the eyes of cattle, the wavelength range used scarcely entered the visible spectrum, the ocular media were not normal, and the final values depended on an assumed correction. In other words, it can hardly be said that Aschkinass determined the absorption of visible light by the normal human eye at all.

Luckiesh,⁵ relying on the investigation of Aschkinass, stated "He [Aschkinass] found that the various eye-media transmitted the visible and infra-red rays in the same manner as like thicknesses of water." Using the data which had been obtained for water, Luckiesh calculated what the absorption of visible light by the eye would be if it were equivalent to that of a layer of water 2.28 cm thick.

Belief in the assumption that the absorption spectrum of the human ocular media may be derived from that of water by appropriate calculations appears to be general. Thus Troland⁶ accepted the truth of this assumption when he corrected the photopic ocular visibility curve for the absorption of the macula lutea and the ocular media in order to derive a photopic retinal visibility curve. Even in the recent text-book of Duke-Elder⁷ reliance is placed on the investigation of Luckiesh,⁸ and the following statement is made: "The transmission of the ocular media is closely similar to that of a layer of water of equal thickness, only a small portion of the visible spectrum (about 8 per cent, Luckiesh, 1913) is absorbed."

The only data of which we are aware which can be used for the purpose of comparison with our results are those of Roggenbau and Wetthauer. These authors have been concerned mainly with the absorption of the ocular media in the infra-red portion of the spectrum.⁹ In

4 Aschkinass, E. Spektrolometrische Untersuchungen über die Durchlässigkeit der Augenmedien für rote und ultrarote Strahlen, *Ztschr f Psychol u Physiol d Sinnesorg* **11** 44, 1896.

5 Luckiesh, M. Infra-Red Radiant Energy and the Eye, *Am J Physiol Optics* **2** 3, 1921.

6 Troland, L. The Retinal Visibility Function, *Abstr-Bull Phys Lab Nat Electric Lamp A* **1** 378, 1913.

7 Duke-Elder, W. S. Textbook of Ophthalmology, London, Henry Kimpton, 1932, vol. 1, p. 809.

8 Luckiesh, M. Radiant Energy and the Eye, *Electric World* **62** 1160, 1913.

9 Roggenbau, C., and Wetthauer, A. Zur Frage der Erwärmbarkeit der einzelnen licht-brechenden Teil des Auges nach Bestrahlung durch einen leuchtenden Körper, *Ztschr f Augenh* **64** 143, 1928.

one paper,¹⁰ however, they presented a graph showing a continuous curve running through the visible spectrum. The graph was unaccompanied by specific figures for the transmission in the visible spectrum. Their measurements in the infra-red portion of the spectrum, and hence presumably in the visible spectrum, were made on the eyes of cattle. They did not state, and we have been unable to ascertain, on what data for the visible spectrum this histogram was based. Since, however, their histogram is the only material even analogous to ours at our disposal, we shall present a graph in which their results are compared with ours.

Occasionally we have found that measurements made by other observers on portions of the eye, although mainly concerned with the infra-red portion of the spectrum, have given some results in the long wave end of the visible spectrum.¹¹ It suffices here to say that so far as comparisons can be made these measurements agree with ours in showing a uniform fairly high transmission in the visible extreme red end of the spectrum. There have also been some authors¹² who have

10 Roggenbau, C, and Wetthauer, A. Ueber die Durchlassigkeit der brechenden Augenmedien für langweiliges Licht nach Untersuchungen am Rindsauge, *Klin Monatsbl f Augenh* **79** 458, 1927

11 (a) Bucklers, M. Zur Absorption des sichtbaren Spektralabschnittes durch die menschliche Linse, *Arch f Augenh* **108** 479, 1934. (b) Hartridge, H, and Hill, A. The Transmission of Infra-Red Rays by the Media of the Eye and the Transmission of Radiant Energy by Crookes and Other Glasses, *Proc Roy Soc, London, s B* **89** 58, 1917. (c) Brucke, E. Ueber das Verhalten der optischen Medien des Auges gegen Licht- und Wärmestrahlen, *Arch f Anat, Physiol u wissensch Med*, 1845, p 262. (d) Klug, F. Ueber die Diathermansie der Augenmedien, *Arch f Physiol* **2** 246, 1878. (e) Schlapfer, H. Experimentelle Untersuchungen über die Absorption des Ultrarot durch Kammerwasser, Linse, und Glaskörper des Rindes, *Arch f Ophth* **119** 22, 1928. (f) Vogt, A. Experimentelle Untersuchungen über die Durchlassigkeit der durchsichtigen Medien des Auges für das Ultrarot künstlicher Lichtquellen, *ibid* **81** 155, 1912. (g) Berner, I. Experimentelle Untersuchungen über die Absorption des Ultrarot durch Linsen von alten Pferden und Kühen, *ibid* **119** 368, 1928. (h) Mandach, E. Experimentelle Untersuchungen über Absorption des Ultrarot durch die Hornhaut des Rindes, *ibid* **119** 361, 1928. (i) Roggenbau and Wetthauer (footnotes 9 and 10).

12 (a) de Chardonnet, E. Pénétration des radiations actiniques dans l'oeil de l'homme et des animaux vertébrés, *Compt rend Acad d sc* **96** 441, 1883. (b) Vision des radiations ultraviolettes, *ibid* **96** 509, 1883. (c) Gayet, A. Sur le pouvoir absorbant du cristallin pour les rayons ultraviolets, *Bull et mem Soc franç d'opht* **2** 188, 1884. (d) Glancey, A. Limit of Visibility in the Ultra-Violet, *Am J Physiol Optics* **4** 145, 1923. (e) Graham, W. The Absorption of the Eye for Ultraviolet Radiation, *ibid* **4** 152, 1923. (f) Hallauer, O. Ueber die Absorption von kurzwelligem Licht durch die menschliche Linse, *Klin Monatsbl f Augenh* **47** 721, 1909. (g) Hoffmann, W. Zur Messung der Ultraviolett-absorption von Hornhaut und Linse am lebenden Auge, *Ztschr f Augenh* **63**

made determinations of the limits of transmission of the ocular media for the short wave end of the spectrum. A method commonly used is that of photographing a line spectrum through the ocular media and noting which is the last line in the violet or the ultraviolet portion of the spectrum appearing in the photograph. This method, of course, gives only qualitative results. So far as we are able to compare our quantitative results with these determinations, they appear to be in reasonably good agreement.

APPARATUS

A general view of the apparatus used by us is shown in figure 1. The arrangement of the optical parts may be seen in figure 2. The light source, S , was a 6 volt, 18 ampere coil filament lamp which was maintained at 18 amperes by the use of a rheostat. The source was enclosed in a ventilated box with two round apertures, S_1 and S_2 , of a radius of 1.5 cm. Jena glass filters could be inserted at F_1 and F_2 . Light from S_1 was focused by means of the achromatic lens, L_1 , on a circular aperture, S_3 , 0.5 mm in radius, and then diverged out into a box 117 cm long which was lined with black velvet. Within this box was a sandwich type of barrier layer or blocking layer photocell, P_1 . This photocell could be moved with respect to aperture S_3 , which now constituted the effective source. The photocell was supplied with louvers, S_4 . The output of the photocell was fed directly and without amplification into a microammeter. Light from aperture S_2 was gathered by an achromatic condensing lens, L_2 , and focused on the entrance slit, S_5 , of a spectrometer which will be described later. The light emerging from the exit slit, S_6 , was reflected upward by a prism of 45 degrees, P_1 , to the second photocell, P_2 . This photocell was also supplied with louvers, S_7 . The output of the second photocell was fed directly into the microammeter in such a fashion as to cause a deflection opposite in direction to that due to the first photocell. Containers, C , of various types could be introduced between prism P_1 and the second photocell. These containers were designed to hold aqueous, vitreous, cornea or lens or the whole eye. In the case of the cornea lens or whole eye, the surfaces were wetted by contact with felt from which fatty material was removed.

38, 1927. (h) Schanz, F. Ueber die Veränderungen und Schädigungen der Augen durch die nicht direkt sichtbaren Lichtstrahlen, *Arch f Ophth* **86** 549, 1913, (i) Der Gehalt des Lichts an Ultraviolett, *ibid* **103** 158, 1920. (j) Shoji, Y. Untersuchung über die Absorption der ultravioletten Strahlen durch die Augenmedien, *Mitt a d med Fakult d k Univ zu Tokyo* **29** 61, 1922, (k) Etude photo-chimique de l'absorption des rayons ultraviolets a travers les milieux oculaires, *Ann d'ocul* **160** 356, 1923. (l) Takamine, T., and Takei, S. Ueber das Verhalten der durchsichtigen Augenmedien gegen ultraviolette Strahlen, *Arch f d ges Physiol* **149** 379, 1912.

by the use of ether and which was moistened with physiologic solution of sodium chloride (fig 3) Figure 3 is a photograph of the container for the whole eye At first a drip method was used to prevent the surfaces from drying, but a gradual drift of about 2 per cent was observed in the results This drift was correlated with the formation of

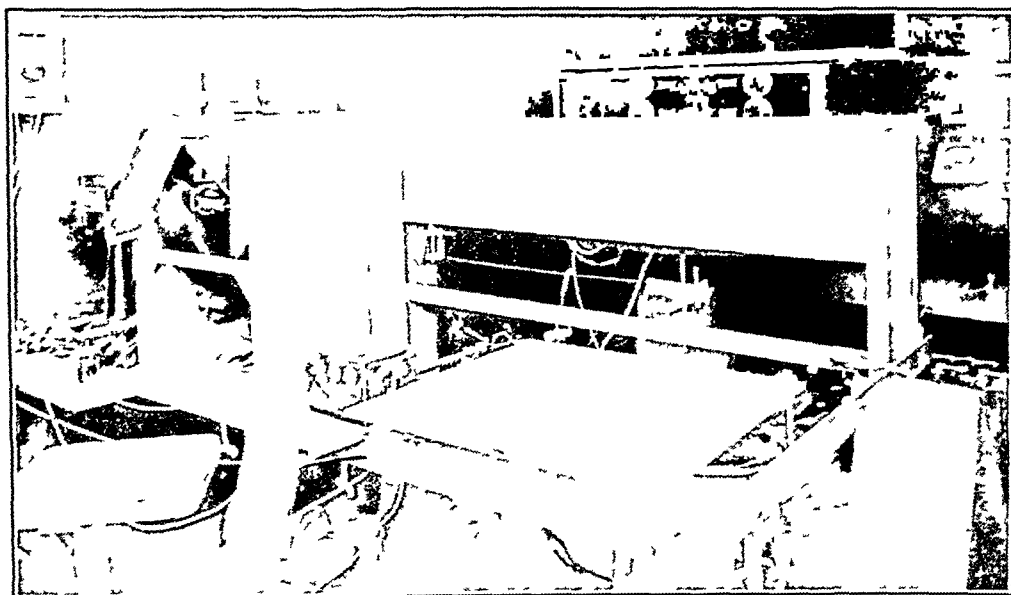


Fig 1—General view of the apparatus used

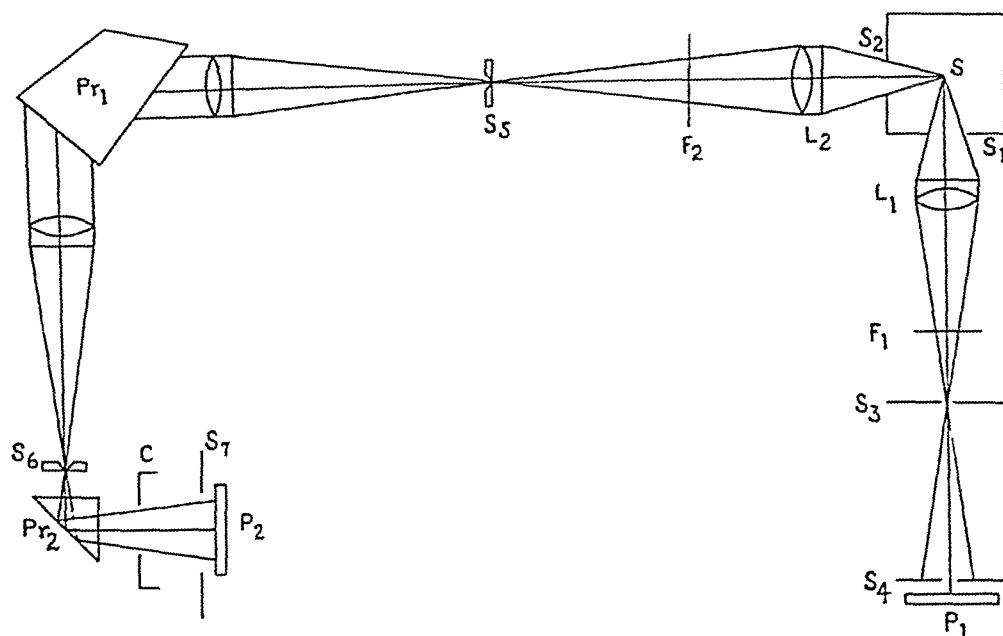


Fig 2—Schematic diagram showing optical system of the apparatus

a drop of solution When there was substituted for the drip method the use of moistened felt, no such drift was observed At first we attempted to utilize Weston 594 photronic cells in conjunction with a Weston model 440 microammeter The results were consistent, but the sensitivity in the blue and violet portions of the spectrum in which we were particularly

interested, was not wholly satisfactory owing to the nature of the relative energy distribution of the light source and the spectral response curve of the photocell. We therefore substituted for the Weston photocells others with a considerably greater current output and had the Weston Company supply us with a special microammeter requiring 7×10^{-8} amperes for unit deflection.

For the production of relatively monochromatic light, we used a Gaertner, L-231, constant deviation spectrometer. Over a period of several months, this instrument was calibrated and partially rebuilt. The refractive index, dispersion and refractive angle of prism P_1 , were determined. The rate of tarnishing of the prism was also determined. The slit jaws, S_5 and S_6 , were removed and refinished, and the adjusting mechanisms for the jaws were rebuilt. Hartmann's formula for dispersion was applied, and the results were experimentally checked. The

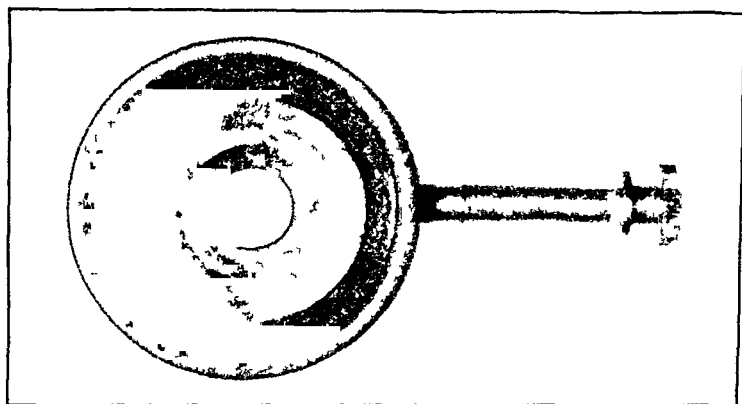


Fig 3—Container for eye (one-half actual size)

widths of the slits were calibrated for various readings of the adjusting mechanisms, and the amount of scattered light from the spectrometer was determined. Louvers, not shown in figure 2, were then fitted so as to reduce stray light. The instrument was again tested for the emission of stray light at the exit slit, and it was found that under the conditions prevailing when we determined the absorption characteristics of the ocular media the amount of stray light remaining was insufficient to cause a noticeable deflection of the galvanometer.

Kranz¹³ reported the existence of doubly refractive lipoids in cataractous lenses. His investigation on 8 normal lenses from patients aged from $1\frac{1}{2}$ to 60 years revealed no doubly refractive lipoids. Since some of our experiments, although not those reported here, were performed on cataractous lenses, the photocells were tested in order to determine the effect of polarized light on them. The effect of variations

13 Kranz, H. Die polarisationsmikroskopische Untersuchung der kataraktos getrubten Linse, Arch f Ophth **118** 571, 1927

in temperature on the output of the photocells was also examined and, within the margin of error of our measurements, was found not to affect the results. The drift effect of the photocells was investigated for the wavelength range from 400 to 820 millimicrons while the cells were in opposition. With our procedure the cells are constantly exposed to light, and a measurement of transmission at a single wavelength occupies between one and two minutes. The result is that for our purposes the drift effect was found to be negligible.

The wavelength ranges for different settings of the spectrometer dial for the widths of the slits used are given in table 1.

TABLE 1—*Wavelength Range for Different Settings of the Spectrometer*

Wavelengths, Millimicrons	Wavelength Ranges, Millimicrons
400-420	52-64
420-450	36-46
450-550	25-49
550-740	26-63

Consideration of the various sources of error leads us to believe that the transmission values given may be in error by as much as 17 per cent of the stated value at 400 millimicrons, 12 per cent of the stated value at 410 millimicrons, 6 per cent of the stated value at 420 millimicrons and less than 2 per cent of the stated values for all other wavelengths.

METHOD OF PROCEDURE

The method of procedure used in examining whole eyes may be briefly described as follows:

Whenever it was known that a patient was about to have his eye enucleated, and when the condition of the eye was such that its physical optical characteristics were expected to be normal, the scheduled time of operation was noted. An hour or two before operation the apparatus was checked for the normal functioning of the photocells, the zero setting of the microammeter and the focusing of the light on the entrance slit of the spectrometer and on the aperture supplying light to the bench photocell. The container for the eye was inserted in position so that it could be swung in and out of the beam, interrupting the path between the 45 degree prism and the photocell. As soon as the eye was enucleated, it was placed in physiologic solution of sodium chloride and brought from the operating room to the laboratory. The surrounding fatty tissue was stripped from the eye, and the optic nerve was cut short so that it would not interfere with the measurement of the transmission. During the entire process, including enucleation, care was taken not to abrade or deform the cornea. In each eye the corneal epithelium remained intact during the experiment. By observing the points of attachment of the extraocular muscles, particularly the inferior oblique muscle, and the position of the optic nerve, it was possible to find the region of the sclera which was approximately behind the fovea. With the use of a trephine 5 mm. in diameter,

the sclera and choroid were carefully cut through, and the pigment epithelium and retina were painstakingly removed from the region of the aperture. The eye was placed in the appropriate container with the pupil facing down, and a no. 0 Hellge Hardan cover slip was placed over the aperture and in contact with the vitreous, which welled up to meet the cover slip when the eye was in the container. Correction was made for reflection and absorption due to the cover slip. The beam emerging from the exit slit of the spectrometer, when incident on the cornea, was never larger than 2 mm square when the transmission was determined. The position of the eye was adjusted in the beam until a clearcut image of the slit was formed slightly above the cover slip. The photocell was now swung into position, and the location and size of the illuminated area on the photocell were noted. Since it was observed that different areas of the photocell showed slight differences in sensitivity, and since the output of the cell was also found to vary somewhat with the total area of the cell stimulated, even though the total energy was held constant, it was thought advisable to insure that the location and area of the stimulated portion of the photocell were the same whether the ocular media were in or out of the beam. A proper adjustment of the distance of the eye from the 45 degree prism and of the photocell from the eye accomplished this.

Ordinarily, we have been able to begin measuring the transmission within half an hour, and always within an hour, after enucleation of the eye. Our previous experiments on the eyes of rabbits, pigs and cats had demonstrated the advisability of examining the transmission within two hours after enucleation. The necessity for the use of fresh material has been emphasized by Mandach^{11b} in his work in the infra-red portion of the spectrum.

After the eye was out of the optical system, the bench photocell and the aperture supplying light to the photocell were adjusted until the microammeter read zero. The distance of the photocell from the aperture was noted. The eye was then introduced into the optical system by rotating the container on a supporting arm until it was restrained from further movement by an adjustable stop. The microammeter showed a deflection, and the bench photocell was moved farther from the light source until the microammeter reading again became zero. The distance of the photocell from the aperture was noted, thus permitting determination of the transmission at the wavelength for which the spectrometer was set, 400 millimicrons. The spectrometer wavelength dial was then set at 410 millimicrons, and the balance was again made. This procedure was repeated every 10 millimicrons up to 500 millimicrons, every 20 millimicrons from 500 to 700 millimicrons, and every 40 millimicrons from 700 to 820 millimicrons. After these measurements of the transmission of the combined media *in situ* had been completed, we measured in a similar fashion the separate transmission of the aqueous, vitreous, lens and cornea, each placed in an appropriate container.

RESULTS

The results of our measurements on 4 human eyes are presented in table 2. The transmission values were averaged at each wavelength and calculated as if the eyes contained lenses of an average age of 21.5 years instead of the lenses which were in the eyes at the time when the actual measurements were made.

Each of the 4 complete eyes was obtained after enucleation for a small sarcoma of the choroid. Since sarcomas occur ordinarily in the later

years of life, the average age of the eyes was 62 years. The absorption characteristics of the human crystalline lens for light of visible wavelength change in a fairly regular fashion with age, as will be shown in a subsequent paper. Changes in the infra-red^{11g} and ultraviolet^{12j} absorption spectrums of animal eyes with age have been noted. In table 2 are given the transmission values for eyes with young lenses, because the results of our investigations will be of most value for the correction of various observations made on relatively young persons.

TABLE 2—*Average Transmission Values for Four Human Eyes*

Wavelengths, Millimicrons	Average Transmission Values, Percentage $\times 0.01$
400	0.086
410	0.106
420	0.160
430	0.248
440	0.318
450	0.388
460	0.426
470	0.438
480	0.458
490	0.481
500	0.495
520	0.525
540	0.559
560	0.572
580	0.594
600	0.610
620	0.631
640	0.649
660	0.664
680	0.690
700	0.705
740	0.711
780	0.716
820	0.716

Our method of calculation may be briefly described. As has already been mentioned, we separately determined the transmission of the lenses of each of the 4 eyes. The transmission of the lenses showed considerable individual differences, amounting to as much as 20 per cent at some wavelengths. We then calculated what the transmission of each of these old eyes would have been without its lens and averaged the results for the 4 eyes at each wavelength. We next calculated the average transmission at each wavelength for a group of 5 normal lenses of an

average age of 21.5 years. Individual differences in this group were still present, although less than in the older age group. We then calculated what each eye would transmit at each wavelength if it were supplied with our lenses of an average age of 21.5 years and averaged the results. These are given in table 2.

The data in this table show that the absorption gradually increases from the long wave end to the short wave end of the visible spectrum. The absorption is considerable and in the blue portion of the spectrum is far in excess of that of water. If Aschknass' results are calculated for a layer of water 2.28 cm. thick, it is found that for the wavelengths from 450 to 737 millimicrons the transmission never drops below 0.95 per cent. It should be noted that if there were any undetected stray light which was affecting our measurements in the short wave end of the spectrum, it could result only in the measured transmission being greater than the real transmission. This means that if it were possible to eliminate stray light completely, the absorption in the violet portion of the spectrum could only be still greater than that indicated by table 2.

There are two possible objections to the adequacy of the data in table 2. One may be based on the pathologic condition of the eyes and the other on the age of the eyes. As to the first objection, there is a possibility, as is indicated by the work of Rados,¹⁴ that the amino acid content of the aqueous may be somewhat higher in an eye with sarcoma of the choroid than it is in the normal eye. This increase in amino acid content of the aqueous humor was attributed by Krause¹⁵ to the degeneration of tissue proteins. The eyes we used, however, had small flat sarcomas. On ophthalmoscopic examination the media appeared normal, and it seems improbable that the transmission of the ocular media of these eyes was significantly affected by the presence of the small sarcomas.

As to the second objection based on the age of the eyes, allowance has already been made for the age of the lens, and it is the lens which accounts for the major part of the selective absorption of the eye in the visible spectrum. It may be that changes of transmission with age also occur due to changes in the vitreous, aqueous and cornea. A priori, these changes would be thought to be of much less extent than those which occur in the lens. Unfortunately, since the 4 whole eyes for which we were able to measure the aqueous, vitreous and cornea ranged in age only from 59 to 66 years, we were unable to demonstrate any differences in the transmission of the cornea, vitreous or aqueous due to age.

14 Rados, A. Untersuchungen über die chemischen Zusammensetzungen des Kammerwassers des Menschen und der Tiere, *Arch. f. Ophth.* **109** 342, 1922.

15 Krause, A. *The Biochemistry of the Eye*, Baltimore, Johns Hopkins Press, 1934, p. 119.

In order to compare our data with those of Roggenbau and Wetthauer, referred to previously, we have attempted to determine with reasonable accuracy from their histogram the transmission values of the bovine eye in the visible spectrum, as is shown in figure 4. Also plotted in figure 4 are the results given in table 2. The qualitative agreement between the two sets of data is close in view of the differences in age, axial length and other factors between the human eye and the bovine eye. Our extremely low transmission values in the violet portion of the spectrum are qualitatively confirmed. These low values should not be surprising if one considers that the refractive media of the eyes of animals may absorb practically completely at 380 millimicrons.

It is not possible to give exact figures for the data of Roggenbau and Wetthauer corresponding to those in table 2, because, as has been explained, their graph is unaccompanied by specific figures for trans-

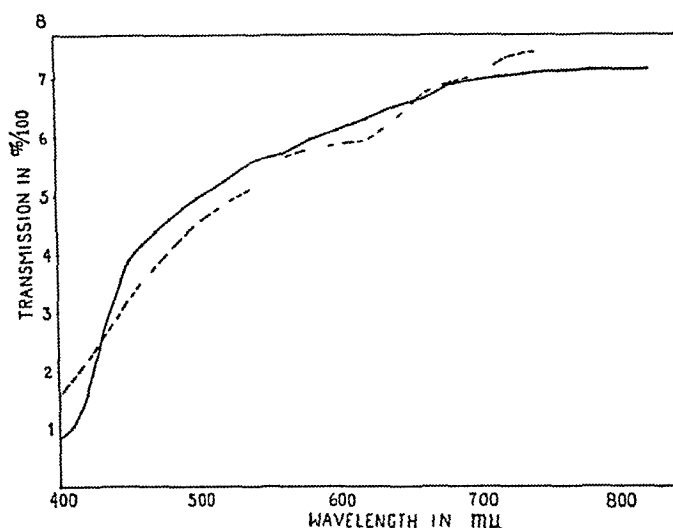


Fig. 4—Graph showing transmission at various wavelengths. The solid line represents data on the human eye, the broken line, data on the bovine eye calculated from the histogram of Roggenbau and Wetthauer.

mission. We have, however, calculated the trichromatic coefficients for our own data and also for the approximate data which we derived from Roggenbau and Wetthauer's histogram. The results for the bovine eye are $x=0.383$, $y=0.385$ and $z=0.232$. An equal energy source passing through a filter of corresponding characteristics would give a dominant wavelength of 578.4 millimicrons with a spectral purity of 37.5 per cent¹⁶. Similar calculations for our data give $x=0.376$, $y=0.382$ and $z=0.242$, corresponding to a dominant wavelength of 577.6 millimicrons and a spectral purity of 35.3 per cent. It is seen that the dominant wavelength is practically the same for the two sets of

¹⁶ Hardy, A. Handbook of Colorimetry, Cambridge Mass., The Technology Press, 1936, p. 80.

data, and the spectral purity is possibly slightly higher for the bovine eye than for the human eye. The greater degree of spectral purity for the bovine eye is attributable to the relative excess of transmission of light at wavelengths of from 660 to 740 millimicrons and to the relative defect of transmission of light at wavelengths of from 430 to 660 millimicrons, which together more than overcome the considerably reduced transmission of the human eye in the extreme short wave end of the visible spectrum below wavelengths of 430 millimicrons.

COMMENT

In general, our results permit a fairly accurate statement of the amount of physical energy actually reaching the retina when external radiant energy of visible wavelengths is used as a stimulus. Many experiments on human subjects with normal eyes have yielded data from which conclusions have been drawn as to the response of the retina to light of visible wavelengths. In view of our experimental results, these conclusions obviously require modification to a greater or less extent.

We shall now briefly consider some applications of the data. As to the decrease in apparent brightness due to the absorption of the ocular media, it is apparent that it is considerably greater than 8 per cent. The average transmission throughout the visible spectrum is 0.531 per cent. In order to find the effect of a reduction in transmission on the apparent brightness of an equal energy source, however, it is necessary to weight the transmission values at various wavelengths by the appropriate values of the relative visibility curve. Thus, although the absorption is high in the blue portion of the spectrum, the effect of this absorption on the apparent brightness of an equal energy source is relatively slight owing to the low visibility factor at the short wave end of the spectrum. If we consider the absorption due to the ocular media alone, neglecting the absorption due to the macula lutea, it will appear that the apparent photopic brightness of an equal energy source will be only 56.9 per cent of the brightness it would have if the energy were directly incident on the retina. Since the scotopic, or low intensity, visibility curve is shifted almost 50 millimicrons in the direction of the short wave end of the spectrum, it is apparent that the absorption of the ocular media will have a more profound effect on the apparent brightness at low intensities than at high. The apparent scotopic brightness of an equal energy source will be 49.8 per cent of the brightness it would have if the energy were directly incident on the retina.

Briefly, to mention some further applications of our work, the data here presented are clearly of significance for the evaluation of the retinal complementation valences of light of different wavelengths and

the evaluation of retinal trichromatic functions. Individual differences of color sensitivity must be considered in relation to this work. The minimum radiation perceptible by the retina is obviously less than that indicated by the various determinations of the minimum radiation visually perceptible. Our results permit a comparison in physical terms of after-images and entoptic phenomena with external stimuli.

Other applications of our data which we have made will be described in subsequent papers. These applications include the correction of the scotopic and photopic ocular visibility curves and the determination of the relation of the resulting retinal scotopic visibility curve to the absorption spectrum of visual purple.

ASSOCIATION OF AN ANNULAR BAND OF PIGMENT ON POSTERIOR CAPSULE OF LENS WITH A KRUKENBERG SPINDLE

WILLIAM ZENTMAYER, MD

PHILADELPHIA

The association of a developmental anomaly, an annular band of pigment on the posterior capsule of the lens, with a Krukenberg spindle, a distinctive collection of pigment on the posterior surface of the cornea, a condition the cause and nature of which is in dispute, is of interest as it gives support to the view that Krukenberg's spindle is an acquired lesion. A case in which this condition occurred is reported.

A youth aged 16 came to me to have his eyes examined for glasses, as distant vision was poor. There was a low grade myopic astigmatism. Each eye showed a characteristic Krukenberg spindle occupying the midline in the lower half of the cornea. It could be plainly seen with the ophthalmoscope and seen in detail with the biomicroscope. It measured about 4 mm in length and 1 mm in its greatest width and extended from the center of the cornea to a point just above the lower limbus. With the biomicroscope the fusiform lesion was seen to be made up of brown pigment granules situated on or in the endothelium of the posterior surface of the cornea.

On the surface of the posterior capsule of the lens of each eye there was an annular band of brown pigment. As seen with a +20 D lens of the ophthalmoscope, it had an average width of 0.5 mm and was situated about 2 mm in from the equator of the lens. The margins of the band were more or less serrated. In the right eye the circular band was broken at points corresponding to 8 and 4 o'clock, the gaps being about 2 mm wide. At these breaks short tags of pigment were seen directed toward the tips of the ciliary processes. In the left eye the band was unbroken, but there were a few lines of pigment running toward the equator of the lens.

The refraction estimated under cycloplegia with homatropine was

O D -0.25 D sph \subset -1.50 cyl, ax 15, vision 6/7.5 (?)
O S -0.75 D sph \subset -2.00 cyl, ax 165, vision 6/7.5

The vision obtained indicates that the eyes were considerably amblyopic, as with the illumination used the normal vision is 6/4.

At the time I saw the patient I could not recall having seen the anomaly described in any textbook or in the literature. Collins and Mayou, however, refer to it. A short time later, on the appearance of

Read before the College of Physicians of Philadelphia, Section on Ophthalmology, Feb 17, 1938

Mann's book on "Developmental Abnormalities of the Eye," I found the anomaly, as it appeared in the case reported here, described and illustrated under the title of "ring-shaped markings near the periphery of the lens," in the following words

They consist of a single continuous line, or a dotted line, or one or two spots only, in the course of the hypothetical line. The line is concentric with the edge of the lens and runs so near the periphery that it practically coincides with the margin of the fully dilated pupil or is a little internal to this

Mann stated that the most likely explanation of these lines is that they are due to the maintenance of the contact between the tips of the ciliary processes and the lens for too long a period. In the 100 mm embryo the tips of the ciliary processes are in close contact with the periphery of the lens. They are at that time already pigmented. (In many animals this contact is maintained throughout life, and in birds

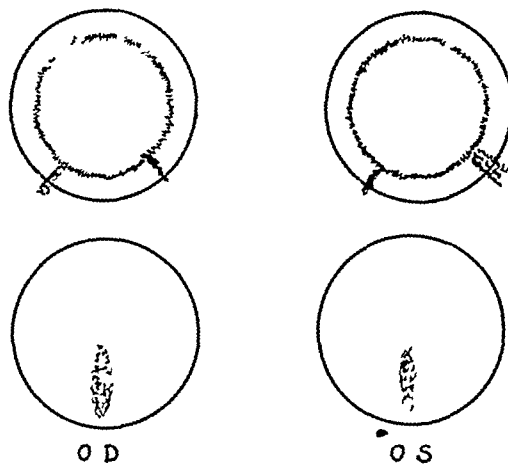


Fig 1—The upper two drawings show the annular band of pigment on the posterior capsule of the lens. The lower drawings show Krukenberg's spindle in each eye

the lens is actually indented by the processes) The ciliary ring increases in diameter more rapidly than the lens, with the result that the ciliary processes are withdrawn from the lens and a space is left between the two

These rare pigmented lines can be looked on as formed of pigment detached from the ciliary processes, which after too long contact finally manages to break away

According to Collins and Mayou

The cilio-posterior-capsular fibres of the ligament instead of becoming elongated into strands of kerato-hyaline may retain their cellular character. These cellular adhesions then hold the ciliary processes in contact with the posterior capsule of the lens, so that on the expansion of the globe the processes become stretched and attenuated. If the lens remains clear these elongated ciliary processes can be seen clinically attached to its posterior surfaces. Pigmented patches on the

back of the lens are sometimes seen as a congenital abnormality. They probably mark the site of adhesions of the ciliary processes which have subsequently given way on growth of the eyeball.

The development of the Krukenberg spindle has been observed by several ophthalmologists. Koby in 1927 reported 2 cases of Krukenberg's spindle in which the condition developed while the patients were under his care. The first case was that of a woman who had iritis. Four months after she recovered from the iritis a pigmented spindle was seen to be forming in one eye. There was no spindle in the other eye. In the second case, that of a man, dots of pigment were seen on the posterior surface of the right cornea, which in eighteen months

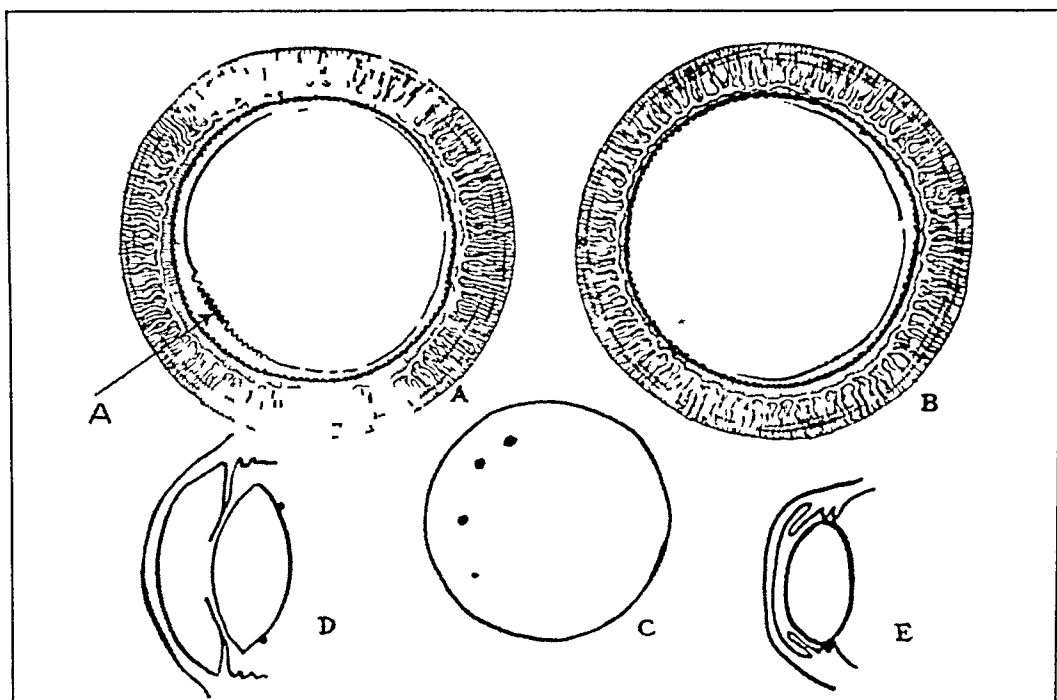


Fig 2—*A* and *B* show the right and left eye with ring markings on the back of the lens (*A*). *C* shows the dots of pigment in another case, *D*, the position of the markings, and *E*, the temporary adhesions of the ciliary processes to the lens (From Mann, I. *Developmental Abnormalities of the Eye*, New York, The Macmillan Company, 1937.)

developed into a typical Krukenberg spindle. There were no signs of ocular inflammation.

Jonas Friedenwald has permitted me to report a case observed by him.

A Negress, first seen in June 1928, complained of spots before the eyes and headaches, which were worse during the night. The eye presented no abnormalities. The Wassermann reaction was positive. The patient was placed under treatment. In August 1928 there was minimal circumscribed congestion in the right eye. The aqueous was filled with pigment granules, and there was a vertical spindle-shaped

deposit of pigment on the posterior surface of the cornea. There were no synechiae, and the vitreous was clear. Vision was 20/30. Though the patient was still under antisyphilitic treatment, the Wassermann reaction was positive. One month later the eye was quiet, and vision was 20/20. One year later the pigmented spindle had entirely disappeared, leaving only a few pigment granules on the back of the cornea. Since then the eye has been repeatedly examined and has never shown recurrence of the spindle or any evidence of intraocular inflammation.

Greeves saw the spindle develop in a woman aged 62.

Haussen examined an eye microscopically in which a clinically typical picture of Krukenberg's spindle was present. At the site of the spindle the wall of the cornea was pigmented, and the endothelial

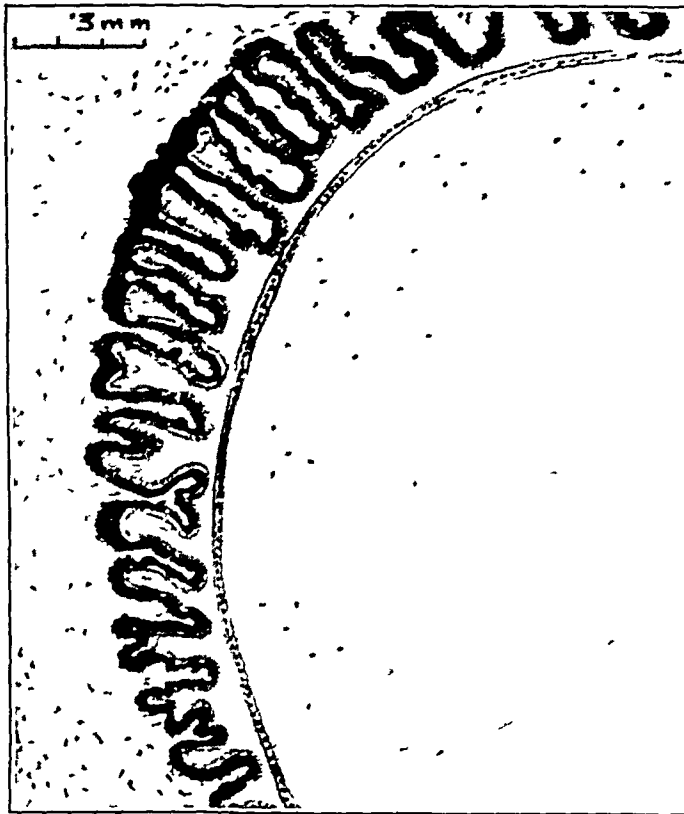


Fig 3—Ciliary processes in contact with the lens at 110 mm. (From Mann, I. *Developmental Abnormalities of the Eye*, New York, The Macmillan Company, 1937.)

cells, which elsewhere were regularly formed and situated, were thickly filled with pigment dust. Therefore, the endothelial cells must have acquired phagocytic properties. The framework of the iridocorneal angle was infiltrated by fine pigment. The lumens of Schlemm's canal were surrounded by particles of pigment. At the edge of Descemet's membrane there was also pigment which had evidently migrated from the iridocorneal angle. Haussen thought that the iris was the source of the pigment.

According to Edgerton, who reviewed the literature on Krukenberg's spindle, the corneal lesion so designated was first described by

Krukenberg in 1899, who gave it the name "bilateral congenital melanosis of the cornea" The explanation Krukenberg gave for the lesion was that in fetal life, when the pupillary membrane is contiguous with the cornea, the pigment is fused with the cornea

Stock stated that he considered the pigmentation to be an anomaly in that the cells remaining on the posterior surface of the cornea after the formation of the anterior chamber have the power of developing pigment at the time of birth

Koby expressed the belief that the condition is due to convection currents in the anterior chamber and, in agreement with Turk, that the granules scratch the delicate endothelium and so facilitate deposit He stated that the force of gravity accounts for the fact that the center of the spindle is below the center of the cornea, that disintegration of pigment is facilitated by myopia, senility and chronic inflammation, and that the spindle forms more rapidly if due to an inflammatory process

Peter, Griscom and Holloway stated that they believed the condition to be acquired Peter expressed the belief that a low grade chronic inflammation of long standing, such as is often observed in cases of myopia, would probably be found in all cases of Krukenberg's spindle

Friedman, according to Duke-Elder, agreed with the views expressed by Koby

Vogt expressed the belief that diffuse senile and presenile endothelial pigmentation and its fusiform arrangement into a spindle are two phases of the same tendency

Duke-Elder stated that the Krukenberg spindle is probably an accentuation of a general process in which pigment derived from the uveal tract is deposited on the corneal endothelium and aggregated into the space of an approximate vertical spindle, although minute examination frequently shows that a much larger area of the cornea is bespeckled to a less degree

He further stated that when one adds to Vogt's careful consideration the facts that the congenital origin of the spindle is merely a surmise in so far as it has never been seen in young persons and that it has been seen to appear in a previously unpigmented eye and to develop gradually under observation, the acquired nature of the spindle must be conceded

Mann expressed the belief that the distribution of the pigment granules suggests some connection with the thermal currents in the anterior chamber, as the spindle occupies the position of Lusse's line (the linearly arranged leukocytes often found on the posterior surface of the cornea in normal eyes, usually of children, the distribution being accounted for by the thermal current)

Bauer Caramazza expressed the same view

Weinkauff suggested that the spindle might well represent an unusual type of arrangement of precipitates

Previous case reports show that the spindle may be acquired both in quiet eyes and in eyes that had been the seat of inflammation of the uvea, also, that in one instance it developed and disappeared while the patient was under careful observation over a period of about one year

In view of the opinions expressed by Vogt, Duke-Elder, Mann and others, it seems reasonable to believe that in the case reported here the granules freed from the pigment line on the posterior capsule of the lens were carried by the circulation of the fluids of the eye into the anterior chamber, where the thermal currents deposited them on the posterior surface of the cornea

DISCUSSION

DR ALFRED COWAN Krukenberg's spindle occurs more frequently than was considered before the advent of the corneal microscope and the slit lamp. Often the same type of pigmentation as is found in Krukenberg's spindle can be seen with the slit lamp but would escape notice by the ordinary methods of examination with oblique illumination. The pigment is deposited all over the posterior surface of the cornea, and the spindle formation is the result, merely, of a more dense arrangement over the spindle-shaped area. While it may be that the imprint of the pigment ring on the posterior surface of the lens is congenital in most instances, I have seen this condition result from inflammation several times.

TESTING FITNESS FOR NIGHT FLYING VISUAL ACUITY

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AND

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Visual acuity may be defined as the power to see clearly, or to discriminate detail in objects. This power may be measured by the angle subtended at the nodal point of the eye by the detail discriminated or, more practically considered, by the angle subtended at the frontal surface of the eye. It may also be measured in terms of the size of the detail and its distance from the eye. It differs greatly with the intensity of light employed, and the amount of difference varies in individual subjects. So great is the effect of intensity of light and so poorly has it been subjected to control that it might be well to emphasize the importance of the factor by qualifying the definition to read: the power to see clearly, or to discriminate detail, at a given intensity of light. An entirely correct expression of acuity of course requires not only the specification of the size of the detail discriminated and of the intensity of illumination but a statement of all the other variable factors which affect power to discriminate detail. Ordinarily, for the sake of convenience this situation is handled by accepting certain conditions as to these factors as standard and always making the test under these conditions.

Visual acuity sustains an important but as yet unknown relation to sensitivity to light. In the testing of night flyers it is important, therefore, to determine not only their sensitivity to light but their power to see objects or to discriminate detail at low illumination and the effect of dark adaptation on this power.

The great effect of intensity of light on acuity and on the relative rating of eyes as to acuity is a matter of comparatively recent knowledge. Our attention was called to the need for the testing of acuity at low as well as at high illumination by a request made during the World War by Commander G B Tribble, who was then of the United States Naval Hospital in Washington, D C. Commander Tribble declared that not more than 25 per cent of the men accepted for service in the navy were able to qualify for the lookout service at night on the bridge

of battleships His request was for a method and a suitable instrument for testing candidates for this service Our recommendation was that the test be of visual acuity at low illumination For making this test we devised a method and an instrument, both of which were used by him with, as he stated, great success and satisfaction

Just what proportion of men accepted for flying service are fit for night flying we are not prepared at this time to say The problem of testing as to fitness for night flying is somewhat similar, however, to that of testing as to fitness for the lookout service at night on the bridge of a battleship There is the same need of unusual ability to see at low illumination and of unusual power of dark adaptation

IMPORTANT FUNCTIONS TO BE TESTED

For night flying important functions to be tested are (1) the ability to see at night and at low illumination and the effect of dark adaptation on this ability and (2) the amount and speed of dark adaptation Normal or better than normal sensitivity in light adaptation is of course also important The eyes that are needed for night flying are the best of what might be called the normal group, that is, of those that have both good dark and good light vision More important than speed and range of adaptation, however, is the place in the scale of sensitivity at which the adaptation or change in sensitivity occurs

1 *The Ability to See at Night and at Low Illumination and the Effect of Dark Adaptation on This Ability*—Two functions are involved in the ability to see, namely, the ability to sense light, or to discriminate differences in light and brightness, and the ability to discriminate detail, or what is commonly called visual acuity Either of these functions may be used in testing the ability to see at low illumination and the effect of dark adaptation on this ability

If visual acuity is chosen, the determinations may be made at any level of illumination or at the end of any period of light adaptation from low to high, in which case the result may be taken as indicating the power to see objects or to discriminate detail under that condition Correspondingly, the effect of dark adaptation on the ability to see at low illumination may be tested by determining the acuity at some predetermined low level of illumination at the intervals selected for consideration from the beginning of dark adaptation until the process is complete The results of these determinations when plotted against time of adaptation in the form of a curve give a complete picture of the course of dark adaptation for the eye in question through a given period so far as its effect on acuity or the power to see objects is concerned Thus one can determine the eye's ability to see objects at any time from the beginning to the end of dark adaptation at any level of

intensity that may be selected for the test. Whether one or a number of intervals is chosen will depend on the purpose for which the test is being made. If the result is wanted approximately at the eye's maximum sensitivity to light, for example, the test should be made at the end of an interval of twenty or thirty minutes. If preferred, instead of complete darkness the adaptation may be to some low level of illumination, for example, of the order experienced in night flying.

Visual acuity at low illumination may be tested in two ways: (a) by determining the smallest detail or visual angle that can be discriminated at a given low intensity of illumination and (b) by determining the lowest intensity of illumination at which a given size of detail or visual angle can be discriminated. Either method gives the smallest detail or smallest visual angle that can be discriminated at the intensity of light employed. Since, however, with the means now available it is much easier and quicker to vary the intensity of illumination in small steps or in continuous series than to vary the size of the test object, the second of these methods is the more feasible and convenient, particularly for determinations of the effect of dark adaptation on acuity. It will therefore be the only method considered in connection with the tests to be discussed in this paper. In the use of this method of determining acuity, the adaptation curves are obtained by plotting against time of adaptation the intensity of light at which the given detail can be discriminated.

In order to show individual differences in the ability to see or discriminate detail at low as compared with high illumination, a study was made of 61 observers, and in order to show the effect of dark adaptation on this ability and individual variations in this respect, a study was made of 21 observers. The results of the latter study are given in figures 1 and 2. In this study acuity was measured by determining the lowest intensity of light at which a suitable size of detail could be discriminated. The results of both studies will be discussed later in the paper.

2 The Amount and Speed of Dark Adaptation—In connection with the function of dark adaptation, two important points are presented for testing: the total amount, or range, of adaptation for the eye in question and the speed of adaptation, or the quickness with which the eye changes its sensitivity with change from dark to light and from light to dark. Those intimately conversant with the ocular needs of the night flyer say that the latter of these two points is the more important. In modern aviation, they maintain, what the night flyer needs more than anything else is the power to change his vision quickly from the illuminated cockpit and instrument panel to the outside world and back again. In this connection it may be noted that the transition from the outside back to the cockpit does not present a serious problem,

because, as will be discussed later, light adaptation takes place with much greater rapidity than dark adaptation. What problem there is comes from the fact that until sufficient light adaptation has taken place a disturbing dazzle may be present. In most cases, at medium intensities this effect will disappear in a few seconds. If necessary, a test for light adaptation can easily be devised. However, in relation to fitness for night flying it is perhaps well to point out again that neither speed

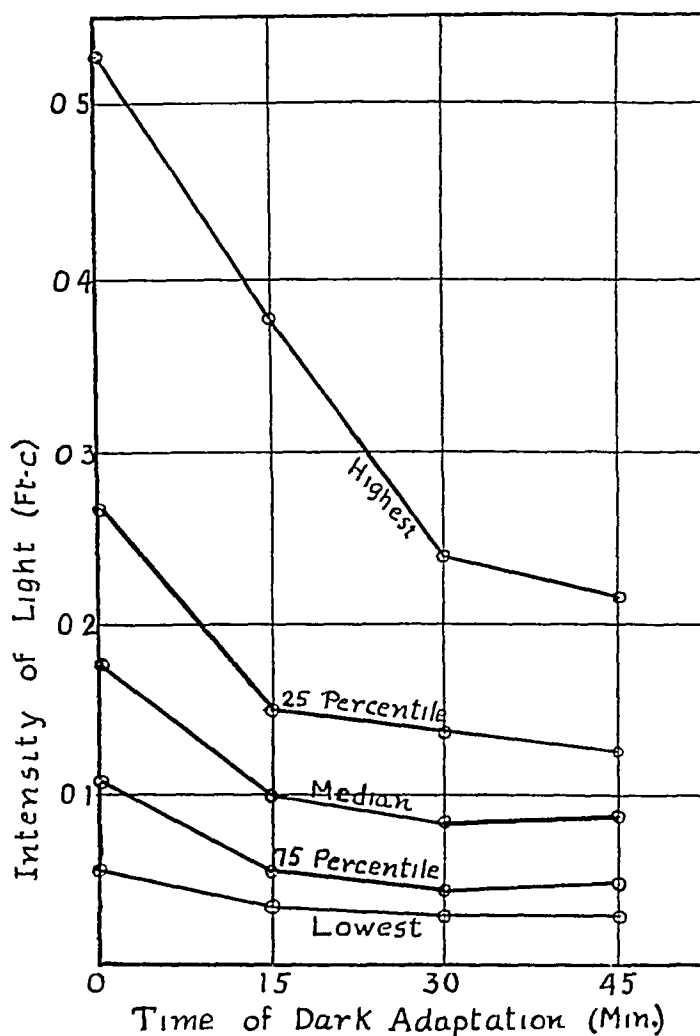


Fig 1—Curves showing the lowest intensity of light needed to discriminate a detail subtending one minute of visual angle at the beginning of a period of dark adaptation and after fifteen, thirty and forty-five minutes, based on the highest, the lowest and the 25, 50 and 75 percentile values for a group of 21 observers, aged from 18 to 28 years. The intensity of light is expressed in foot candles. The distance between the limiting curves represents the total range of values for the group in question, that between the 25 and 75 percentile curves, the middle 50 per cent range of values. The adaptation represented in these curves is foveal.

nor amount of change in sensitivity is as important as the place in the scale of sensitivity at which they occur. That is, it is quite possible that a candidate might have a good range and speed of adaptation and

still a comparatively poor power to see at low illumination both at the beginning and at the end of the period of dark adaptation. Such a person would be obviously unfit for night flying. To be fit for night flying the candidate must have a normal or better than normal rating in power to see at low illumination at the beginning of the period of dark adaptation and throughout its course.

For an approximate determination of the total amount, or range, of adaptation as measured by the light minimum for the discrimination of detail, the test can be made at the beginning of the period of dark adaptation and at the end of some suitable interval, preferably fifteen or twenty minutes. The curves, previously referred to, in figures 1

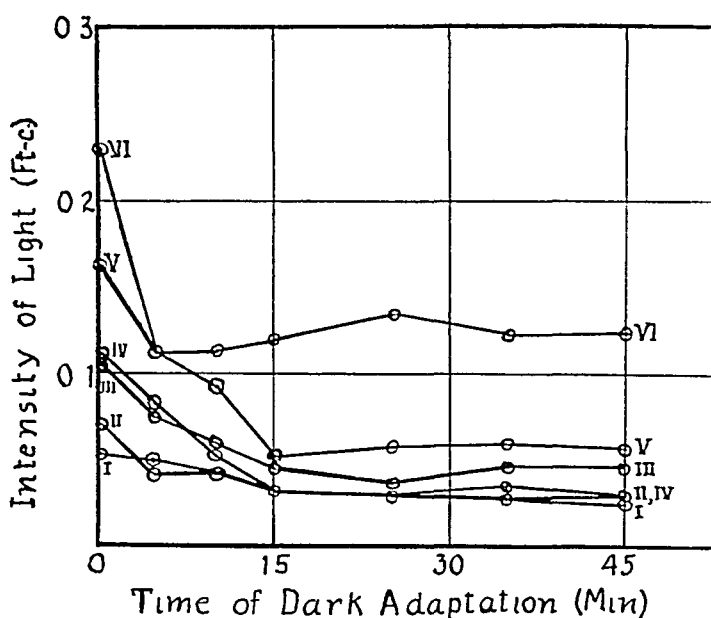


Fig 2—Curves for 6 observers, aged from 18 to 28 years, showing the lowest intensity of light needed to discriminate a detail subtending one minute of visual angle at the beginning of a period of dark adaptation and at the end of five, ten, fifteen, twenty-five, thirty-five and forty-five minutes. The intensity of light is expressed in foot candles. The adaptation represented in these curves is foveal

and 2 show the amount of adaptation that was obtained for 21 young healthy pairs of eyes. A fairly good conception of the individual variation in this result may also be had from these figures, although the curves in figure 1 were not plotted specifically to bring out this point.

For testing the speed of adaptation, one of the following methods may be used. For the first two, alternate procedures are given for measuring the speed of adaptation in general, regardless of the purpose for which the measurements are to be used. The second two are presented as quick tests for the acceptance or rejection of candidates on

the basis of predetermined critical values and for the rating of those whose adaptation falls above or below the critical value

In method A the amount of light required to discriminate the test object is determined at the beginning of the period of dark adaptation and again at the end of some interval sufficiently long to cover the greater portion of the period of rapidly changing sensitivity, for example, at the end of five minutes. Either the total change in result that has occurred in this time or the average rate of change, i.e., the total change divided by the time, can be taken as the index of the speed of adaptation. In this method the time of adaptation is fixed and the amount of light required to discriminate the test object is determined at the end of that time.

In method B the amount of light required to discriminate the test object is determined at the beginning of the period of dark adaptation, and then the time that must elapse until the discrimination can be made at an intensity of light of a predetermined critical value is measured. Again, the change in result divided by the time required to obtain this change may be taken as the index of the speed of adaptation. In this method the illumination of the test object is fixed and the time required to attain the sensitivity needed for the discrimination of the test object is measured.

With method C, in case a critical value has been determined in preliminary work for the time of adaptation needed to discriminate the test object with a given amount of light, the discrimination required may be attempted at the end of that interval. If the discrimination cannot be made, the candidate is ruled out. If, however, a rating of candidates who have failed is wanted, then rating can be determined by decreasing the difficulty of the task set for discrimination, that is, by increasing the intensity of illumination of the test object. Likewise, if a rating is wanted of candidates whose speed of adaptation is greater than that indicated by the critical value, this rating can be determined by increasing the difficulty of the task, that is, by decreasing the intensity of the test object. In this method both the time and the task set for discrimination are fixed, and it is determined whether the candidate can meet the requirement.

In method D the task set for discrimination may be given a predetermined value and the time required to attain this value measured directly. This time may then be compared with a predetermined critical time. Or the alternative procedure may be used, that is, the time of adaptation may be set at a predetermined value and the amount of light required to discriminate the test object determined at the end of this time. The result may then be compared with a predetermined critical value. From these results the candidate may be rated merely as meet-

ing the requirement, more than meeting the requirement or failing to meet the requirement, or, if desired, the rating can be made quantitative.

With respect to these methods, the following comments may be offered: (a) Methods C and D can be used only on the assumption that all the candidates tested have good power to see at low illumination while still light adapted, that is, at the beginning of the period of dark adaptation. The night flyer should have normal or better than normal ability to see objects the instant he looks from the cockpit to the outside world, as well as normal or better than normal power to increase this ability as dark adaptation is prolonged. Obviously the point should be tested, not assumed, that is, the test should be made at the beginning

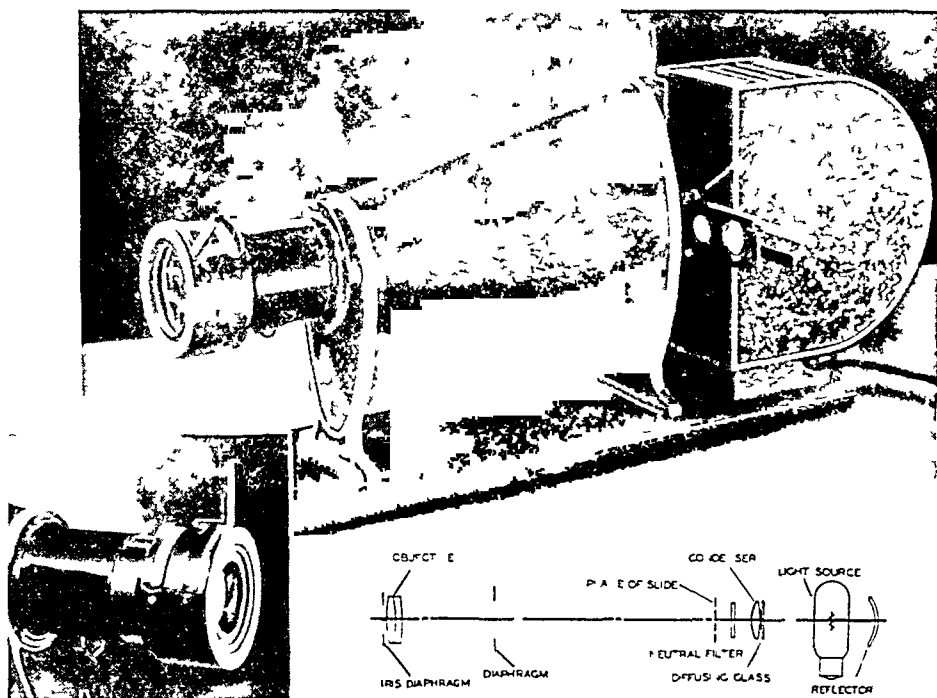


Fig 3—The Ferree-Rand projector and a drawing of its optical system. This instrument can be used for testing visual acuity and errors of refraction and for determining the light minimum for the discrimination of detail.

as well as at the end of the period of dark adaptation selected, as is done in methods A and B. Methods C and D have been considered here only because they are of the type that has been suggested by other writers and was used during the World War for testing the light sense. (b) When the intensity of light on the test object is fixed and the time required to discriminate the test object determined, a serious objection is encountered if the method is used for more than a short period of adaptation. To require the observer to attempt to hold a state of fixation of the eye for any considerable length of time induces an ocular condition that is detrimental to the purpose of the test, and unless the

observation is continuous an exact measurement of the time cannot be made. Any one who has ever read a photometer knows that a few seconds of staring at the test field will obliterate somewhat the difference in brightness in the surfaces compared. In other words, a fixed eye quickly loses its sensitivity to differences in brightness. A single ocular movement or blink will restore the sensitivity that is lost by several seconds of fixed exposure. It is almost impossible to standardize the ocular movements and blinking that will occur in from three to five minutes. The candidate who takes his task most seriously and gives the steadiest fixation will recover his sensitivity most slowly. In fact, if it were possible to rule out blinking and ocular movement he would lose rather than recover the power to discriminate differences in brightness. It does not take long, particularly under low illumination, to stare out small differences in brightness in the test field and the details formed by these differences. A fixed eye, or an eye that is as nearly as possible motionless, in the dark soon passes into an abnormal condition of inability to make objective discriminations, owing in part to the masking influence of a great variety of subjective visual phenomena. Ocular movement operates strongly to prevent these veiling phenomena, a single movement often bringing out in strong relief differences in brightness and details that have been completely obscured. A fixed eye is never a sensitive eye, and the best way to obtain high sensitivity and at the same time to standardize sensitivity is perhaps to move the eye quickly before each observation. In general, therefore, experimental methods or test conditions requiring unduly long periods of fixation of the eye should as far as possible be avoided.

THE TEST INSTRUMENT

A satisfactory instrument for making these tests is the one we devised, as already stated, for the use of the navy during the World War¹. It has been called an acuity projector. A photograph of the commercial model and a drawing of the optical system are given in figure 3. The instrument has the following features and advantages:

- 1 It is small, compact, easy to operate and conveniently portable.
- 2 It can be used either to illuminate a printed test chart or to project the image of a test chart or test character on a blank screen. Means are provided for focusing for distances ranging from 10 to 20 feet (from 304 to 609 cm), images being given in each case of the size correct for that distance.

¹ Ferree, C. E., and Rand, G. Visual Acuity at Low Illuminations. Apparatus and Results, *Tr. Am. Ophth. Soc.* **17** 370-395, 1919, *Lantern and Apparatus for Testing the Light Sense and for Determining Acuity at Low Illuminations*, *Am. J. Ophth.* **3** 335-341 (May) 1920.

3 It is provided with a means for varying the intensity of illumination of the test objects over a wide range without changing the color or composition of the light or the size, shape or location of the illuminated area. This means consists of an iris diaphragm located as close as possible to the back or the front surface of the projection lens, so that it cuts uniformly each cone of light that is focused to form the various points in the projected image. It is because of this position of the diaphragm that it cuts down uniformly the light in the image without changing its size, shape or location.

4 It not only delivers the light to the test surface in variable amounts as desired but measures the amount delivered. That is, it is provided with a scale from which the amount of light delivered can be read or easily derived.

5 We have devised an attachment for changing the size of the test object in continuous series without changing its intensity of illumination. It provides means for changing independently of each other both the intensity of the illumination and the size of the test object in continuous series. The attachment, however, is not furnished at this time with the commercial model.

6 The instrument is provided with the best type of test object known to us for standardizing the testing and rating of acuity. It is provided also with a complete auxiliary equipment for testing errors of refraction. This equipment includes the two most sensitive tests for astigmatism known to us at this time, a radial line chart at high illumination and a rotatable double-broken circle at low illumination. Further, because of its control of intensity the instrument provides a sensitive means of completing the correction for any type of refractive error at low illumination, and not only does it provide a sensitive and highly accurate means of testing for errors of refraction, but it is said by many authorities to furnish the quickest means known to them of completing a refractive correction.

7 It has the great convenience and advantage that all the changes and operations required for the test can be made by the examiner within easy reach of both the person tested and the trial case when a trial case is used.

8 It does not require the use of auxiliary charts. All the test characters and test objects can be prepared in the form of lantern slides, which can readily be kept clean and stored in a small space. The use of lantern slides permits of the employment of a great variety of test material without undue inconvenience in using and storing. It also permits of less opportunity for the memorization of the test material by the person tested. Furthermore, it permits of the convenient rotation of such test objects as the broken circle, the double-broken circle and the illiterate E.

The instrument makes it possible to determine acuity and the curve of dark adaptation as measured by changes in the amount of light required to discriminate the test object under the precise conditions of measurement and control that are needed in making standard determinations. Some of the conditions are: 1 The determinations may be made at constant and reproducible intensities of illumination from very low illumination to the brightness equivalent of 100 foot candles on an ordinary chart. 2 The intensity of light delivered may be read directly or readily derived from the scale of the instrument. 3 All instruments are carefully standardized before they are delivered to the customer. 4 The best type of test object known to us for standardizing the testing and rating of acuity has been adopted. The test objects furnished by us have been constructed with great accuracy, and care will be taken that a high standard of uniformity is maintained in all test equipment and material. 5 The test equipment and material are put out in such a form that they may easily be given proper care and deterioration may be prevented. This is important if consistency of result is to be obtained over a period of time.

THE TEST OBJECT

In any standard rating of acuity or in any test involving a standard rating of acuity or a correct and high reproducibility of result, the type of test object and the type of judgment required are of fundamental and paramount importance—of greater importance than any acuity instrument or projector. With respect to the testing of acuity, it is absolutely essential to understand in the beginning that a correct test cannot be made unless a correct test object is used. It is for this reason and because high sensitivity and high reproducibility of result are required in the type of experiments considered here that space will be given to a discussion of the type of test object that should be employed. A further important requirement in the choice of a test object is that it can be presented as many times as may be needed without danger of memorization by the person tested. None of these requirements can be satisfied by the test objects of the capital letter type.

After many years of preliminary study and experimentation, we have adopted the double-broken circle as the best test object for the standard measurement and rating of acuity. Test objects of the broken circle type satisfy extremely well all the essential requirements of a good test object for use in every way in which such an object may be used. 1 The task set to the judgment is ideally simple, yet the resolving power in the different meridians is tested remarkably well. That is, when there is an inequality of resolving power in the different meridians, the effect is to give a false location to the opening or to produce the appearance of openings where they are not. 2 When a sufficient num-

ber of positions is used, the results of the test afford directly a great deal of information as to the correctness of focusing in the different meridians. The test series has therefore considerable analytic as well as general merit. 3 The broken circle, particularly when it is used in connection with variable illumination, as described in our former papers,² is a most excellent test object for the detection of astigmatism. The combination with variable illumination affords the most sensitive test for astigmatism we have as yet had the opportunity to try. 4 When used in positional series the test object provides an ideal objective check on the judgment and one that is effective for children and illiterate subjects. 5 The broken circle is exceptionally well suited for the construction of a rating scale. There is but one type of detail, the break in the circle, to be discriminated, and the form of the test object is such that this detail can be easily measured and accurately and easily constructed in accord with this scale. 6 As was noted in former papers,⁷ test objects of the type of the broken circle show greater sensitivity and give higher reproducibility of result than other test objects commonly used, such as the Snellen letters or the letter E. 7 The broken circle, particularly if a rotatable test object is used, can be presented as often as may be desired in an experimental series without danger of memorization. This feature, as already indicated, is especially important in work of the kind discussed here, namely, the testing of the ability to discriminate detail at low illumination and of the effect of dark adaptation on this ability.

As compared with the Landolt circle, or the circle with a single break, the circle with the double break has the following additional advantages. 1 The two openings, 90 degrees apart on the arc of the circle, provide a more sensitive and a more correct test of the resolving power of the refracting medium than is provided by a test object with a single opening. 2 There is the further advantage that the test in both meridians can be made simultaneously. Simultaneous comparisons are always to be preferred to successive comparisons, particularly when the test is made in the absence of cycloplegia. When the test is made in the absence of cycloplegia, this feature is of great service in preventing what is known as selective or meridional accommodation. 3 The openings may be turned in double the number of directions with a given

2 Ferree, C. E., and Rand, G. (a) Visual Acuity at Low Illumination and the Use of the Illumination Scale for the Detection of Small Errors in Refraction, *Am J Ophth* 3 408-417 (June) 1920, (b) Sensitivity of the Illumination Scale for Determining the Exact Amount and Placement of Correction for Astigmatism, *ibid* 4 22-27 (Jan.) 1921, (c) The Testing of Visual Acuity. II The Comparative Merits of Test Objects and a New Type of Broken Circle as Test Object, *ibid* 17 610-618 (July) 1934.

3 Ferree and Rand ^{2a,c}

number of positions of the circle. The testing of the resolving power in double the number of meridians for any position of the circle is of special value in the measurement of dark adaptation by the acuity method because of the small amount of time that is available for making each determination. It is also of advantage in the ordinary work of testing acuity and of constructing charts for this purpose. In constructing charts, for example, only half the number of objects of the same size is required, and therefore double the number of sizes can be used without increasing the dimensions of the chart. Further, the chart can be used as readily upside down as right side up, so that the effect is given of doubling the number of positions of the openings and changing the order of their presentation. It is of great advantage too in our special test for astigmatism based on the use of the broken circle in conjunction with a variable intensity of illumination. By the use of two breaks, or openings, in the circle the time required for making this test is reduced one half.

The double-broken circle is furnished with our acuity projector both as a single rotatable test object and in the form of an acuity chart. Some of the advantages of this chart are: 1. The test objects have been constructed with great accuracy. In the chart furnished by us, measurements were made to $1/1,000$ inch (0.00025 mm) by means of a micrometer comparator. Similar measurements made on capital letter charts now in use showed errors in construction amounting sometimes to as much as 25 per cent. 2. More than twice the number of sizes of the test object ordinarily used in charts of the capital letter type are provided, so arranged as to permit of the optional use of the extra sizes. 3. In the charts in common use there is a deficiency not only in the number of sizes used but in the distribution of sizes with respect to adequate provision for eyes of poor and better than medium vision and with respect to the variation in acuity with age and intensity of illumination. Due care has been taken in the construction of our chart to remedy this deficiency as far as is feasible.

Charts of the capital letter type do not satisfactorily meet even the most fundamental requirements of a measuring scale. The construction of a measuring scale presupposes the selection of a unit, or portion of the thing to be measured, that is maintained equal in value or magnitude throughout the scale. The capital letters present a multiplicity of units, one for each letter used in the series. In such a situation comparison and evaluations are difficult and numerical ratings impossible. Can any one say that in a chart the task set by a first line which, for example, contains only the letter L sustains any known relation to that set by a second line which contains the letters T and E or to that set by any other line? In different charts the series consists of seven, fourteen, eighteen or twenty different letters of the alphabet distributed

in various ways in the different lines. The situation is further complicated by the fact that the number of letters used in the different lines may vary from one to eight or nine. Thus, considering the individual letters in the line or the line in toto as a division of the scale, the situation is hopeless for the purpose of numerical comparison or measurement. The use of such a plan in the construction of a scale of physical magnitudes would receive scant consideration, and the test letters as such are, it will be remembered, physical magnitudes and can be treated as physical magnitudes. Any attempt to treat the lines of letters on the chart as divisions in a subjective scale would require an enormous amount of empiric calibration, an amount that has thus far been found to be prohibitive in any department of sensory work. Considering the test characters as physical magnitudes, it is obvious that much greater possibilities for rating and measurement are at one's command with the use of a single well selected object of the sizes wanted. With such an object, when all other factors that affect one's powers of discrimination are held constant, a fairly good approximation to a correct numerical rating and a comparative measurement may be made.

Further, in the case of charts of the capital letter type the judgment is in terms of recognition instead of discrimination of detail, and it necessarily has to be because of the type of test object used. To be recognized objects need not be seen clearly. The results obtained are thus not in terms of the function tested and sustain an indeterminate relation to that function. In the use of test objects of the type of capital letters it would not be easy to make a judgment in terms of one or more critical details of uniform and measured size. The judgment in terms of recognition is inherent in this type of test object.

In previous papers⁴ we have discussed and demonstrated the effect of various factors in the determination of acuity. We have not, however, as yet shown the effect of type of judgment, and it is not our intention to do that here in detail. We wish here merely to note the factor and to give some slight indication of the magnitude of its importance. For example, a comparison of the ability to discriminate the opening in a broken circle accurately constructed to the one to five minute scale and the ability to recognize the letter E accurately constructed to the same scale (a favorable letter for this construction)

4 Ferree, C. E., and Rand, G. (a) The Effect of Increase of Intensity of Illumination on Acuity and the Question of the Intensity of Illumination of Test Charts, *Am J Ophth* 6 672-675 (Aug) 1923, (b) The Testing of Visual Acuity I Factors in the Sensitive Use of the Test for the Detection of Errors of Refraction, *ibid* 17 29-36 (Jan) 1934, (c) III Types of Test Field and a Projection Apparatus, *ibid* 17 1147-1150 (Dec) 1934, (d) Age as an Important Factor in the Amount of Light Needed by the Eye, *Arch Ophth* 13 212-226 (Feb) 1935 (e) footnote 2c

showed a difference in the rating of acuity of the order of 15 per cent, the rating being that much higher when made with the letter E. This difference can be considered to be due almost if not entirely to difference in the type of judgment.

It may further be noted that the test of power to recognize belongs of necessity dominantly to the class of what are called specific performance tests, not to the class of basic tests of capacity, that is, the result will vary with the object that has to be recognized. For example, as already noted, equal power is not had for the recognition of different capital letters, even when these letters are constructed to the same scale. Further, this power will not be the same for other objects as for capital letters. Obviously, the power to recognize varies both with the type of object and with the individual person over and above the power to discriminate detail.

While it may be worth while to test the power of recognition in a rating of human capacities, the test has to be made for each object separately. The results obtained for no one object alone can be taken as measuring the individual's power to recognize. Because of the low transfer value of the results obtained, little general importance is usually ascribed to such tests and they are used no more than can be helped in a program of testing. It can thus readily be seen that it is wrong to make the basic test of vision in terms of the power to recognize and not well to substitute a test of the power to recognize for a basic test of vision. From the standpoint, then, both of correctness of principle and of reproducibility of result from person to person and from time to time with the same person, a type of test object should be employed with which the judgment of power to discriminate detail not only can be conveniently used but is required.

It is clear that the greatest possible care should be exercised in the selection and the construction of characters which are to be used for the testing of visual acuity. The test objects and the test characters, not the projector or other means of presenting them, are the foundation of the test. With charts constructed in a hit or miss fashion—some made up of authentic Snellen letters, some made up of capital letters not constructed to meet the one to five minute requirement of the Snellen scale and not of the same order of discriminability in different charts, some made up of other types of test object and all too often inaccurately constructed as to size—the situation in acuity testing is chaotic. Is it any wonder that in the air service, army, navy and commercial, and in many similar fields the complaint is made that tests conducted at different stations and at different times do not give the same rating of acuity? It is a recognition of this situation that leads to the appointment of committees for the standardization of test charts. That these committees have to be appointed over and over again is due

largely to the fact that a proper rating scale can never be obtained with capital letters as test objects

In stating results, must one always be required to give a detailed specification of the kind of chart used, or should not the endeavor be made to get a test object and a test chart that best satisfy the requirements of a rating scale and adopt and use them as standard? For the test object we have recommended the double-broken circle, the critical details of which, the two breaks in the circle, can be constructed to meet absolutely the one minute requirement for such details

In the use of the equipment recommended for measuring the eye's ability to see at low illumination and its power of dark adaptation, either the slide containing the double-broken circle test objects of different sizes or the single rotatable test object may be employed. As a routine test for fitness for night flying we strongly recommend the use of the single rotatable test object

THE PRELIMINARY LIGHT ADAPTATION OF THE EYE

One of the most discussed points in the study of dark adaptation or of its effect on any of the visual functions is the standardization of the preliminary light adaptation. This standardization consists in the choice of the intensity of light and the time the eye is to be exposed to it before any of the determinations of the sensitivity of the eye in the dark are made. The tendency in general is to choose a longer time of preexposure than is needed in most cases to standardize the sensitivity of the eye. This tendency is due to the fact that the choice is usually based on a knowledge of dark adaptation alone. Investigation shows that light adaptation takes place much faster than dark adaptation. In a later paper the results of a study of light adaptation will be published, including curves for a comparison of dark adaptation and light adaptation. These results show (a) that in general light adaptation takes place much faster than dark adaptation, (b) that light adaptation takes place rapidly for the first few minutes and then slowly, soon becoming negligible unless long periods of adaptation are compared, and (c) that the sensitivity which was gained in twenty-five minutes of dark adaptation after three minutes of exposure to the pre-exposure field of our instrument for testing the light sense,⁵ which instrument was used in obtaining the results, was lost in three minutes of subsequent exposure to the same field and that the further loss of sensitivity by exposure to this field was comparatively little, becoming negligible after five or ten minutes. The rapid loss of sensitivity at the beginning of light adaptation is shown by the fact that in the first

⁵ Ferree, C. E., and Rand, G. A New Type of Instrument for Testing the Light and Color Sense, *Am J Ophth* **14** 325-333 (April) 1931

fifteen seconds, the interval represented by the nearly vertical section of the curve, the light minimum was increased more than 100,000 per cent. For the first five minutes, the interval representing the section extending just beyond the knee of the curve, the increase was 227,173 per cent, for the second five minutes, 34 per cent, for the third five minutes, 4 per cent, for the fourth five minutes, 5 per cent, and for the last five minutes, 1 per cent.

We can perhaps best discuss the question of preadaptation, or pre-exposure, by giving some of our own experiences. When we devised our instrument for testing the light sense, for example, our thought was to provide in the instrument a preexposure field the intensity of which could be controlled and made constant for all time and at all places. Whether this could be considered to give sufficient light adaptation, we did not at that time know. We began our work with the instrument by allowing twenty minutes of preadaptation in a room with white walls and ceiling, illuminated to a given intensity with well diffused light, the observer reading from a page the intensity of which was also measured and kept constant. The observer was then taken into the dark room and located in position at the instrument as quickly as possible. Further to standardize the sensitivity of the eye, three minutes of exposure was then given to the preexposure field of the instrument before the test was begun. These conditions were found to give a wide range of dark adaptation and a high reproducibility of result with all our subjects except those who had been confined for some days in a darkened room. For them a longer period in the light room was needed. In later work we found that unless the observer had been confined for some time in a darkened room, the use of the three-minute preexposure alone gave no difference in the adaptation curves, except possibly in respect to the initial determination of the light minimum and the value obtained at the end of the first minute of dark adaptation. For tests, therefore, in which the results for the first minute are not important, and perhaps for all tests, a preexposure of from three to five minutes to the illuminated field of the instrument should be sufficient unless the observer has previously experienced some unusual situation with respect to adaptation, that is, has been exposed for a considerable length of time to high intensity of light, as might be the case if he was brought in directly from the flying field or had undergone a considerable period of dark adaptation. Similarly, therefore, we consider that a pre-exposure of from three to five minutes to the intensity of illumination selected for this purpose should be adequate in all cases for the determination of acuity in dark adaptation unless there has been some unusual situation with respect to adaptation, such as one of those mentioned. In any event, it should not be difficult for the individual examiner to determine for himself whether or not the preexposure is adequate

in any situation that may arise. In determining this point, reproducibility of result is of course an important consideration. In this connection it should be remembered too that determinations of acuity in dark adaptation will not be affected as much by the length of the preexposure to light as would determinations of the light minimum.

REPRESENTATIVE RESULTS

As we have already stated, the Ferree-Rand acuity projector was devised especially for the testing of visual fitness for the lookout service at night on the bridge of a battleship. We recommend it as being equally well suited for the testing of night flyers. The following results may be regarded as representing the type of work that may be done with this instrument.

In a test made by us of 61 observers all under 28 years of age and all rating 6/4 by the conventional acuity test with 5 foot candles of light on the test chart, 13 per cent rated less than 6/6 with 0.55 foot candle and 33 per cent less than 6/6 with 0.2 foot candle. The acuity of the remainder was 6/6 or better at these illuminations. If speed in the use of the eye at low illuminations is added to the requirement, the scatter is much greater still. The amount of time required just to discriminate one minute of visual angle by this group of observers, who were all put in the same class by the acuity test at 5 foot candles, covered a range from fastest to slowest of 1.333 per cent at 0.55 foot candle and 1.443 per cent at 0.2 foot candle. These values are cited here to show that a greater differentiation of observers with respect to acuity will be had if the test is made at low illumination.

The curves in figure 1 are representative of the type of work that may be done in studying dark adaptation with the instrument. For this work 21 observers were used, ranging in age from 18 to 28 years and all rating 6/4 by the conventional acuity test at 5 foot candles. For these curves the amount of light was determined that was needed for the subject to discriminate the opening in a broken circle turned in different directions. This opening subtended one minute of visual angle at the eye. The amount of light required for the discrimination was determined at the beginning of the period of dark adaptation and at the end of intervals of fifteen, thirty and forty-five minutes. There was of course some variation in the time consumed in completing a determination. Because of this the results obtained at the beginning of dark adaptation show a greater variation than those obtained later in the adaptation process, since the exact time at which the determination is completed exerts a greater influence on the results obtained at the beginning of the period, when the rate of change of sensitivity is at its maximum. In conducting the test care was taken to reduce this variability to a minimum. In figure 1 are given the adaptation curves

derived from the highest, the lowest, the 50 percentile (median), the 25 percentile (first quartile) and the 75 percentile (third quartile) values at each period tested. The distance between the limiting curves represents the total range of values, and that between the 25 and 75 percentile curves, the middle 50 per cent range of values. The amount of light needed to discriminate the test object expressed in foot candles is plotted along the vertical coordinate and the time of adaptation in minutes along the horizontal coordinate.

An examination of the data shows that the scatter in the results at the beginning of the period of dark adaptation and at the end of fifteen, thirty and forty-five minutes of adaptation averages for the entire group 800 per cent and for the middle 50 per cent of the group 159 per cent. It should be remembered, however, that none of the observers was above 28 years of age. Much greater individual differences would be expected had older observers been included in the group.

In figure 2 individual curves for six of the observers are plotted for intervals of five, ten, fifteen, twenty-five, thirty-five and forty-five minutes of adaptation.

It will be noted in figures 1 and 2 that for the greater number of observers the light minimum for the discrimination of detail was lowest at the end of fifteen minutes of dark adaptation and that in some cases it even increased if the series was continued beyond this time. The increase in these cases was doubtless due to fatigue of the muscles of adjustment and to other physiologic disturbances, that is, in the case of observers more susceptible to these influences, the loss in power to discriminate detail more than compensated for the slight gain in sensitivity to light after the first fifteen or twenty-five minutes. In this connection it may be noted that the muscular strain imposed by determining acuity is much greater at the minimum illumination than at the illumination ordinarily employed. Even with a rest period of five or ten minutes between determinations and an interval of two seconds between the individual observations making up one determination, noticeable fatigue was present at the end of a series lasting forty-five minutes.

In relation to the problem in hand, fitness for night flying, it may be of interest to note certain characteristics of the curves shown in figure 2. Curves I and II are the only ones that make a satisfactory showing on all points that are important with respect to fitness for night flying, namely, superior rating in power to see at low illumination both at the beginning and at the end of the period of dark adaptation and a normal or better than normal rating as to amount and speed of adaptation. The remaining curves are defective with respect to some or all of these points. Curves III and IV, for example, show only a medium and curve V a poor rating in power to see at low illumination at the

beginning of the period of dark adaptation, while curve VI shows a very poor rating in power to see at low illumination, not only at the beginning of dark adaptation but throughout its entire course

A ROUTINE TEST OF FITNESS FOR NIGHT FLYING

If a more specific recommendation were wanted for a routine test of fitness for night flying, the following one might be suggested. After a suitable period of adaptation, perhaps from three to five minutes, to an intensity of light selected for the purpose, the adaptation light should be turned off and the minimum light at which the test object can be discriminated determined with the projector. In making this determination one should start the intensity low and increase it quickly until the discrimination can just be made. As an objective check on the judgment the observer is required to state the direction in which the two openings of the test object are turned, and as a means of preventing the learning of these directions, the opening may be rotated into any position within 360 degrees. After two, three or five minutes of adaptation, the determination is repeated. From the results may be determined the minimum amount of light required to discriminate the test object with the light-adapted eye, the rate of change in this amount for a given period of dark adaptation and the amount at the end of this period. This would seem to give the most important information needed, namely, the power of the light-adapted eye to see objects at low illumination, the power that is acquired after a selected period of dark adaptation and the rate of change in this power as affected by dark adaptation. This information would enable the examiner to exclude eyes defective in power to see at low illumination when either light adapted or dark adapted (e g, eyes affected by hemeralopia or avitaminosis) and to select the best of the normal eyes.

For deciding which is the best of any set or number of test methods under consideration, either of the following procedures may be used. (a) Two groups of aviators may be selected for examination, one whose success in night flying has been proved and the other whose unfitness has been demonstrated. The test which shows the widest difference in result between the two groups should be considered as the most sensitive and significant. (b) All entering candidates who have passed the other tests of ocular fitness for aviation may be tested and the results of the test compared with their subsequent success in night flying.

Critical values to serve as a standard of reference for the acceptance and the rejection of candidates for night flying may be obtained from the results of either of these procedures. With the first, separate distribution curves may be plotted from the test data obtained for the successful and the unsuccessful night flyers, and from the overlapping

area of these curves results may be chosen which shall be accepted as borderline or critical. With the second, a distribution curve may be plotted from the test data obtained for all the entering candidates who have passed the other tests of ocular fitness for aviation, and on the basis of this curve a certain percentage of candidates may be selected as best qualified for night flying. The selection thus made may be improved by checking against subsequent records in night flying, and the critical values may be revised accordingly. On the basis of our meager data we might hazard the suggestion that for a detail of one minute of visual angle (size for 20/20 vision, Snellen scale), 0.1 foot candle might be taken as the critical value at the beginning of the period of dark adaptation and 0.05 foot candle at the end of five minutes of adaptation. Of the six observers represented in figure 2, 2 would be acceptable in terms of this criterion.

COMPARISON OF VISUAL ACUITY AT LOW ILLUMINATION AND LIGHT SENSE AS A MEANS OF TESTING

1 For testing sensitivity to light under any condition of adaptation, the test of the light sense is of course the direct measure.

2 For testing the power to discriminate detail in objects at any intensity of light or under any condition of adaptation, the acuity test is the direct measure.

3 For determining the amount, or range, and the speed of dark adaptation and for determining individual differences in these respects, the test of the light sense when made with our instrument has much greater sensitivity than the test for acuity and greater feasibility as well. As the night flyer is required to discriminate not only faint light and faint differences in light but detail in objects, perhaps both methods are needed.

4 The power to discriminate differences in light and brightness is an important factor in the power to discriminate detail in objects, but so far as we know it is not the dominant factor. The dominant factor is discrimination of space. The effect on the power to discriminate detail in objects cannot, then, be inferred quantitatively from any change, variation or difference in the light sense.

5 The power to discriminate detail at low illumination is strongly affected by age, much more strongly than the power to sense light. The acuity test at low illumination, therefore, offers greater possibilities for detecting visual unfitness due to age than does the test for the light sense.

6 The refractive condition, the space sense, the light sense and the power of dark adaptation are all factors influencing the results obtained with the acuity test at low illumination. The results for this test are

therefore subject to greater irregularities and variations than those obtained with the test of the light sense. In the test of the light sense with our instrument, it will be remembered, all defects in the formation of the image on the retina are practically eliminated by the use of a small pupil in the eye piece.

7 In respect to the light sense the eye is compounded of two, a day-seeing eye and a night-seeing eye, modified to suit the combined function. Roughly speaking, the fovea, provided with a highly developed space sense and a comparatively low power of dark adaptation, may be regarded as the day-seeing component and the paracentral and midregions of the retina, provided with a poorer space sense and a high power of dark adaptation, as the night-seeing component. With the test of acuity it is the foveal part of the retina that is tested. With the test of the light sense a much larger area of the retina may be examined. For our instrument this area ranges from near zero to 36 degrees. The stimulus customarily used by us subtends 3 by 10 degrees of arc at the eye and is made rotatable to provide an objective check on the judgment.

A conclusion as to which method is preferable for the testing of night flyers will have to depend on the results that are obtained in the use of these two methods in the actual testing of night flyers with the equipment now available.

SUMMARY

In determining fitness for night flying, important functions to be tested are (a) the ability to see at night and at low illumination and the effect of dark adaptation on this ability and (b) the amount and speed of dark adaptation. In the latter function, the speed of adaptation seems to be more important than the amount. The night flyer needs especially the power to change his vision quickly from the illuminated cockpit and instrument panel to the outside world and back again. Normal or better than normal sensitivity in light adaptation is of course also important. The eyes that are needed for night flying are the best of what might be called the normal group, that is, of those that have both good dark and good light vision. More important than speed and range of adaptation, however, is the place in the scale of sensitivity in which the change occurs. That is, it is quite possible that a candidate might have a good range and speed of adaptation and still a poor power to see at low illumination both at the beginning and at the end of the period of dark adaptation. Such a person would be obviously unfit for night flying. To be fit for night flying the candidate must have a normal or better than normal rating in power to see at low illumination at the beginning of the period of dark adaptation and throughout its course.

Two functions are involved in the ability to see, namely, the ability to sense light, or to discriminate differences in light and brightness, and the ability to discriminate detail, or what is commonly called visual acuity. Either of these may be used in testing the ability to see at low illumination and the effect of dark adaptation on this ability. Tests are described in which visual acuity at low illumination is used for this purpose and for testing the amount and speed of dark adaptation. A special test of fitness for night flying, which is sufficiently quick and convenient for use in routine testing, is recommended, and a procedure is discussed for proving the significance of the test and for determining the critical values to be used in accepting or rejecting candidates for night flying. A suitable instrument and test object⁶ are recommended and their advantages for making the test briefly discussed. A comparison is made of the merits of visual acuity and those of the light sense as a means of testing fitness for night flying.

⁶ The following studies have been made with this instrument and test object at the School of Aviation Medicine, Randolph Field, Texas, under the direction of Major John M. Hargreaves. Allman, T. L., and Jenkins, P. H. A Test for Night Visual Efficiency, *Flight Surgeon Topics* 2 40-46 (Jan.) 1938. Hargreaves, J. M. A Test for Nocturnal Visual Efficiency, *ibid* 2 74-75 (April) 1938. Platt, R. J., and Griffis, L. E. Report on Test for Night Visual Efficiency, *ibid* 2 90-93 (April) 1938.

BIOCHEMISTRY OF THE LENS

VI EFFECT OF GALACTOSE ON PERMEABILITY OF THE CAPSULE OF THE LENS

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AND

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CHICAGO

No satisfactory reason has as yet been advanced for the phenomenon of lactose and galactose cataract in rats, as first observed by Mitchell and Dodge¹. One possible explanation is the injurious effect of galactose on the capsular epithelium, as reported by Kirby².

In an attempt to find a more likely explanation, we undertook the investigation of the effect of galactose on the permeability of the capsule of the lens. It is obvious that any abnormality in this membrane might lead to changes in the lens itself, because the lens, being a nonvascular organ, must depend on its immediate surroundings for its supply of nutrients. Substances in the aqueous humor when entering the lens must pass through the capsule of the lens, and waste products from the lens must penetrate the capsule in the opposite direction.

Some investigators have considered abnormalities in capsular permeability as important factors in the production of cataract. Reports on the study of the permeability of the capsule of the lens have been made by Hess,³ Friedenwald,⁴ Gifford, Lebensohn and Puntenny⁵ and

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This article forms part of a report for which the Lucien Howe Gold Medal was awarded to Dr. Bellows.

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1 Mitchell, H. S., and Dodge, W. M. *J. Nutrition* **9** 37, 1935. Mitchell, H. S. *Proc. Soc. Exper. Biol. & Med.* **32** 971, 1935.

2 Kirby, D. B., and Wiener, R. von E. *Tr. Am. Acad. Ophth.* **37** 142, 1932. Kirby, D. B., Estey, K., and Wiener, R. von E. *ibid.* **37** 196, 1932.

3 Hess, C. *Pathologie und Therapie des Linsensystems*, in von Graefe, A., and Saemisch, E. T. *Handbuch der gesamten Augenheilkunde*, ed. 3, Leipzig, Wilhelm Engelmann, 1911, vol. 6, p. 35.

4 Friedenwald, J. S. *Permeability of the Lens Capsule*, with Special Reference to the Etiology of Senile Cataract, *Arch. Ophth.* **3** 182 (Feb.) 1930, *Permeability of the Lens Capsule to Water, Dextrose and Other Sugars*, *ibid.* **4** 350 (Sept.) 1930.

5 Gifford, S. R., Lebensohn, J. E., and Puntenny, I. S. *Biochemistry of the Lens. Permeability of the Capsule of the Lens*, *Arch. Ophth.* **8** 414 (Sept.) 1932.

Borley and Tainter⁶ Hess showed that the normal capsule is fully permeable to water and electrolytes. Friedenwald in experiments on removed capsules demonstrated that the capsule acts as a semipermeable membrane and that the ease with which substances pass through it varies in relation to the size of the molecule. He also showed that permeability decreases with the age of the animal. Gifford and his co-workers in experiments on the capsule *in situ* confirmed Friedenwald's observations and furthermore showed that the permeability of the capsule of a cataractous lens was approximately the same as that of a normal lens. They also found that albumin and glutathione diffuse through the capsule of the lens with difficulty, whereas globulin does not pass through until postmortem changes have affected the capsule. Borley and Tainter obtained negative results when attempting to explain dinitrophenol cataract on the basis of altered capsular permeability.

Since we had previously found the glutathione content of the lens to diminish rapidly on feeding galactose, we thought that this substance may have diffused through the capsule of the lens because of increased permeability caused by galactose. However, the data herein published prove the reverse.

EXPERIMENT

An adaption of Friedenwald's technic, similar to that used by Borley and Tainter, was employed by us in order to determine the effect of galactose on capsular permeability. The capsule of a fresh beef lens was carefully removed after a posterior cruciate incision and was freed from adherent lens material by repeated shaking with physiologic solution of sodium chloride. The membrane was then tied over the flare-out end of a glass tube 5 mm in diameter, with its inner surface facing the tube. In the tube was placed 1 cc of a solution prepared with approximately 400 mg of glutathione in 100 cc of an 0.86 per cent solution of sodium chloride. We selected glutathione as the test substance because of its fairly large molecular size. It was noted that it passed through the capsule of the lens at a slow rate *in vitro*, and its rate of diffusion through the membrane, therefore, should change with any variations in capsular permeability. The tube was then immersed in a solution containing the substance whose effect on permeability was to be tested (as shown in the table). The level of the solution of glutathione in the tube was kept 2 cm above that of the outer solution. The ability of the capsule to support this pressure for the duration of the experiment indicated its freedom from any leak. The solutions inside and outside the tube were buffered to physiologic reaction with phosphates and kept at room temperature. Differences in osmotic pressure were eliminated by adjusting the concentration of the solution of sodium chloride.

From five to six hours later the amount of glutathione in the tube as well as that in the outer medium was determined. This afforded an index to the relative permeability of the capsule of the lens. The greater the permeability, the greater the amount of glutathione found in the outer solution and the less remaining in the tube. The converse is also true. Since the glutathione in the tube

⁶ Borley, W. E., and Tainter, M. L. Effects of Dinitrophenol on the Permeability of the Capsule of the Lens, *Arch. Ophth.* **18**: 908 (Dec.) 1937.

would undergo a smaller degree of oxidation because of its higher degree of concentration and lesser surface of exposure to the air than that found in the outer medium, we considered the determination of the amount in the former to be the better criterion for judging permeability

RESULTS

The results of the experiments can be seen from a study of the accompanying table. When approximately 4 mg. of glutathione in solution is placed in each tube of a series and immersed in the various mediums, and when the former is separated from the latter by the capsule of a lens, the greatest loss of glutathione occurs in the tubes immersed in physiologic solution of sodium chloride, less is lost in the tubes immersed in dextrose solution, a lesser amount is lost in those immersed in galactose solution, and the least amount is lost in those immersed in a combination of all three mediums. Capsules from beef eyes kept in the refrigerator overnight showed a decrease in their permeability.

COMMENT

It can be seen from our results that galactose and, to a slightly less extent, dextrose cause a definite retention of glutathione by the capsule of the lens. There is no reason to believe that a similar action should not take place *in vivo*. The conditions prevailing in experiment 3, setup E, in which the surrounding medium consisted of a 0.9 per cent solution of sodium chloride, a 0.1 per cent solution of dextrose and a 0.3 per cent solution of galactose, most closely approximate the actual findings in the aqueous humor of animals which have been placed on a galactose diet (as shown by Sasaki⁷). It even appears possible that dextrose and galactose have a synergistic action, although there are not enough experiments to prove this point as yet. From these data we can reasonably accept a decreased permeability of the capsule of the lens as a factor in the genesis of galactose cataract.

Further support of the effect of galactose in diminishing the permeability of the capsule of the lens is afforded by the work of Sasaki. He has shown that the lenses of rats fed dextrose contain a high amount of reducing sugar, while those of rats fed galactose possess only normal amounts, this occurs in spite of the fact that the aqueous humor of rats fed galactose contains much greater amounts of reducing sugar than that of rats fed dextrose. It seems to us that these findings afford additional proof that galactose causes a decreased permeability of the capsule of the lens.

That rats on a diet high in dextrose do not develop cataract as do those on a diet high in galactose may be explained by the relative blood

⁷ Sasaki, T. Personal communications to the author.

Effect of Galactose and Dextrose on the Permeability of the Capsule of the Lens

Experiment No	Time, Hours	Mg of Glutathione Added	A		B		C		D		E	
			Inside Tube	Outside 0.9% NaCl (5 Ce)	Inside Tube	Outside 0.8% NaCl, 0.3% Galactose	Inside Tube	Outside 0.8% NaCl, 0.5% Dextrose	Inside Tube	Outside 0.9% NaCl, 0.1% Dextrose	Inside Tube	Outside 0.9% NaCl, 0.1% Dextrose, 0.3% Galactose
1	6	3.39	2.72 2.44 2.34	0.27 0.33 0.45	3.27 3.27 3.30	0.01 0.12						
Average Glutathione passed			2.50 26%	0.35	3.28 3%	0.08						
2	5½	3.43	2.55 2.64 2.60	0.24 0.53 0.52	3.43 3.42 3.31	0.03 0.03 0.08						
Average Glutathione passed			2.60 24%	0.43	3.39 1%	0.05						
3	5½	3.99	2.31 2.36	0.68 0.70	2.88 2.90	0.65 0.54	2.76 2.74	0.63 0.68	2.50 2.31	0.81 0.83	2.98 3.60	0.51 0.20
Average Glutathione passed			2.33 41%	0.69	2.89 27%	0.59	2.75 31%	0.65	2.40 40%	0.82	3.29 17%	0.35
4	6	3.86	2.83 3.00	0.52 0.50	3.37	0.39	2.94 3.19 2.91	0.55 0.41 0.53				
Average Glutathione passed			2.91 24%	0.51	3.37 12%	0.39	3.01 22%	0.50				

sugar findings in these animals. Day⁸ pointed out that the blood sugar of rats on a diet high in dextrose rose only to 121 mg per hundred cubic centimeters, showing dextrose to be readily utilized by rats. Galactose, however, is utilized with difficulty by rats fed this substance, the blood sugar reaching 372 mg per hundred cubic centimeters. Sasaki found corresponding values in the aqueous humor of rats. Mitchell and Cook⁹ have shown that the increase in the blood sugar content was due to galactose.

However, in conditions in which the normal metabolism of dextrose is disrupted and consistently high values for the dextrose content of the blood are found, such as in pancreatectomized dogs or in young persons who are subject to severe diabetes, cataract is not uncommon and may be explained on the same basis. Diabetic cataract has previously been explained without experimental evidence either on a basis of osmotic effect or on a basis of accompanying acidosis. However, the explanation indicated in this report appears more likely in view of actual data obtained.

SUMMARY AND CONCLUSIONS

The effect of galactose and dextrose on the permeability of the capsule of the lens was investigated. It was found that galactose caused a marked decrease in the capsular permeability, a similar effect was produced by dextrose, but to a lesser extent. The significance of these findings in relation to galactose cataract in rats and diabetic cataract in the human being is discussed.

The foregoing experiments were repeated with a physiologic solution balanced with respect to sodium, potassium, calcium and magnesium ions instead of the sodium chloride solution. The results were essentially the same as those reported except that the effect of dextrose on the permeability of the lens capsule approached more nearly that obtained with galactose.

8 Day, P. L. *J. Nutrition* **12** 395, 1936.

9 Mitchell, H. S., and Cook, G. M. Galactose Cataract in Rats. Factors Influencing Progressive and Regressive Changes, *Arch. Ophth.* **19** 22 (Jan.) 1938.

Clinical Notes

DACRYOCYSTITIS IN LYMPHATIC LEUKEMIA

WILLIAM H. STOKES, M.D., OMAHA

It has been recognized for many years that certain specific ocular lesions may occur at some stage during the course of leukemia of both the lymphocytic and the myelogenous form. These changes have been observed more commonly in the retina, but their presence has also been noted in the conjunctiva, choroid, orbital tissue and lacrimal glands. It is of particular interest to note, in contrast, the infrequency with which leukemia has involved the lacrimal sac and in so doing has given rise to signs and symptoms of chronic dacryocystitis. A search of the literature revealed only 1 such case, reported by Creutz.¹ Bilateral lymphoma of the lacrimal sac was reported by Sulzer and Duclos,² Pascheff³ and Weve,⁴ but there were no associated changes in the blood characteristic of the leukemias in these cases.

When one considers the remarkable change which the blood undergoes in leukemic states it is not difficult to assume that certain secondary changes may occur in the endothelium of the blood vessels and so disturb its function that the cellular elements of the blood escape into the perivascular tissues by diapedesis. It is not known what precisely occurs to cause this phenomenon, but there are two theories, both of which sound plausible and each of which has its advocates. Stock⁵ advanced the view that an infiltration of leukocytes occurs into the tissues, with local proliferation and multiplication of the cells in the perivascular spaces and contiguous tissues. Meller,⁶ however, attributed the pathologic changes to a stimulation, with subsequent overgrowth of preexisting lymphoid elements.

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1 Creutz, cited by Henke, F., and Lubarsch, O. *Handbuch der speziellen pathologischen Anatomie und Histologie*, Berlin, Julius Springer, 1931, vol. 7, pt. 2, p. 347.

2 Sulzer, E., and Duclos, L. *Lymphome double du sac lacrymal, suite de lymphadénie généralisée sans leucémie*, *Rec. d'opht.* **28** 353, 1906.

3 Pascheff, C. *Symmetrical Lymphoid Tumor of Lacrimal Sacs*, *Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch.* **46** 433, 1927.

4 Weve, H. *Ein lymphatischer Tumor des Tränensackes*, *Nederl. tijdschr. v. geneesk.* **72** 696, 1928.

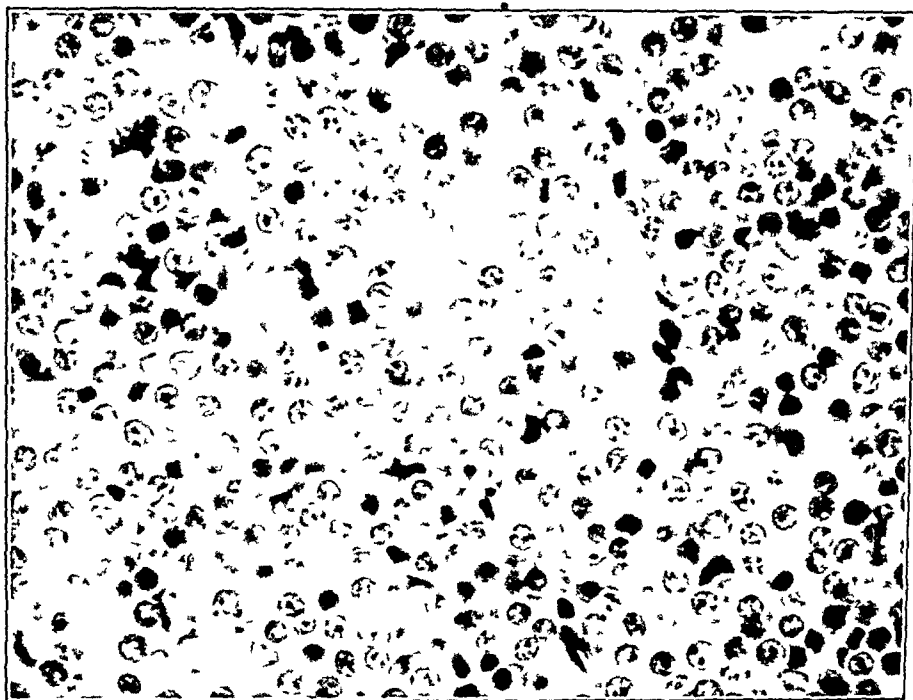
5 Stock, W. *Ueber Augenveränderungen bei Leukämie und Pseudo-Leukämie*, *Klin. Monatsbl. f. Augenh.* **44** 328, 1906.

6 Meller, J. *Die lymphomatösen Geschwulstbildungen in der Orbita und im Auge*, *Arch. f. Ophth.* **62** 130, 1906.

The case reported in this communication is so unusual that its inclusion in the literature seems desirable because of its singular rarity and also because its pathologic aspects favor the assumption of overgrowth of lymphoid tissue in a situation where this tissue is but slightly in evidence

REPORT OF A CASE

Mrs E S S, aged 68, examined on Feb 14, 1938, gave the following history Both eyes began to water about one and one-half years before examination Twelve months before the region over the tear sacs began to swell, and abscesses formed on each side of the nose, which were lanced by a local physician This procedure gave palliative relief for about one month, but the swelling recurred



Photomicrograph of a section of the wall of the tear sac, showing intense infiltration by lymphoblasts and lymphocytes in a case of chronic dacryocystitis associated with lymphatic leukemia Photo-ocular, 15, objective, 4 mm—high dry, magnification, 600

at this time and again within four weeks At each recurrence the abscesses were evacuated by simple incision, resulting in permanent drainage through fistulous tracts This condition was present when the patient was referred to me for treatment, approximately one year after the onset of her illness

Examination showed bilateral chronic dacryocystitis, with the formation of a fistula The skin in the neighborhood of the fistula on each side was reddened, and through the fistula there exuded thick mucopus There was a symmetric enlargement of the lacrimal glands, easily recognized by palpation

The patient was referred to Dr Harry B Stokes for rhinologic examination, who reported that there existed an old chronic hyperplastic ethmoiditis and maxillary sinusitis, due in part to a badly deviated nasal septum which interfered markedly with normal ventilation and drainage of the nose and paranasal cavities

The changes in the nose and sinuses were so advanced that conservative medical therapy seemed futile. A submucous resection of the nasal septum as a preliminary measure to any treatment was clearly indicated, but the age of the patient and her general physical condition militated against this procedure. The opinion was expressed that in view of the pathologic process in the nose and in the sinuses, extirpation of the tear sacs would be attended with better results than dacryocystorhinostomy.

Extirpation of the lacrimal sac on the right side was performed on February 15 and of that on the left side on February 22, local anesthesia being used. It was noted during the operation that there was little bleeding and that the tissues were unusually edematous. The tear sacs were large, the walls were markedly thickened and gelatinous and were friable and easily torn.

The tissue was sent to the laboratory of Dr. Charles P. Baker, of the Nebraska Methodist Hospital, who made the following report. Tissue sections revealed marked infiltration of the wall of the tear sac with cells which appeared to be lymphocytes and lymphoblasts, causing a marked thickening of the structures. The tissue had the appearance of a lymphocytoma or a leukemic infiltration. There was a superimposed chronic pyogenic inflammation, which is shown in the accompanying photomicrograph. The blood count on February 20 revealed the following changes: hemoglobin, 65 per cent, red blood cells, 3,470,000, white blood cells, 52,900, polymorphonuclears of the segmented form, 8 per cent, and of the staff form, 2 per cent, lymphocytes, 90 per cent, monocytes, 2 per cent, reticulocytes, 0.8 per cent, and degenerated cells 23 per cent. On February 26 there were 72,800 white blood cells, with 90 per cent lymphocytes and 27 per cent degenerated cells. The foregoing changes in the blood supported the diagnosis of lymphatic leukemia.

The general physical examination showed a slight but distinct enlargement of the glands of the neck, axilla and groin. These glands were hard, but not tender, and were movable under the skin. The lacrimal glands were also enlarged and palpable. There was no splenic enlargement noted. The blood pressure was 200 systolic and 92 diastolic. The urine contained some albumin and casts.

Examination of the fundus showed no evidence of leukemic retinitis. Vision was 20/30 in each eye.

COMMENT

This case again demonstrates the importance of a careful examination of the patient, even though the treatment of chronic dacryocystitis in the great majority of patients is obvious. It also emphasizes the fact to which I have called attention in a previous communication,⁷ that in all cases in which dacryocystorhinostomy is contraindicated, extirpation of the tear sac should be the method of election rather than destruction of the sac with cauterizing agents. Cauterization of the tear sac with the various chemical agents in vogue may at times be successful, but it does not carry with it the finality of extirpation. Moreover, cauterization by the very nature of its action will obscure the fundamental pathologic processes in those cases in which correct diagnosis is of the utmost importance. In conclusion, I should like to reiterate that in every case in which the tear sac is to be dealt with radically it should be removed and examined routinely under the microscope.

⁷ Stokes, W. H. Refinements in Tear Sac Surgery, Nebraska M. J. 20:388, 1935.

OCULOGLANDULAR TULAREMIA

IRVING I CRAMER, M D, CLEVELAND

E F, a school boy aged 16, was first seen in the ophthalmic department of the Cleveland City Hospital on Nov 29, 1937, complaining of pain and swelling of the right eye and the presence of painful kernels in the right side of the neck.

The boy went hunting on November 16 and 22, catching and subsequently dressing a number of rabbits. He stated that he wore gloves while hunting and dressing the animals and that neither his gloved hand nor any of the material came in contact with his eyes. However, two days after the second hunt the right eye became slightly irritated. The patient caught more rabbits on the morning of November 27. During the hunt and the dressing process he kept rubbing his irritated eye. That night he had a moderately severe chill and began to complain of headache and aching of the muscles of the back and legs. The right eye became intensely painful, and by morning the lids were markedly swollen. The right side of the neck was painful, and the patient could feel some small tender kernels



Appearance of the patient's eye

Examination revealed an acutely ill boy with a temperature of 39.4 C (102.9 F). The lids of the right eye were red, swollen and edematous. A small amount of seropurulent discharge was present, causing the lids to stick together. The palpebral conjunctiva was deep red, granular and slightly edematous and that of the lower lid contained about eight or nine rather sharply demarcated, shallow yellow ulcers, from 1 to 2 mm in diameter. The bulbar conjunctiva was moderately injected, and the cornea was clear. Examination of the deeper structures showed them to be normal. Many large, firm, tender nodes were palpable in the left preauricular and the anterior cervical region. Nodes were palpable in the left side of the neck, in both axillae and in the groins, but they were not tender. The mucous membrane of each side of the nose was intensely congested, and there was a moderate mucopurulent discharge. The pharyngeal walls were deeply injected, and the lymphoid tissue was hyperplastic. The spleen was slightly enlarged.

Because of the typical history and physical findings, a diagnosis of oculoglandular tularemia was made.

Examination of the blood showed 4,000 red blood cells and 7,100 white blood cells, with 80 per cent hemoglobin, 76 per cent polymorphonuclears, with a marked

shift to the left, and 24 per cent mononuclears. The Wassermann and Mantoux reactions were negative. Agglutinations for *Bacillus tularensis* on November 29 were negative in dilutions of 1:40 to 1:640.

On November 30 the patient was given 15 cc of antitularia serum intravenously. This was repeated on December 1 and again on December 2. On each of these three days he received 100 roentgens of radiation to the right side of the neck. The temperature, which had risen to 40.5 C (104.9 F), began to descend slowly, until on December 10, when it reached 38 C (100.4 F), serum sickness developed. On December 3 and 8 agglutinations for *B. tularensis* were still negative. During this time local treatment consisted of the application of moist heat, irrigations of a solution of boric acid and applications of a 1 per cent solution of silver nitrate. The condition of the eye began to improve slowly after the institution of local treatment, but the general condition of the patient remained unchanged. Small shallow yellow ulcers identical with those seen on the conjunctiva developed on the lymph follicles of the posterior pharyngeal wall. Roentgenograms showed a haziness of the frontal and maxillary sinuses. Pharyngeal smears were negative, but cultures could not be obtained.

On December 13, fourteen days after the patient was admitted to the hospital, agglutinations for *B. tularensis* were positive in dilutions of 1:40 to 1:160. Two days later the titer rose to 1:640.

At about this time the patient began to improve slowly. The lesions on the conjunctiva and in the throat disappeared rapidly. On December 19 the temperature, which had previously hovered around 39 C (102.2 F), dropped to 37.5 C (99.5 F). Convalescence was slow, being complicated by a severe labial herpes. On Jan. 18, 1938, agglutinations for *B. tularensis* were positive in dilutions of at least 1:640 and negative for *Bacillus typhosus* and *Brucella melitensis*.

COMMENT

This apparently is a typical case of oculoglandular tularemia, the disease having been contracted by rubbing an irritated eye with hands contaminated by an infected rabbit. One point that deserves comment is the appearance of an ulcerative process in the pharynx during the progress of the disease. Judging from the literature this is either a rare complication or one that has been overlooked. Unfortunately, cultures could not be obtained. Clinically, however, the ulcers closely resembled those seen on the conjunctiva. Contamination of the throat via the lacrimal duct is suggested. There is also a possibility that the involvement of the paranasal sinuses was specific.

ACCIDENTAL VACCINATION OF THE CONJUNCTIVA

F. NECDET SEZER, M.D., MALATYA, TURKEY

Accidental vaccination of the conjunctiva is a rare condition and as a rule is secondary to an infection of the margins of the lids. It occurs when vaccine material is unintentionally brought in contact with the lids of persons vaccinated or of those who are associated with them.

The early literature contains cases reported by Purtscher,¹ Rost,² Eagleton³ and Pihl⁴. Bedell⁵ summarized the literature and collected reports of 93 cases which have been published since Jenner introduced vaccination in 1796. Cases were reported in more recent years by Ball and Toomey,⁶ Lohlein,⁷ Friede⁸ and Folk and Taube.⁹

REPORT OF A CASE

Cemal Ahmed, an 8 year old boy, came to the hospital on July 8, 1937, with a history of swelling of the lids of the right eye. After the first two days the swelling began to discharge and then broke into an "open sore."

The right eye was completely closed, and there were redness and swelling. On the ciliary borders of the lids there were multiple ulcers, which were large and circular and were covered with a white detachable coating. The ulcers were



Fig 1—Patient before treatment

situated among the cilia and extended over onto the adjoining skin (fig 1). The lower palpebral conjunctiva was injected and presented a defined oval ulcer about 3 mm wide, with its long diameter on the horizontal level. The preauricular glands on the right side were enlarged and palpable.

1 Purtscher *Centralbl f prakt Augenh* **19** 83, 1895

2 Rost, R. *Ueber das Vorkommen von Vaccinepusteln auf der Augenhaut*, Dissert., Wurzburg, 1896

3 Eagleton, S. P. *Ophth Rec* **8** 325, 1899

4 Pihl *Klin Monatsbl f Augenh* **38** 454, 1900

5 Bedell, A. J. *Am J Ophth* **3** 103, 1920

6 Ball, J. M., and Toomey, N. *Vaccinia of the Eyelids by Homo-Inoculation*, *J A M A* **79** 935 (Sept 16) 1922

7 Lohlein, W., in Henke, F., and Lubarsch, O. *Handbuch der pathologischen Anatomie und Histologie*, Berlin, Julius Springer, 1928, vol 1, p 136

8 Friede, R. *Klin Monatsbl f Augenh* **85** 427, 1930

9 Folk, M. L., and Taube, E. L. *Am J Ophth* **16** 36, 1933

A smear showed no organism, culture and dark field examination gave negative results

General examination showed one pustule on the right cheek, and it was learned that three brothers of the patient had been vaccinated five days before

The patient's mother was inoculated with serous material removed from the ulcers. After an incubation period of three days, redness and swelling of the skin occurred at the site of inoculation, these increased rapidly, and a typical scab and scar resulted

After treatment with packs moistened with a 30 per cent solution of mild protein silver and placed between the margins of the lids three times a day for an hour and packs moistened with a solution of an acridine derivative,¹⁰ the lesion healed, leaving symblepharon, entropion of the lower lid and defects on the ciliary margin. These sequelae have been treated by surgical methods (fig 2)



Fig 2—Patient after treatment

COMMENT

Taking into consideration all the facts and findings, I am led to conclude that the source of infection in this patient was contamination with the vaccination vesicles of his brothers

¹⁰ The preparation used was 2-ethoxy-6,9-diaminoacridinium hydrochloride

A MODIFIED CORNEAL CLAMP TO FACILITATE THE INSERTION OF STITCHES

T L TERRY, M D, BOSTON

The edges of linear wounds through the cornea, particularly when the laceration is L shaped, do not always aline properly when covered with a conjunctival flap. One edge may be turned inward, a condition more liable to occur if the conjunctival flap exerts uneven pressure

before the anterior chamber is restored (fig 1) To close the wound itself with stitches in the cornea sufficiently close together to insure the desired approximation of the edges would be ideal Similarly, in some instances of postoperative prolapse of the iris after cataract extraction



Fig 1—Delayed healing of a corneal wound due to misalignment of the margins Over half of the right edge of the wound is in contact with the anterior surface of the cornea of the other side Note the downgrowth of epithelium on each lip of the wound

it would be advantageous to stitch the edges of the wound together after removal or replacement of the prolapsed iris

Stitches can be introduced for closure of corneal or corneoscleral wounds by means of the corneal clamp designed by Verhoeff¹ The original Verhoeff clamp in my hands is a rather difficult instrument to use, since it necessitates the pulling of the knurled sleeve, grasped between the thumb and the first finger, upward while bracing the end of the handle against the metacarpophalangeal joint An alternate type of mechanism, illustrated in Verhoeff's paper published in 1934, likewise is somewhat difficult to use

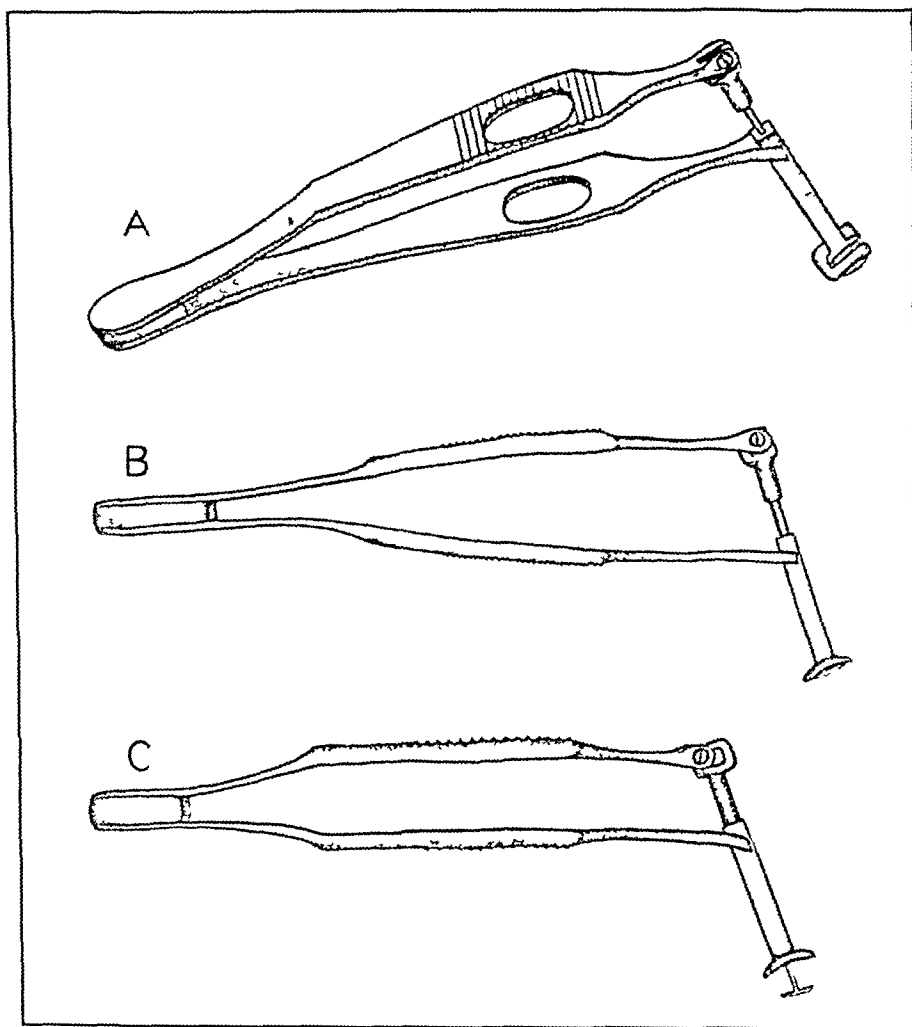


Fig 2—Corneal clamp *A* is an oblique view of the instrument with the jaws closed, *B*, a side view with the jaws closed, and *C*, a side view with the jaws open

1 Verhoeff's instrument was originally developed to facilitate the introduction of corneoscleral stitches in operations for cataract (Verhoeff, F H An Instrument for Simplifying the Insertion of Corneo-Sclero-Conjunctival Sutures in Operations for Cataract, *Am J Ophth* **17** 53 [Jan] 1934) Newer and better methods devised by Verhoeff (A New Instrument for Facilitating the Suturing of the Wound in Operations for Cataract, *Tr Am Ophth Soc* **34** 236, 1936) make the use of the clamp obsolete in operations for cataract

The corneal clamp herein illustrated (fig 2) is opened and closed in the manner of ordinary tissue forceps. The mechanism of this instrument was devised by Mr. A. H. Schmitt.³ He modified the jaws of the clamp by curving the under surface of the upper part. The flat lower portion of the clamp, the foot plate, compresses the cornea against the upper concave surface, holding the two edges of the cornea as firmly as the needle points of the Verhoeff instrument.

If the first suture is inserted near the right end of the wound, it is easy to open the clamp moderately, slip the upper arm of the suture free and slide the clamp to the left to put in additional sutures. This obviates the necessity of completely removing the clamp and reinserting it for each stitch introduced.

Little practice is necessary to gauge the depth of the sutures. At first it was my custom to bring the arms of each suture through a conjunctival flap previously prepared, but experience has shown that it is more advantageous to tie the suture and bring a flap over the corneal stitches. In 2 instances no conjunctival flap was used in repairing a gaping wound near the limbus following postoperative prolapse of the iris.

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Ophthalmologic Review

VERTICAL PRISM VALUES IN COMMONLY USED BIFOCAL LENSES

SIDNEY L. OLSHO, M.D.

PHILADELPHIA

The mere statement of lens powers and the word "bifocals" do not constitute a complete or safe prescription.

Most refractionists are confident that the finished glasses will duplicate the test lenses accepted by the patient during examination. If the patient sees well with the test lenses, he will, they assume, see comfortably and equally well with glasses purchased on the analogous prescription. The type of bifocal lens used is of little importance or is not their specific concern.

A number of special factors which have a bearing on the selection of the most suitable type of bifocal lens are known only to the refractionist. These include a knowledge of the whole of the patient's refractive condition, the visual acuity expected in each eye, the presence of amblyopia in one eye, the muscle balance, the distance for which the reading portion was focused, the seeing field which is to be covered, the exact purpose for which the lenses are prescribed, and the psychologic makeup of the patient. Such clinical data are as a rule not evident to a salesman having before him only an array of numerals. But as this is commonly the case, the refractionist becomes dependent entirely on others, frequently uninformed persons, for interpretation of his prescription. The results cannot help being diversified. But if the refractionist can supplement his clinical information with a wider optical knowledge, then it is he who will be in the most advantageous position, uninfluenced by any commercial motive, to specify the desirable bifocal lens. The optical knowledge he should possess includes a conversance with the different types and makes of bifocal lenses and the optical attributes of each and with the adaptability or lack of adaptability of each type to his patient and the prescription. This is consistent with the idea that a wise physician knows the pharmacy and therapeutic value of the drugs he employs.

Read before the College of Physicians of Philadelphia, Section on Ophthalmology, March 17, 1938

Certain bifocal lenses are advertised directly to the wearer, who is obviously not qualified to select the most suitable type

A meticulous correction of the distance vision, with careful attention to small cylinders and their axes, and the certainty that all of the manifest hyperopia is corrected and that myopia is not overcorrected will effect the following advantages 1 A less high addition is adequate, and in the segment of any bifocal lens the lower the power of the addition the less there is of chromatic and marginal aberration 2 The lower the power of the addition, the less there is of accommodative and adjustive change for the eyes

The distance between the optical centers of the upper, or far seeing portions, of the bifocal lens should be decided by the prescriber One reason may be mentioned here The refractionist knows the results of his examination of muscle balance If he found convergence weak, he will be particularly careful that the centering of the lenses for distance vision is such as not to act adversely For instance, convex lenses set excessively wide apart or minus lenses set with optical centers too close together act as prisms base out, an adverse condition for a person with poor convergence A slight intentional error in the favorable direction will not cause discomfort If the prescriber learns to specify the depth and inset of the segment, his therapeutic powers will be further enhanced He should be able to determine whether his specifications have been precisely carried out

When the finished glasses are inspected, the upper parts of the bifocal lenses may be found to center correctly for ordinary distance seeing If the segments are somewhat closer together or "inset," the average prescriber may assume that the optical centers for reading will be found near the geometric centers of the segments, he is surprised when he is informed that this is rarely the case

Such an ideal might be achieved in the old fashioned two part Perfection bifocal lens, the reading portion of which was a horseshoe-shaped, separately inserted lens unit having its independent optical center But all modern bifocal lenses are composite lenses The segments are not independent units The upper, or main, lenses serve as carriers of the segments This is true for both the fused and the one piece Ultex type The optical center of the main lens is reserved for distance seeing An infracentral, therefore an excentric, area of the main lens becomes an integral part of the composite reading portion It introduces a prismatic effect in the reading part of the bifocal lens

This opens up a problem requiring a general discussion of prismatic effects in the two components of a bifocal lens, those in the distance lens, or carrier component, and those in the segment

PRISMATIC EFFECTS IN THE DISTANCE COMPONENT

For the sake of clarity, I shall first devote myself exclusively to those prismatic effects which are found in the distance component, reserving discussion of those in the segment for later

The eye is confronted by a prism not only when a prism is placed in front of it but when it must look through a lens at some excentric point. The amount of the prism at any excentric point on the lens may be ascertained by multiplying the distance from the center, expressed in centimeters, by the refractive power. The product equals the prism diopters at that point. Examples of this computation follow

- (1) 10 D lens 5 mm from center (10×0.5) = 0.50 prism diopter
- (2) 10 D lens 10 mm from center (10×1.0) = 1.00 prism diopter
- (3) 2.5 D lens 3 mm from center (2.5×0.3) = 0.75 prism diopter
- (4) 2.5 D lens 6 mm from center (2.5×0.6) = 1.50 prism diopters
- (5) 5.0 D lens 2 mm from center (5.0×0.2) = 1.00 prism diopter
- (6) 5.0 D lens 3 mm from center (5.0×0.3) = 1.50 prism diopters

In reading with bifocal lenses the lines of sight pass unavoidably through infracentric areas of the distance lens component

When the eye is directed several millimeters below the optical center of a lens of convex power to an infracentric point, which I shall call the reading point, it encounters a prism base up. When the eye is directed several millimeters below the optical center of a lens of concave power to this reading point, it encounters a prism base down.

The skilful prescriber of bifocal lenses must give consideration to these base up and base down prism effects at the reading points. Of course lateral prism effects and the proper inset of reading and segment centers are important, but in the remainder of this part of my presentation I shall restrict myself to a consideration of the vertical, or base up and base down, prism effects which are introduced by the main, or carrier, lens. These vertical prisms resident in the reading zones are inescapable but have heretofore received little or no consideration by the prescriber of bifocal lenses. He should at least be cognizant of their presence.

In some instances, as will later appear, the vertical prism effects should be counteracted, either wholly or in part and either in the segment or otherwise. In some, but by no means in the majority of, lenses they may be disregarded. When in the two lenses of a pair the vertical prism effects at the reading points are of unequal values, there results a vertical prism imbalance. The uncorrected vertical prism imbalance may be sufficient to make the bifocal lenses unwearable. If hyperphoria is present, such a vertical prism imbalance establishes in the reading zone a dominance either of the appropriate or of an adverse prism effect. This must be foreseen by the refractionist. Whenever bifocal lenses are

prescribed it will be found highly advantageous to consider the precise vertical prism values at the two infiacentric reading points. These values are easily determined by application of the Prentice principle after the vertical effective power of the lenses is known and the depth from the optical centers of the reading points is established.

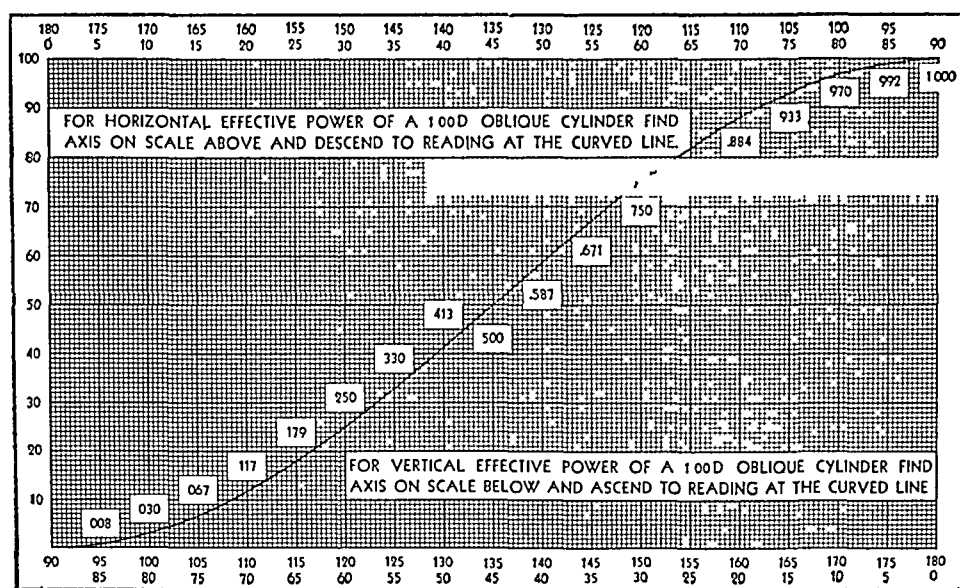
The term vertical effective power will be explained and the depth of the reading point discussed.

Spherical lenses are 100 per cent vertically effective.

Cylinder lenses at axis 180 are 100 per cent vertically effective.

Cylinder lenses at axis 90 are not at all vertically effective.

Cylinder lenses at axis 45 and 135 are vertically effective 50 per cent.



Graph used to estimate the net vertical effective power of any oblique cylinder (Supplied by Bausch & Lomb Optical Company)

Cylinder lenses at other oblique axes are less easily resolved.

To estimate accurately the percentage of vertical power effective for cylinders at oblique axes other than 45 and 135 degrees involves the use of rather complicated trigonometric calculations. It is easier to use a graph which is based on accurate computations. On this graph is read the percentage of power effective in the central vertical meridian.

To obtain the net vertical effective power of any oblique cylinder, the dioptric power is multiplied by the percentage for its particular axis, as found in the accompanying chart.

For spherocylinder lenses, to all of the spherical power is added the net vertical effective power of the cylinder. The algebraic sum equals the total vertical effective power.

Examples illustrating the estimation of the net total vertical effective power (V E P) of lenses with the aid of a graph follow

(1) +3 00 cyl, ax 120	$+3.00 \times 0.25 = +0.75$	V E P
(2) +2 00 cyl, ax 150	$+2.00 \times 0.75 = +1.50$	V E P
(3) -1 50 cyl, ax 125	$-1.50 \times 0.33 = -0.50$	V E P
(4) +3 00 sph +2 50 cyl, ax 75	$+3.00 \times 1.00 = +3.00$	
	$+2.50 \times 0.067 = +0.167$	
	<hr/>	
	+3 17	V E P
(5) -2 75 sph +1 75 cyl, ax 30	$-2.75 \times 1.00 = -2.75$	
	$+1.75 \times 0.75 = +1.31$	
	<hr/>	
	-1 44	V E P

To find the prism values at the reading points, I shall presently apply the Prentice principle to the lens combinations just given. Before this is done, some definite idea of the depth from the optical center of this reading point is necessary. It is too laborious to calculate the prism effects at all the possible depths below the centers. For simplicity's sake, I shall assume that the centers of the distance lenses will be set not lower than the levels of the external canthi. Only then will distance vision be most comfortable through moderately strong lenses. I shall arbitrarily assume that the upper edge of a contemplated segment will be 4 mm below the optical center of the distance lens, a good average. I shall then assume that the reading level will be down an additional 4 mm, arriving at a point which is a total of 8 mm below the optical centers of the distance lenses. This depth is conservative, but greater and lesser figures have been used by those interested in promoting some particular bifocal lens. As there can be but one point at a time for calculation, a depth that the eyes will use naturally and most frequently must be selected, namely, one down 8 mm.

The net vertical effective power of the distance lens multiplied by the depth of the reading point (arbitrarily 0.8 cm) equals the prism diopters present at the reading point. The prism is base up if the net vertical effective power is plus and base down if the net vertical effective power is minus.

Examples illustrating the estimation of the vertical prism resident at the reading point for the lens combinations just given follows

(1) V E P	$+0.75 \times 0.8 = \triangle 0.6$	B U at reading point
(2) V E P	$+1.50 \times 0.8 = \triangle 1.2$	B U at reading point
(3) V E P	$-0.50 \times 0.8 = \triangle 0.4$	B D at reading point
(4) V E P	$+3.17 \times 0.8 = \triangle 2.5$	B U at reading point
(5) V E P	$-1.44 \times 0.8 = \triangle 1.2$	B D at reading point

Summary—The main, or carrier lens is a component of the reading part of a modern bifocal lens.

The main lens is centered for distance seeing.

Near vision is obtained through a zone considerably below the optical center of the main lens

The main lens introduces a vertical prism to the composite reading part of the bifocal lens

The prism introduced to the reading part is base up or base down, accordingly as the net vertical effective power of the main lens is plus or minus

The net vertical effective power of the main lens is multiplied by the depth in centimeters of the reading point. The product is the vertical prism introduced by the main lens to the composite bifocal lens

VERTICAL PRISM EFFECTS IN THE SEGMENT COMPONENT

In the preceding part of this article the vertical prism effects of the carrier lens component of a bifocal lens were considered without regard for the segment component

For the sake of clarity, I shall now devote myself exclusively to the segment component

One can temporarily nullify the influence of the distance, or carrier lens by assuming that it is of zero power—plano. This excludes it yet enables one to concentrate on the vertical prism values of segments fulfilling their destiny in their natural positions

Bifocal additions are convex and spherical. The intended addition is *always* generated in the form of a circle. In grinding standard additions the optical centers are established in the center of these, the circles of origin

These circles are at the present time of a comparatively few and soon familiar diameters

When the addition becomes a bifocal component, only a part or segment of the circle is used, which accounts for its usually noncircular shape

The optical effect of the segment can be understood only by visualizing the complete circle of its origin. This makes it easy

When the segment is in situ, the accepted theoretic reading point is 4 mm (0.4 cm) below its upper margin. The optical center of the segment is farther down (the length of the radius less the 0.4 cm). At the reading point, a prism base down is introduced by the segment equivalent to $D(R - 0.4 \text{ cm})$, in which D equals the power of the addition, R , the radius of its circle of origin, and 0.4 cm, the depth of the reading point from its upper margin

I shall consider a distance lens of zero power and a supposed circular reading addition 8 mm in diameter. In this case there is no vertical prism 4 mm below the upper margin of the addition, for under these conditions the reading point is directly at the optical center. But a bifocal addition of this diameter would allow the eye too little excursion. It would have too little vista

I shall now employ a circular addition 16 mm in diameter (radius, 0.8 cm), as is used in the almost invisible fused nokrome Orthogon C and Tillyer C bifocal lenses. Additions even of this size are considered small and therefore have not found wide popularity. The reading point (0.4 cm from the upper margin of the addition) is no longer at the optical center but is 0.4 cm above the center.

Continuing with a distance lens of zero power, I shall employ a circular addition 20 mm in diameter (radius, 1 cm), as used in the ordinary fused or Kryptok lens (average 20 mm), the fused Widesite D lens (20 mm) and the nokrome fused Orthogon D (20 mm) and nokrome Tillyer D (20 mm) lenses. In the finished bifocal lens more or less of the circle has been cut away below, leaving the familiar upright arch, horseshoe-shaped barely visible segment. But as the whole circle was 20 mm in diameter (radius, 1 cm), it follows that the reading point 0.4 cm below the upper margin is 0.6 cm above the center of the segment ($1 - 0.4 = 0.6$ cm).

Size of Circle of Origin	Bifocal Lens	Distance of Reading Point from Center of Circle (R - 0.4 cm)	Calculation	Prism in Segment at Reading Point
16 mm	Fused { Orthogon C Tillyer C }	$0.8 - 0.4 = 0.4$ cm	$0.4 \times -2.50 = \Delta 1.00$ B D	
20 mm	Fused { Kryptok, Orthogon D Tillyer D Widesite D }	$1.0 - 0.4 = 0.6$ cm	$0.6 \times -2.50 = \Delta 1.50$ B D	
38 mm	One piece { Ultex A Ultex B Ultex E }	$1.9 - 0.4 = 1.5$ cm	$1.5 \times -2.50 = \Delta 3.75$ B D	
22 mm		$1.1 - 0.4 = 0.7$ cm	$0.7 \times -2.50 = \Delta 1.75$ B D	
32 mm		$1.6 - 0.4 = 1.2$ cm	$1.2 \times -2.50 = \Delta 3.00$ B D	

The one piece or Ultex, additions, like all other types are generated from circles. In these, the original circles for A style segments are 38 mm in diameter (radius, 1.9 cm), for B style segments, 22 mm in diameter (radius, 1.1 cm), and for E style segments, 32 mm in diameter (radius, 1.6 cm).

It follows that the reading point in A style is $1.9 - 0.4 = 1.5$ cm above the center of the segment, in B style, $1.1 - 0.4 = 0.7$ cm above the center of the segment, and in E style, $1.6 - 0.4 = 1.2$ cm above the center of the segment.

The Prentice principle is applied as follows. From the radius of the segment, as expressed in centimeters, 0.4 is subtracted and the difference multiplied by the segment power, as D (R - 0.4). The product equals the vertical prism base down effect of the segment at the reading point.

For example, consider a +2.50 addition on a distance lens of zero power with the reading point 0.4 cm below the upper margin of the segment. The calculation and the prism in various segments are shown in the accompanying table.

In this table it is observed that the larger the segment the greater is the prism base down contribution of the segment at the reading point

This opens up the too frequently neglected opportunity of selecting that standard segment which has the amount of prism base down which most nearly counterbalances at the reading point the prism base up of the plus distance component. A perfect counterbalance would mean a true optical center for reading.

A few prescriptions, the same addition being used in all instances, demonstrate that the careful choice of a segment may sometimes be ideal.

- (1) R +0.75 +0.50 cyl, ax 180
 L +0.75 +0.50 cyl, ax 180 vertical effective power = +1.25
 Prism base up at reading point = $+1.25 \times 0.8 = \Delta 1.0$ B U
 With an Orthogon O or Tillyer C segment there will be a perfect optical center at the reading point for the prism of the +2.50 addition at the reading point (table) is $\Delta 1.0$ B D
- (2) R +1.00 +1.75 cyl, ax 45
 L +1.00 +1.75 cyl, ax 135 vertical effective power = +1.87
 Prism base up at reading point = $+1.87 \times 0.8 = \Delta 1.50$ B U
 Prism at reading point with bifocal segments of the 20 mm group in the table and the +2.50 addition = $\Delta 1.50$ B D
- (3) R +2.75 +2.00 cyl, ax 180
 L +2.75 +2.00 cyl, ax 180 vertical effective power = +4.75
 Prism base up at reading point = $+4.75 \times 0.8 = \Delta 3.80$ B U
 Prism at reading point with the Ulte\ A segment = $\Delta 3.75$ B D
- (4) R +2.25
 L +2.25 vertical effective power = +2.25
 Prism base up at reading point = $+2.25 \times 0.8 = \Delta 1.80$ B U
 Prism at reading point with the Ulte\ B segment = $\Delta 1.75$ B D
- (5) R +3.00 +1.50 cyl, ax 135
 L +3.00 +1.50 cyl, ax 45 vertical effective power = +3.75
 Prism base up at reading point = $+3.75 \times 0.8 = \Delta 3.0$ B U
 Prism at reading point with the Ulte\ E segment = $\Delta 3.0$ B D

The desirability of the upright arch segments for plus distance corrections is apparent. When the plus distance correction is strong, its excessive contribution of base up prism at the reading point can be at least partially counteracted, but only by the largest available standard upright arch segment. Prisms base up at the reading point move the images down and uncomfortably toward the reader's body. They fatigue the depressor muscles of the eyes.

If the signs in the foregoing five formulas are changed to minus, the distance corrections introduce the given prisms at the reading points, all base down. Were the same upright arch segments employed, in the same order, the prism base down effects at the reading points would be doubled. Prisms base down at the reading points are tolerated better than prisms base up. Prisms base down elevate the reading matter

There are no standard segments with prism base up effect which will reduce the heavy prism base down effect at the reading point of strong minus distance corrections. But there is a group of segments which, as regularly supplied, neither add to nor subtract from any vertical prism which may already be present at the reading point. These segments appear in the form of a reversed arch with a flat or modified horizontal top margin.

In this group of modern fused bifocal lenses with similar optical characteristics are included Panoptik, Fulvue, Widesite A and Univis D and B lenses. The upper margins of these horizontal top segments have been modified with the purpose of diminishing their visibility and eye-ward reflections. These segments provide a good field considering their comparatively small size, but only when they are skilfully positioned. As for all other bifocal lenses, the addition is generated in the form of a circle. From this circle, an arc above the equator is cut off and discarded. The segment is shaped up from the remainder, so that the equator, and therefore the segment's optical center, falls from 3.5 to 4.5 mm below the flat upper margin, namely at the theoretic reading point. (In the larger flat top Univis R type the optical center is 6.5 mm below the upper margin. It is likely to exert a slight prism base down effect.)

As these reversed arch segments neither add to nor subtract from vertical prism already present at the reading point, they are particularly suitable when the upper lens is a high minus. The reversed arch segment does not increase the inescapable prism base down effect at the reading point.

If, on the other hand, the distance correction is a high plus, there is at the reading point a heavy prism base up effect. It may be desirable to let the segment counteract or partially neutralize it. In these instances, therefore, one does not use the reversed arch but preferably an upright arch segment, namely, one which has a counteracting prism base down effect and plenty of it.

The thicker upper margins of the reversed arch segments are more visible than those in the upright arch. Hence optical considerations may sometimes have to be sacrificed to appearance. The upright arch segments with small circles of origin are less objectionable on minus corrections than those originating from large circles. The Orthogon or Tillyer C invisible fused bifocal lens can be used, the entire 16 mm circle being employed.

As the minus distance power increases, upright arch segments become progressively less suitable. The larger the upright arch segment, the less it is suitable for high minus corrections.

The vertical effective power of a distance correction may be nil, as in a plane cylinder, axis 90 degrees. If an upright arch segment

is used, the optical center is well below its upper margin, it is 10 mm below with the average 20 mm segment and 19 mm below with an Ultex A segment. But if a reversed arch segment is used, the optical center for reading will be only about 4 mm from the upper margin of the segment, which is almost ideal.

Upright arch segments will, however, continue to appear on these low distance powers and on plane cylinders, axis 90, because they are less visible. The refractionist should know that the optical center may then be even beyond the lower margin of the lens.

It is evident that it is an almost unique experience to find true optical centers at or close to the two reading points of any pair of present day bifocal lenses. They are so located only when at the two reading points the infiacentric prisms of the distance lenses are just counterbalanced by the prism contribution of the segments.

In the majority of prescriptions the elimination at the reading points of vertical prism effects is impossible with regular segments. Images are therefore nearly always displaced. Visual requirements are best served when there is a minimum of vertical prisms at the reading points. This is, therefore, an important consideration in the discriminating selection of the bifocal lens. Specification of the segment type by the informed refractionist may insure his patient's securing the type most advantageous.

Correspondence

OCULAR DOMINANCE

To the Editor —The significance of ocular dominance, interest in which was renewed by the appearance of the paper of Walter H. Fink in the April issue of the ARCHIVES, page 555, is more far reaching than the apparent lack of ophthalmologic interest in it would justify

In neglecting the great leads which the earlier studies of ocular dominance provided, ophthalmologists missed a fertile field of usefulness, especially in education, which now has been almost preempted by neurologists, psychologists and educators

Earlier studies established the fact of ocular dominance and demonstrated that approximately 25 per cent of all persons are left handed, although left handedness is obvious in but 4 per cent

I noted the confusion in speech, the difficulty in acquiring reading skills, the reversals in reading and writing and the changes in behavior which were found chiefly among the 21 per cent of children and adults who had been born left handed but trained to right handedness. I suggested (*Unilateral Sighting, California West Med* 28:189 [Feb] 1928) the retraining of this group to left handedness

This work is being followed up in the field of education. A great literature has already arisen attesting to the fact that the reeducation of children having confused dominance to their native unilateral dominance can and will produce coordination in speech, reading, writing and general motor control, and will overcome faults of behavior which have sprung from educational frustrations. The determination of eyedness was the first step which finally led to this great and growing scholastic achievement, and the determination of eyedness and that of handedness still remain as the cornerstones of this great line of mental and physical therapy

LLOYD MILLS, M D, Los Angeles

SURGICAL TREATMENT OF STRABISMUS

To the Editor —I have read the article by Dr. Maynard C. Wheeler in the December 1937 issue of the ARCHIVES (page 1000) regarding the surgical treatment of strabismus by Dr. Barraquer, formerly of Barcelona, Spain, by the myocompter

I have also read the article by Walter I. Bristow, of Columbia, S. C., in the May issue (page 797). I wish to offer a word to substantiate Dr. Bristow's stand. On my last visit to Barcelona in 1931 I had a set of the Briggs instruments with me. I demonstrated the operation to Dr. Barraquer, and he was enthusiastic about it, particularly for children. On my return home I sent him a set, and a short time afterward he sent me his modified instrument.

Dr. Barraquer made no claim to originality. However, in his last book he cited a number of cases in which he used the operation suc-

cessfully In a communication about a year afterward he told me he was still using it with success

I have used the Briggs instrument for fifteen or eighteen years with much success, particularly for children in whom the external rectus muscles were small and for persons who were to begin orthoptic exercises the same day as the operation

I had the pleasure of advising Dr Briggs of my success with his technic on several occasions before his death I regard Dr Briggs's method as a real contribution to the field of ocular surgery and feel that he is entitled to full credit for this work

OTIS WOLFE, M D , Marshalltown, Iowa

Abstracts from Current Literature

EDITED BY DR WILLIAM ZENTMAYER

Biochemistry

INCINERATION OF SECTIONS OF NORMAL HUMAN LENSES B LUDERITZ,
Klin Monatsbl f Augenh 99:75 (July) 1937

Luderitz reports on his method of preparing lenses obtained within the capsule from corpses and their reduction to ashes after sectioning them with the microtome into sections of 30 micromillimeters. Sections of brittle lenses of aged persons broke frequently, whereas those of young persons tore on account of their softness. The changes of the structure of the lenses during the process are described, and results of the histochemical analysis are presented. Calcium was found in every lens, phosphates were absent in the calcium ashes and present in most instances in the total ashes, especially in the lenses of young persons. Complete transformation into ashes occurred at a temperature of 600 C. Pathologic lenses were not considered with the exception of the lens of a woman aged 83 with numerous spheruliths.

Luderitz found the inorganic substances equally distributed in the lenses of juvenile subjects. The ashes of the cortex grow denser with advancing age. The density is not due to an accumulation of calcium, yet the nucleus consists chiefly of calcium. Phosphates were present predominantly in the fibers of the lenses of young persons. Some of the inorganic substances of the lenses were coupled to organic substances.

K L STOLL

Color Sense

CAN DEFICIENCY OF COLOR PERCEPTION BE CURED BY NEOPHAN GLASSES? H O LINDEMAN Klin Monatsbl f Augenh 99:224 (Aug) 1937

Neophan glasses were recommended in certain cases of defective color sense by Birch-Hirschfeld in 1932 and by Schubert in 1935. Velhagen Jr, after examining 71 patients with the charts of Stilling and of Ishihara without neophan glasses, found that only a relatively small percentage of patients with defective color sense showed an improvement after correction with the glasses. Lindeman examined 1,116 students, 1,022 boys and 94 girls, with these two kinds of charts. None of the girls and 83 of the boys were found to have defective color sense. The 83 boys were subjected to five different tests without and with neophan glasses. None of the 83 were able to pass the five tests without essential errors after insertion of neophan glasses of 50 or 70 per cent absorption. Incidentally, Lindeman found out that 964 of these 1,022 boys had previously taken color tests at recruiting stations and that 26 of the 83 with defective color sense had escaped detection by means of various tricks.

K L STOLL

Conjunctiva

STAPHYLOCOCCIC CONJUNCTIVITIS J H ALLEN, *Am J Ophth* 20: 1025 (Oct) 1937

Allen reaches the following conclusions after the experimental reproduction of conjunctivitis with staphylococcus toxin

"1 Staphylococci of the hemolytic aureus type isolated from ocular sources are capable of producing toxin potent in lethal, hemolytic, and dermonecrotic factors

"2 Toxin of this type is capable of producing a conjunctivitis and a keratoconjunctivitis after instillation into the conjunctival sac of rabbits, monkeys, baboons, and man This superficial punctate keratitis is similar to that so frequently observed in clinical cases of staphylococcal conjunctivitis

"3 The action of the toxin probably accounts for the production of conjunctivitis by staphylococci, even though they are not epithelial parasites in the sense of Lindner and Howard "

W S REES

DOMINANT INHERITANCE OF PTERYGIUM J STREBEL, *Klin Monatsbl f Augenh* 99: 35 (July) 1937

Extrinsic influences, such as tropical climate and ocean air, were considered more potent in the manifestation of pterygium than heredity Strebel publishes a genealogic study of a family in which pterygium occurred in three generations, either unilaterally or bilaterally There was no evidence of this formation in the family of the patient's father However, his mother and his daughter had bilateral pterygium, and his son had unilateral pterygium The condition was absent in the third child, a boy Pterygium was not seen in 1 of the patient's sisters or her children, while another sister presented the condition bilaterally The pterygium of Strebel's patient would have been considered an acquired occupational disease had the genealogic tree not proved the hereditary basis

K L STOLL

Cornea and Sclera

MARGINAL DEGENERATION OF THE CORNEA J FRANCOIS, *Arch d'ophth* 53: 432 (June), 540 (July), 616 (Aug) 1936

This condition is considered by Terrien to be the rarest of all the diseases of the cornea Up to the present only about 130 cases have been reported These are all listed by the author, who adds detailed descriptions of 2 cases of his own, including photographs The whole subject is exhaustively reviewed under the chapter headings of terminology, symptomatology (objective, accessory and functional), evolution, complications, prognosis, pathologic anatomy, pathogenesis and treatment In its first stages this condition is characterized by peripheral vascularization and opacity similar to gerontoxon The second stage consists of a circumscribed or diffuse thinning of the cornea The third stage is that of ectasia of the marginal defect, and the final stage is that of ectasia of the whole cornea The biomicroscopic findings are presented The principal complication is that of rupture of the cornea,

spontaneous or traumatic. Secondary infection of the affected zone is not common. Histologic examination has been carried out in 12 cases. Photomicrographs and drawings are reproduced. The theories attempting to explain the origin of this condition are discussed. The author believes it to be a true degeneration. Curative treatment is largely surgical. Iridosclerectomy, cauterization and excision have all been advocated. The author believes that excision gives the best results, as it reduces the astigmatism, increases the resistance of the thinned zone, and favors improved nutrition. A complete bibliography is given.

S B MARLOW

PARTIAL FUNNEL DETACHMENT OF DESCMET'S MEMBRANE A
BUSACCA, Arch d'opht 53: 694 (Sept) 1936

This condition was observed in a case of leukoma of the cornea due to a purulent conjunctivitis of the eyes at birth. A drawing of the condition as pictured at slit lamp examination is presented. The mechanism of its production is suggested as being due to the firmness of adhesions between the posterior surface of the cornea and the lens, which resulted in tearing off of Descemet's membrane when the anterior chamber was reformed.

S B MARLOW

INFORMATION BASED ON THREE HUNDRED AND FORTY-NINE CASES OF
HEREDOSYPHILITIC KERATITIS D LAZARESCU, Arch d'opht 53:
756 (Oct) 1936

This report is a statistical study of 349 cases of interstitial keratitis. According to the author this condition occurs in more than 4 per cent of patients with ocular disease. In 76.8 per cent of the cases of heredosyphilitic keratitis the patients were between 5 and 20 years of age. The condition was unilateral in 32.4 per cent. Four per cent of the patients had acquired syphilis. A history of abortion was given by 25.6 per cent of the mothers. Stigmas of inherited syphilis were present in 77 per cent of the entire group. The Wassermann test was positive in 83.3 per cent. At the time of the patients' admission to the hospital the disease was unilateral in 101 and bilateral in 248. In 57 cases the appearance of the second eye was not noted. In 80 cases both eyes were affected simultaneously. The average time between the attacks in the two eyes was one hundred and nine days. The conclusions drawn are as follows: 1. The most frequent signs of congenital syphilis are bone lesions (38 per cent) and Hutchinson's teeth (36 per cent). 2. Antisyphilitic treatment often prevents or delays involvement of the second eye (87 per cent). 3. Trauma is capable of causing an inflammation limited to one eye. Visual results were not determined in this series of cases.

S B MARLOW

PERFORATING SCLEROMALACIA F J SORIANO and A RIVA, Arch
de oftal de Buenos Aires 12: 139 (March) 1937

A case of perforating scleromalacia is reported in which perforating holes were situated at the limbus. This case, although in some aspects

atypical, according to the authors fits in with the process first described by van der Hoeve. With the exception of one hole, the others were small and irregular. The lesion in this case and in reported cases was degenerative and unaccompanied by inflammatory symptoms. In this case there coexisted a chronic rheumatoid arthritis, which was also found in some of the reported cases. The authors agree with van der Hoeve in considering the nature of the lesion degenerative (trophic).

C E FINLAY

TRAUMATIC PARENCHYMATOUS KERATITIS IN CONGENITAL SYPHILIS
G VON GROLMAN, Arch de oftal de Buenos Aires 12 449 (July)
1937

A case of severe parenchymatous keratitis developing after a contusion is reported. After several weeks the cornea began to clear and finally cleared entirely. Shortly afterward, the other eye became affected with typical parenchymatous keratitis, which ran the usual course. There was a 4 plus Wassermann reaction.

Von Grolman discusses at considerable length the different opinions on the relation of trauma to syphilitic lesions in general and of the eye in particular. He also discusses the theory that parenchymatous keratitis is not an essentially syphilitic lesion, the syphilis being a predisposing cause (Kummel). The author is inclined to accept this view.

C E FINLAY

Experimental Pathology

OCULAR THERMOMETRY A BUCALOSSI, Ann di ottal e clin ocul 65.
507 (July) 1937

A fine needle embodying a thermopile of steel and constantan was used to record temperatures in the anterior chamber, the lens and the peripheral and central portions of the vitreous of rabbits exposed for half an hour to various temperatures in the incubator or refrigerator. Temperatures of 6, 18, 30 and 42 C were employed, and the intraocular temperature was compared with the rectal temperature each time. In each experiment one eye was left open, while the other was closed by lid sutures. Exposure to a temperature of 6 C produced a slight lowering of the rectal temperature (from 38.6 to 38.3 C) and a marked reduction in the intraocular temperature. The reduction in the intraocular temperature was especially marked in the anterior segment of the open eye, the temperature reaching 30.85 C in the anterior chamber and 34.75 C in the lens. In the central portion of the vitreous it reached 36.37 C, while in the peripheral portion there was a slight reduction (to 37.6 C). The closed eye of the same animal showed similar but much slighter changes (36.65 C in the anterior chamber). Exposure to a temperature of 18 C produced changes that were similar but much slighter than those produced at 6 C. At a temperature of 30 C, a slight reduction of temperature in the anterior chamber was observed in both the closed and the open eye. Exposure to 42 C produced a slight rise in the rectal temperature and in the temperature of all portions of the eye, but the temperature in the eye was always less than the rectal temper-

ature, the greatest difference being observed in the temperature of the anterior chamber. In the last experiment there was little difference between the temperature in the closed eye and that in the open eye. In general, the experiments showed that the intraocular temperature is always lower than the rectal temperature in their relation to the surrounding temperature. The intraocular temperature diminishes progressively from the deep to the superficial constituents of the eye. The lowering of the intraocular and rectal temperatures is related to the surrounding temperature. The temperature of the peripheral part of the vitreous was not influenced by opening or closing the lids, being dependent on the temperature of the body as a whole.

S R GIFFORD

General

PHOTOPHOBIA A MAGITOT, *Ann d'ocul* 174:817 (Dec) 1937

In a communication in 1936, as well as in an article which recently appeared in the *Annales d'oculistique*, Magitot drew attention to the fact that therapeutics applicable to ocular pain were capable of relieving tearing and photophobia. Photophobia, as it is known in the ophthalmologist's daily practice, is a painful sensation brought on by light. Many persons have a deep sense of pain when exposed to bright light.

The different kinds of photophobia are described, and different conceptions as well as ideas concerning the problem are discussed.

It is difficult, says Magitot, to follow Lebensohn and accept all his arguments. The experience of Bruce and Rochat obliges one to accept the theory of a vegetative reflex brought on in an antidromic manner by the excitation of light acting on the nerve terminals compressed and hyperanesthetized by an inflammatory phenomenon. But the vasodilatory action, which seems to indicate the action of epinephrine hydrochloride in subconjunctival injections, does not accord with the vasoconstrictive effect of light (Sven Larsson) or with the clinical facts. Magitot has observed the disappearance of photophobia under the influence of anesthesia of the sphenopalatine ganglion, notwithstanding marked congestion of the vessels of the iris. On the other hand, it must be remembered that photophobia may appear also in the course of certain meningeal reactions. For these reasons it seems that photophobia should be considered not only as a vasomotor disturbance but as a sympathetic allergy, brought on by sensitive excitations, direct or recurrent.

Anesthesia of Meckel's ganglion through the palatine canal is brought on without producing the slightest anesthesia in the ophthalmic and suborbital fields, which seems to indicate that the light excitations are brought on by long or short reflexes, tearing, pain and blepharospasm and often by accommodative asthenopia.

If Magitot's remarks are well founded, and it is admitted that light is able to provoke pain of the sympathetic type, due to a hyperallergy of the trigeminal nerve, the practitioner will readily remember that painful photophobia may be helped by three methods, any one of which is easily applied. These are the subconjunctival injection of drops of epinephrine hydrochloride, infiltration of the sphenopalatine ganglion with procaine hydrochloride and alcoholization of the orbit.

S H MCKEE

Instruments

SPATULA-FORCEPS FOR HEINE'S OPERATION P F ARCHANGHEL'SKY,
Arch d'opht 53. 696 (Sept) 1936

A spatula-forceps copied from de Wecker's iridectomy scissors is described. It is inserted closed through the usual scleral incision into the anterior chamber. It is then opened and withdrawn. The author has successfully used it on 5 eyes of 4 persons. He believes that wider separation of the iris, ciliary body and choroid from the sclera can be obtained by its use.

S B MARLOW

PRESENTATION OF A SMALL MODEL ENTOPHTHOSCOPE E P FORTIN,
Arch de oftal de Buenos Aires 12.456 (July) 1937

Fortin describes a small model entophtoscope which, although giving a less clear image, owing to its size and less costly makeup is of greater utility clinically than a larger instrument. Its size is that of an ordinary microscope, and it has six sets of oculars. A small Philipps mercury lamp is used instead of the Cooper-Hewitt lamp, as in the first instrument. The Nicol prism is replaced by a Zeiss polarizer, and the filter, by a Zeiss uviole glass. Fortin gives the indications for each of the six sets of oculars in detail.

C E FINLAY

Lacrimal Apparatus

RECURRENT PNEUMOCOCCIC DACRYOADENITIS G E DE SANCTIS,
Ann di ottal e clin ocul 65. 551 (July) 1937

The previous literature on recurrent pneumococcic dacryoadenitis is reviewed, and apparently only 3 cases have been reported. The author's patient, a woman of 41, gave a history of swelling in the region of the right lacrimal gland every spring for the past nine years. Each time treatment with hot packs was followed by an escape of pus from the upper cul-de-sac, with relief of all symptoms. The region of the left gland had been involved in a similar process on two occasions. The patient had suffered from an attack of pleurisy and bronchopneumonia sixteen years before the first attack of adenitis. When she was seen by the author, the typical picture of acute dacryoadenitis was present. Pneumococci were recovered from pus expressed by pressure on the gland and also from pus removed by mouth later, when all symptoms had subsided. Smears and cultures from the upper cul-de-sac were negative. It seems possible that pneumococci remained in the gland in a latent form between the numerous attacks.

S R GIFFORD

Lens

LUXATION OF LENS THROUGH A RETINAL TEAR INTO THE SUBRETINAL SPACE F B FRALICK, Am J Ophth 20 795 (Aug) 1937

Fralick reports an apparently unique case in which retinal detachment occurred in the only eye of a 36 year old man with congenital subluxation of the lens, the fellow eye, in which there had also been congenital subluxation of the lens, having been lost through operation.

and subsequent injury There were two tears in the retina, and through the lower tear the periphery of the luxated lens could be seen The patient was placed in a Bradford frame, face down, and the next day the lens appeared in the anterior chamber, whence it was removed An unsuccessful diathermy operation was done a month later, and the vision remained limited to moving objects

W S REESE

CATARACT DUE TO ELECTRIC SHOCK C DJACOS, Arch d'ophth 53: 454 (June) 1936

The interest in the case reported by Djacos lies in the observation that the cataract did not progress to complete opacity The author was able to aid in its regression, with improvement of vision This case has an important medicolegal aspect and supports the contention of Genet that it is unnecessary to hurry operation Cataract due to electric shock is not a distinctive type, biomicroscopic examination does not always give the same picture Although for the most part the cataract described here closely resembles that described by Rollet and Pautique, it is not always possible by biomicroscopic examination to recognize such a cataract The only common features are that it is subcapsular and that as it progresses it spreads from the superficial to the deeper layers of the lens

S B MARLOW

METABOLISM OF THE LENS AFTER PARATHYROIDECTOMY R CAMPOS, Ann di ottal e clin ocul 65:481 (July) 1937

With the technic of Warburg, Campos estimated the oxygen consumption and anaerobic glycolysis of the cortex removed from the capsule of the crystalline lens The oxygen consumption of the normal rabbit's cortex was $Q_{O_2} = 0.49$, and the glycolysis was $O \frac{n_2}{M} = 0.51$ The eyes of 9 of 24 rabbits subjected to parathyroidectomy were suitable for examination, showing partial opacities of the lens of various degree The eyes of 2 other rabbits showing complete cataract were also employed There was a slight decrease in the oxygen consumption of the cortices of animals killed about forty days after parathyroidectomy, but those of the rabbits with incomplete cataract as a whole showed no definite decrease The lenses with complete cataract, on the other hand, showed a marked decrease in oxygen consumption, which was 0 in 1 animal and 0.10 in the other Anaerobic glycolysis was reduced in all the lenses examined, the reduction being most marked in eyes removed after from fourteen to twenty-one days and again after fifty days The average figure for the lenses with partial cataract was 0.30 Of those with complete cataract, one showed a glycolysis of 0.30 and the other of 0 When the amount of calcium in the testing fluid was increased and that of potassium was decreased, a more marked decrease in glycolysis occurred Such an increase of calcium was observed in the aqueous of the parathyroidectomized animals (the blood calcium was low), and it is thought that such an increase in calcium may be an important factor in the precipitation of the proteins of the lens and in the impairment of glycolysis which was observed

S R GIFFORD

OPERATION FOR SECONDARY CATARACT P KNAPP, Klin Monatsbl f Augenh 99. 15 (July) 1937

Knapp discusses operations for secondary cataract and the after-care on a broad scale and offers valuable technical suggestions, especially for the discussion. He arrives at the following conclusions. The operation for secondary cataract is free from danger in most of the cases, providing the eye is free from irritation and remnants of the cortex. The absence of precipitates on Descemet's membrane and folds in it must be ascertained by means of the slit lamp. If these requirements are fulfilled, an early discission is preferable as long as the posterior capsule is still thin. Peripheral incision with a sharp knife and posteroanterior discission, which guards the vitreous best, are recommended, the aqueous humor must not escape. Glaucoma is the only actual danger, therefore, a solution of pilocarpine should be instilled after operation. Nevertheless, the danger of glaucoma cannot be entirely eliminated any more than it can be totally excluded after intracapsular extraction of cataracts. Predisposing factors for the development of secondary glaucoma, aside from impaction of the iris and synchysis or other changes of the vitreous, are likely to occur, especially lesions of the limiting membrane of the vitreous. Knapp suspects that in these cases the dissolved vitreous entering the anterior chamber causes obstructions in the irido-corneal angle. The operations for secondary cataract should be postponed as long as symptoms of a glaucomatous tendency are present, or else extended postoperative control is indicated.

K L STOLL

PHACOCELE REPORT OF A CASE H SKYDSGAARD, Acta ophth 15. 343, 1937

A large ulcer developed on the lower third of one cornea of a 17 year old girl suffering from gonorrheal conjunctivitis. A descemetocoele formed, which ruptured when the patient struck her eye with her hand. More than half of the lens prolapsed through the corneal perforation, it became constricted into the shape of an hourglass and effectively prevented any prolapse of the iris. Thirty-six hours later the capsule of the lens ruptured, and the lens substance extruded. Remains of the capsule continued to keep the corneal perforation closed, and the subsequent course was entirely favorable. Vision of 5/60 was recovered.

O P PERKINS

Lids

CICATRIZATION WITHOUT ECTROPION FOLLOWING THERAPY WITH CARREL-DAKIN SOLUTION E PUSCARIU, Arch d'opht 53 536 (July) 1936

In the author's experience wounds of the lid can be quickly sterilized and rapid epithelization promoted by the Carrel-Dakin method. In this way dense thick scar tissue is avoided, and the danger of ectropion is lessened. Two cases are cited, and photographs of the results obtained accompany the article.

S B MARLOW

BLEPHAROCHALASIS P PANNETON, *Arch d'opht* 53:729 (Oct) 1936

Blepharochalasis has been considered an acquired pathologic condition by all authors reporting it, none having mentioned the possibility of hereditary transmission. Panneton reports that he has been able to trace 79 persons so affected in the same family. In 47 persons scattered through three generations there is evidence of this condition, as indicated by the history and photographs. The author has been able to examine the mother and 2 brothers of his patient. A genealogic tree is presented. From a study of this case the author suggests that sex is of no importance and that transmission can take place either through the father or through the mother. There is no characteristic heredity, as is the case in hemophilia, and the heredity does not follow the mendelian law. The author describes three degrees of the condition. He has found 93 cases described in the literature, all of which he includes in a table. A complete bibliography accompanies the paper. His conclusions are (1) that heredity can have and probably does have a part in the causation of blepharochalasis which has as yet not been recognized, (2) that there are degrees of the condition from the minimum of "baggy lids" up to blepharochalasis itself and (3) that it is from the anatomic standpoint that one must look for the essential cause of the condition.

S B MARLOW

Methods of Examination

CONTRIBUTION TO THE THEORY AND PRACTICE OF TONOMETRY J S FRIEDENWALD, *Am J Ophth* 20:985 (Oct) 1937

This comprehensive and rather mathematical article does not lend itself to abstracting. By allowing for the distortion of the cornea due to the foot plate of the tonometer irrespective of the indentation produced by the plunger, a method has been developed for calculating the true intraocular pressure which is free from errors owing to the rigidity of the ocular coats. For eyes of average rigidity, the pressure so determined is slightly higher than that determined by the Schiotz nomogram and is more nearly in accord with the experimental results of Wessely and Seidel on living eyes.

W S REESE

CONDEMNATION OF COLOR INDEXES IN CLINICAL PERIMETRY A MAGITOT and DUBOIS-POULSEN, *Ann d'ocul* 174:649 (Oct) 1937

In a medical or surgical clinic the objective examination of the patient is given precedence over the subjective. In the practice of ophthalmology the contrary is true, as so often the majority of methods consist in nothing but making use of the sensations of the patient. Research concerning the meaning of light in all its forms is considered so essential at the present time that Morax, with good reason, was accustomed to require its precedence over other exploratory methods.

As in all other subjective studies, the clinician must consider well the physical condition of the patient, that is, that he is not weakened by disease or disturbed with emotions or fear. Examination of the visual field must be done with patience and care and in a standard manner.

From the authors' comparison of the results of their study with colored and with white fields, the following conclusions are drawn. The

colored perimetry introduced in this research, which reflected on the instrument and on the patient himself, was the cause of numerous errors 2 The colored fields were no more exact than white fields, and a chromatic scotoma always corresponded to a scotoma for white 3 Perimetry with the white index must be done in a precise manner, the light must be constant and the fixation good A perimeter arc of 330 mm radius answers all clinical demands Usually for the peripheral field two dimensions of the index suffice For the zone between 10 and 30 degrees, an index of from 1 to 1.5 mm was used For the field between 30 and 90 degrees, an index of 3 mm is advised

S H McKEE

Neurology

LESIONS OF THE CHIASMA IN A CASE OF CRANIAL TRAUMATISM J MALBRÁN, Arch de oftal de Buenos Aires 12 150 (March) 1937

A case of cranial traumatism in the right frontal region is reported, in which injury was followed by loss of the right and left temporal fields and the lower visual field, vision being solely preserved in the upper nasal quadrant The presence of a lesion of the chiasma was confirmed at an operation to which the patient was subsequently subjected This proved of little benefit to the ocular defect

Malbian accepts Liebrecht's explanation of such cases, that is, the chiasma is torn apart by a violent broadening of the transverse diameter of the base of the skull, which follows a violent contusion of the frontal region He has not found any other case reported in the literature in which the presence of a lesion was confirmed by means of a surgical operation He refers to a study of such cases by Coppez, in which the latter reported 3 clinical cases and the results of several experiments

C E FINLAY

HISTOLOGIC EXAMINATION OF THE CEREBRUM IN UNILATERAL PERIPHERAL BLINDNESS REPORT OF TWO CASES A JUBA and A SZATMÁRI, Klin Monatsbl f Augenh 99 173 (Aug) 1937

Two cases of unilateral peripheral blindness are reported in which histologic examination of the brain was made Detailed descriptions, especially of the geniculate bodies and striated area, are given, and the relation between the pathologic cerebral changes and the blindness is discussed

The first case was that of a man aged 68 who died from carcinoma of the stomach The left eyeball was missing, the time of the occurrence of the ocular lesion was unknown Aside from the usual changes in the external geniculate body, alterations were found in the striated area which were associated with the lesion of the first optic neuron The atrophy of the fourth layer predominated

In the second case, that of a man aged 72 who died from a peritoneal tumor, the time of the loss of the right eye remained unknown The usual secondary and transneural degenerations were combined with degeneration of the papillomacular bundle, i e, the fibers of central vision, which apparently was due to chronic toxic retrobulbar neuritis Complicated but evidently uniform areas of degeneration had developed,

so that the almost mathematically regular construction of the external geniculate body could be observed by investigation of the transneural degenerations. The external geniculate body showed beginning secondary indistinctness of the distribution of the lamellar atrophy.

The visual cortex was intact in this case and in a third case, that of a patient suffering from bilateral atrophy of the optic nerve.

The authors explain the rarity of changes in the visual cortex in cases of unilateral peripheral blindness. In their opinion the lesion of the striated area is started by the transneural atrophy of the second optic neuron, the nutritive center of which is the external geniculate body. Hence, this lesion is a transneural atrophy of the second degree. Transneural atrophy is not intensive as a rule and is rarely accompanied by considerable destruction of cells. Therefore, the atrophy of the external geniculate body is bound to be intensive, as it causes certain changes in the visual cortex.

K. L. STOLL

Operations

TRANSPLANTATION OF THE CONJUNCTIVA FROM THE CADAVER'S EYE
EXPERIMENTAL PART, PRELIMINARY REPORT M. G. ROSENZWEIG,
Vestnik oftal 11: 311, 1937

The history of transplantation of the cornea and mucous membranes is reviewed. Experiments were done on 3 rabbits. In the first experiment a conjunctival flap, 6 by 4 mm, was transplanted from 1 rabbit to another. In the second experiment the conjunctiva was transplanted from a rabbit two hours after it was killed. In the third experiment the rabbit was killed, one hour later 6 cm of the blood was taken from the heart. The eye was enucleated and placed in that blood and kept in it for five days at a temperature of from 41 to 50 F. The conjunctiva from the enucleated eye was transplanted into a rabbit's eye and "took" well. In all 3 rabbits the transplanted conjunctiva looked normal in about ten days after the operation. Tarsorrhaphy was usually done after the operation, so that the flap would stay in place. Transplantation of a cadaver's conjunctiva which had been conserved for nine days in the blood of the donor was carried out, and the conjunctiva "took" well.

Thus the problem of conjunctival transplantation from a cadaver's eye conserved in cadaver's blood is solved in a positive way. The new source of the transplant opens wide possibilities in the field of plastic operations and in burns of the eye. The conservation of a cadaver's conjunctiva is connected with the problem of blood transfusion.

O. SITCHEVSKA

Orbit, Eyeball and Accessory Sinuses

ORBITAL VARICES F. J. SORIANO and H. R. PICCOLI, Arch de oftal de Buenos Aires 12: 408 (June) 1937

Two cases of orbital varices are reported. In 1 the varix was situated superficially at the orbital margin near the internal angle of the upper lid. The varix increased in size on increase of the cephalic pressure and disappeared when the patient was lying down. It had no influence on position of the eyeball. In the second case the varicose dilatation was

situated deep in the orbit. There was alternate production of enophthalmos and exophthalmos according to position of the head and to conditions affecting the congestion of this region of the body.

C E FINLAY

TOMOGRAPHY OF THE ORBIT. M. IBAÑEZ, M. ORIBE and M. MALENCHINI, *Arch. de oftal. de Buenos Aires* 12: 464 (July) 1937

After reference to difficulties in the interpretation of roentgenograms of the orbit due to the projection of cranial formations into the orbit, which are only partially avoided by serial roentgenograms of the cranium, including the frontal and the lateral views and pictures made in special positions, the authors consider the problem solved by the employment of tomography. This is a selective procedure which allows roentgenographic study in different planes and an appreciation of the anatomic structure and relations and even of small differences in roentgenographic density. In the future still more can be expected from this method with improvements in the apparatus and technic.

The authors review the literature on the subject, giving to Baese the credit for priority in first attempting to use the method and to Bocage the credit for inventing the procedure.

They describe in detail the structures to be observed in roentgenograms of the normal orbit taken in the frontal position at different levels separated 1 cm. from each other and also in those taken in a sagittal position.

They next consider tomographs taken with a special objective (visualization of the sella turcica, the sphenoid sinus and the foramen rotundum).

This is followed with a study of tomographs of pathologic orbits, with which their experience is limited, their usefulness being specially indicated in tumors of the orbit and of neighboring cavities.

They conclude that tomography permits observation in detail of lesions impossible to appreciate in ordinary roentgenograms and anatomic study in serial roentgenographic planes. They believe that the method is particularly useful in conjunction with ordinary roentgenograms taken in the classic positions, when the presence of a lesion is suspected but a more complete study is desirable. This paper contains the first description of orbital tomographs.

C E FINLAY

Physiology

CLINICAL STUDY OF CONSENSUAL OPHTHALMOTONIC REACTIONS. L. WEEKERS and J. FANCHAMPS, *Arch. d'ophth.* 53: 513 (July) 1936

Consensual ophthalmotonic reactions have been observed in animals experimentally as the result of many different procedures, which are listed in the article. The clinical study of these reactions has only just begun. Magitot has collected all the known data on the subject. Sometimes these reactions are striking clinically. More often they escape attention and must be looked for. The authors cite 2 cases in detail. The essential idea of consensual oculo-vascular reactions resulting in consensual ophthalmotonic reactions seems to them to be entirely justified.

and to be a fundamental physiologic phenomenon. Clinically, it is an aid in understanding facts. In some pathologic cases an inverse reaction occurs, but the explanation is clear and must be visualized in each case. Reference is made to the observations recorded in regard to other paired organs of the body. The question is raised as to how much a part this reaction plays in the development of sympathetic ophthalmia.

S B MARLOW

ENTOPTIC PHENOMENA—INTRARETINAL FLUIDS. E P FORTIN, *Arch de oftal de Buenos Aires* 12: 183 (April) 1937

In this paper Fortin discusses his peculiar views in relation to the structure of the retina, the ordinary description of which he deems faulty, due to observations made under unfavorable conditions and the production of artefacts. He considers that the retina is not composed of solid tissue but of a semifluid or gelatinous tissue, in which the different constituent elements float, and that it can be properly studied only by entopic observation with appropriate apparatus and with different kinds of light. He details at some length a description of the different layers and retinal elements observable by his methods and his views in connection with the same.

C E FINLAY

EXPERIMENTAL STUDY OF THE INFLUENCE OF MUSCULAR WORK ON INTRAOCULAR TENSION. V P FILATOV, I G YERSHKOVITCH and V E SHEVALEV, *Vestnik oftal* 11: 161, 1937

By the method of elastotonometriy Kalfa established the presence of a uveal reflex in the eye which regulates the intraocular tension. Filatov and his co-workers presume that substances of fatigue which accumulate in the blood after muscular activity stimulate the aforementioned reflex. Wolff and de Young observed a decrease of intraocular tension after convulsions. The authors undertook a series of experiments in order to verify these findings.

Rabbits were made to run in a closed wheel which turned around its horizontal axis. Stairs were laid out in the wheel so that the rabbit would not slip. The wheel turned from six to ten times per minute, and the experiment lasted from three to ten minutes. The tension was taken before and after the race. In all animals the tension was lowered from 7 to 14 mm of mercury after the race.

Muscle extract of the fatigued animals was introduced into the blood of control animals, it was also instilled into the conjunctival sac and injected under the conjunctiva, the intraocular tension was not influenced by these injections. Small doses (from 5 to 10 cc) of blood serum of the fatigued animals injected intravenously into the control animals produced no effect on the tension. Large doses (30 cc) of the blood of fatigued animals reduced the intraocular tension of the control animals considerably.

Thus these experiments confirm the hypothesis of Filatov that substances of fatigue accumulate in the blood and cause a decrease of intraocular tension. Further study of the subject is necessary, and it may bring about a better understanding of the cause of glaucoma.

O SITCHEVSKA

Retina and Optic Nerve

RETINO-CHOROIDITIS RADIATA T W BROWN, Brit J Ophth
21. 645 (Dec) 1937

An apparently unique condition was seen in a man aged 47 At 27 years of age he had an illness which was suggestive of spondylitis tuberculosa The eyes showed disseminated chorioiditis For a year he had had alopecia areata In each eye the disk was normal, with a zone of atrophy $1\frac{1}{2}$ disk diameters surrounding it Within this area was scattered pigment Partially confluent with this zone, extending peripherally almost to the equator, and centered on the retinal veins like beads on a string, were numerous patches of chorioidal atrophy Within these atrophic areas there were large prominent punctate masses of pigment, which generally ensheathed the vessels but sometimes lay below them The atrophic areas were $1\frac{1}{2}$ disk diameters in breadth and followed the distribution of the veins so exactly that in many places where the latter bifurcated so did the former They were in most instances bounded by retina of normal appearance, but here and there they were partially demarcated by a narrow zone of pigment Those parts of the veins in which no atrophy was present were in many instances delineated by a sheath of pigment The optic papillae were normal There was a low compound myopic astigmatism, with corrected vision of 6/9 in each eye The visual fields were contracted, with scotomas corresponding to the atrophic areas but with no defects of the nerve fiber bundles

The author could find no similar case reported in the literature and believes that the condition is a disease sui generis, for which the name retinochoroiditis radiata is suggested

W ZENTMAYER

SOME PATHOGENIC FACTORS IN RETINITIS PIGMENTOSA M MECCA,
Ann di ottal e clin ocul 65: 544 (July) 1937

Mecca defends a previous article against an attack by Schupfer Little new evidence is advanced, but Mecca insists on the importance of vascular spasm in cases of retinitis pigmentosa and on the efficacy of treatment by acetylcholine and other vasodilating agents

S R GIFFORD

PATHOLOGIC AND CLINICAL REPORTS OF THREE CASES OF EXUDATIVE
RETINITIS C S DAMEL, E ADROGUE and J MALBRAN, Arch
de oftal de Buenos Aires 12 384 (June) 1937

One of the 3 cases reported here had previously been reported by Sená New sections were made of the eye, which had been enucleated because of glioma, and the Malloiy method of staining was used Of the other cases, 1 was observed by Damel and the third by Adroque and Malbran In each, the eye had been enucleated because of secondary glaucoma

After a detailed study of the pathologic changes, which are illustrated by photomicrographs, the authors conclude 1 Exudative retinitis may exist without prior arteriovenous lesions, no signs of real angiomas being found in any of the 3 cases 2 The principal pathologic lesion

is the exudate, which at first detaches and later lifts the retina; calcareous deposits, choroidal degeneration, connective tissue transformation and cystic formations are secondary to the primary exudative retinitis 3 The pathologic picture in these cases confirms Koyanagi's discovery of the clear vacuoles which replace a large number of the pigment cells 4 The content of the clear spectral, or ghost, cells is albuminous The authors are inclined to favor Leber's hypothesis as to the origin of the clear cells Leber and Demaria consider them as proliferating pigment epithelium, which on separating from the lamina vitrea act as macrophages in the subretinal exudate

C E FINLAY

DARK ADAPTATION AND ITS DIFFERENTIAL DIAGNOSTIC VALUE IN CASES OF NEURITIS AND CHOKED DISK L DYMSHITZ, A KALIOMINA, L LUKOVA and K RAPOPORT, *Vestnik oftal* 11:176, 1937

Systematic examinations of dark adaptation were made during the past few years on 30 patients suffering from various diseases of the optic nerve There were 10 with choked disks, 11 with optic neuritis, 7 with retrobulbar neuritis and 2 with atrophy of the optic nerve The patients with choked disks were examined at the height of the development of the condition and in the regressive period In some there was a marked disturbance of the visual fields The Nagel adaptometer was used at a distance of 60 cm

The following conclusions were arrived at after a careful and prolonged study

1 There is a marked disturbance of the dark adaptation in cases of choked disk at the height of the development of the condition

2 A disturbance of the dark adaptation of varying intensity is also observed in cases of optic neuritis

3 The dark adaptation is partially preserved in the regressive atrophic period in cases of choked disk and optic neuritis to a degree not corresponding to the ophthalmoscopic changes in the optic disk

4 In cases of retrobulbar axial neuritis the adaptation is changed, but not as much as the visual acuity, which demonstrates the change also in the peripheral fibers

5 In some cases of axial neuritis the dark adaptation is markedly lowered, while the peripheral portion of the visual field is preserved

6 Examination of the dark adaptation in cases of disease of the optic nerve is a useful additional method for examination of the function of the eye, but its differential diagnostic value cannot be considered definite

O SITCHEVSKA

Society Transactions

EDITED BY W L BENEDICT

FRENCH OPHTHALMOLOGIC SOCIETY

Fiftieth Congress, Paris, June 28-30, 1937

TRANSLATED BY PERCY FRIDENBERG, M D, NEW YORK

First Session, Monday, June 28, 8 a m

OUR DISTINGUISHED COLLEAGUE POPE JOHN XXI (Lantern Slides)
DR H VILLARD, Montpellier

Pietro Hispano, born at Lisbon in 1215 or 1226, became pope on Sept 20, 1276. He died, crushed by the fall of a ceiling, on May 16, 1277. He wrote about a dozen philosophic treatises, of which the best known are the book "De l'âme" and, above all, the "Summulae logicales," which had such a great success that Dante referred to the author in his "Paradiso." Pope John also published two medical works, the "Thesaurus pauperum," which is a book on elementary medicine, and the "Liber oculorum," which gives one an excellent idea of what ophthalmic science was like in the thirteenth century.

SELECTION OF CONTACT LENSES BY MEANS OF METALLIC PROSTHESES
DR JULES SZYMANSKI, Warsaw, Poland

The prostheses are composed of two parts, a scleral and a corneal portion, identical to the Zeiss contact lenses but separate and made of metal. The scleral prostheses consist of three conical rings with a radius of 11, 12 and 13 mm each, with an opening of 12 mm above and one of 20 mm below. The adjustment is empirical. If the upper border projects too far, one adjusts the prosthesis lower; on the other hand, if it is the lower border which projects, one adjusts the prosthesis higher. The corneal curvature is calculated by a diagram and verified by the metallic corneal prostheses—the four shells with radiuses of 6, 7, 8 and 9 mm. These are placed on the cocaineized eye to see that they do not press on the cornea.

SURGICAL TREATMENT OF HIGH MYOPIA DR ALFONSO MOTOLESE,
Florence, Italy

This paper was not read

DEVELOPMENT OF THE LACRIMAL PASSAGES IN MAN (Lantern Slides)
DR GEORGES LEPLAT, Liege, Belgium

No abstract was submitted

SIMPLIFIED TECHNIC FOR THE TOTI OPERATION (Lantern Slides) DR
R RUBBRECHT, Biuges, Belgium

No abstract was submitted

DACRYOCANALICULORHINOSTOMY (Lantern Slides) DR BENEDETTO STRAMPELLI, Rome, Italy

This paper was not read

PALPEBRAL AUTOPLASTY DR EPIMACO LEONARDI, Rome, Italy

No abstract was submitted

DIPLOPIA PRECEDING A MARKED INCREASE OF GROWTH IN A DERMOLIPOMA OF THE CONJUNCTIVA DR JEAN SÉDAN, Marseille

A bilateral tumor which had been stationary since childhood started to grow rapidly on one side in a woman aged 36. This growth was preceded by the development of diplopia due to paralysis of the abducens nerve before there was any displacement of the globe inward. The diplopia, which was the signal symptom of the growing tumor, did not increase appreciably, and several months later the dermolipoma, the volume of which had increased enormously, crowded the globe inward. The tumor was removed in its entirety. The patient was syphilitic and poorly nourished and showed an irreducible Wassermann reaction of the blood. She had had 4 stillborn children. The rapid growth of the tumor and the peculiar diplopia, which evidently preceded the appearance of the tumor, are the rare and striking features in this case. The syphilis may have been the actual cause of the palsy which resulted in diplopia and also a factor in the proliferation of fatty cells. The growth was 85 mm long and 48 mm broad.

HISTOLOGIC AND CLINICAL STATISTICS ON EIGHTY CASES OF TUMOR OF THE BULBAR CONJUNCTIVA (Lantern Slides) DR HENRI TILLÉ, Paris, and DR PIERRETTE PILLET, Lyon

The authors report on the histologic classification and clinical prognosis of neoplasms, which are so variously interpreted. One category, that of nevocancer, is so predominantly frequent that one may say that the conjunctiva is, above all, a network of Langerhans cells. The usual cellular polymorphism of these tumors, which are epithelioid, sarcomatous, etc., is found, whether they are derived from a true fourth layer, whether they are of epithelial origin or whether they are of conjunctival origin, or, as Masson claims, of neural origin. Pigment is often absent, and this has to be brought out by nitration. Next in order of frequency are the epitheliomas of malpighian lineage. Aside from common benign new growths, certain varieties of cancer are invariably absent, especially the fibroblastic sarcomas. There are no neurinomas, which are so frequent in other regions of the body, and no neurogangliomas or sympathomas. It would seem that most of the tumors reported in the literature have to be regrouped and classified anew, especially the nevocancers. The following histologic material formed the basis of this study: nevocancers, about 15 per cent, epitheliomas, 8 per cent, papillomas, 7 per cent, cancerous papillomas, 2 per cent, various forms of nevi, including dermoepithelioma, 10 per cent, epithelial cysts, 8 per cent, dermoid cysts, 7 per cent, lipomas, 3 per cent, fibromas, 15 per cent, and lymphomas 15 per cent. There was

osteoma, probably a fragment of a dermoid cyst. Of the 18 nevocancers, 5 recurred repeatedly. Two of the patients died. One, a child of 3, had cerebral metastases within a year after exenteration of the orbit and the application of radium, the other, an adult, died within three years after exenteration. The prognosis in the 11 other cases was not clear. It seems to be favorable within from one to fifteen years. Of the 10 patients with epithelioma, 1 died in thirteen years after extensive recurrence, which extended to the tissues of the face, 4 had local recurrences. The subsequent course of the other tumors was not definitely determined. All the other 39 tumors were benign histologically. Follow-up study by the social service department confirmed this prognosis. The same can be said of 2 conjunctival lymphomas subjected to radiotherapy.

EXTENSIVE PANNUS TREATED BY RADIUM THERAPY (Lantern Slides)

DR C. ROCHE, Marseille

This paper was not read.

TREATMENT OF TRACHOMA ACCORDING TO RECENT EXPERIMENTAL RESEARCH. DR A. CUENOD and DR ROGER NATAR, Tunis, Tunisia

Preliminary studies proved that the virus of trachoma is connected with the presence of a morphologically demonstrable rickettsia. The louse is an essential carrier of the agent active in trachoma and at the same time is a reservoir for the virus of trachoma. Delousing must be added to the prophylactic measures for trachoma. Phenol is efficacious in the treatment of this condition. One must continue to apply "grattage" together with general treatment. The authors' results have been noticeably better since they have been using phenol in the form of subconjunctival injections or local applications, instillations, wet dressings and massage. This agent has a marked effect on rickettsias in general.

TETANUS FOLLOWING WOUND OF THE CORNEA. DR G. QUENTIN, Reims

No abstract was submitted.

INCIPIENT (FRUSTE) KERATOCONUS. DR MARC AMSLER, Lausanne, Switzerland

The textbooks and treatises deal almost exclusively with advanced, fully developed forms of this anomaly and with their well known characteristic signs and neglect the mild and abortive (fruste) forms, although the Javal-Schiötz ophthalmometer and Placido's disk allow one to detect them without much difficulty. Keratoscopic photography adds to this means of diagnosis the valuable aid of objective documents, bringing into light the flattening (*affaissement*) of the horizontal axis of the cornea, which constitutes the pathognomonic sign of keratoconus in its early stages. Its angle is measurable. Keratoconus can be classified into four groups on the basis of the degree of the condition. The last two groups include the cases of marked involvement and represent only 25 per cent of the total. The first two groups consist of cases of a mild degree and comprise 75 per cent of the total. Taking into account all

the cases of keratoconus which he has seen, the author finds that this condition is present in 1 of every 250 patients with ocular diseases, a proportion which is so much greater than one could expect from the statements and figures in the textbooks. The entire study of keratoconus must be taken up on a wider and more solid base.

HAS CORNEAL TRANSPLANTATION (KÉRATOPLASTIE PÉNÉTRANTE) ANY DEFINITE RESULTS? (Lantern Slides) DR NIZETIC, Belgrade, Yugoslavia

This paper was not read

CATARACT DUE TO DINITROPHENOL DR RENE ONFRAY and DR GILBERT-DREYFUS, Paris

The authors give a detailed report of a personal case and a summary of a case of Gallois, of Besançon. The first patient, a woman of 44, showed obesity without definite endocrine disturbance, rather the exogenous form due to overeating. For eighteen months she had been taking dinitrocresol, an English preparation, reducing her weight from 100 to 67 Kg. The treatment had been marked by the production of abundant perspiration, at which time vision became affected and cataracts appeared, becoming mature in one month. The right eye was operated on uneventfully by linear extraction and aspiration without iridectomy. However, four days later an attack of subacute glaucoma developed in the left eye, necessitating an emergency operation, iridectomy and aspiration of the cataract.

Gallois's patient, a woman of 48 who weighed 118 Kg, had taken a tablet of dinitrophenol daily every other week, and after seventeen months her weight had been reduced to 89 Kg. Cataracts developed in both eyes, they were operated on successfully without any untoward complications.

The authors made a complete and careful study of the nutrition of the first patient. The basal metabolic rate was practically normal, but there was a reversal of the relation of serum-globulin in the blood which was characteristic of lipoid nephrosis and of hepatic cirrhosis with a tendency to diopsy. They have demonstrated that dinitrophenol causes hepatic insufficiency. The ensuing toxicosis may bring about ocular disturbances in the form of glaucoma or cataract. In the latter case, a disturbance of water metabolism was probably brought into play, leading first to dehydration and later to a sharp (*brutale*) rehydration of the lens with imbibition of fluid and opacification.

Second Session, Tuesday, June 29, 8 a. m.

ACTION OF YOHIMBINE HYDROCHLORIDE ON INTRAOCULAR TENSION
DR RENE NECTOUX, Paris

Studies on normal and glaucomatous eyes were made. Data were given of some reactions observed after the subcutaneous injection or oral administration of 5 mg of this drug. The author takes up in detail the relation between variations in intraocular tension and those in systemic blood pressure. On account of its sympatholytic and vasodilating properties, yohimbine hydrochloride is capable of reducing

tension in certain forms of glaucoma, but hypertonic reactions are also noted if the local, i. e., intraocular, regulatory system, has been injured

SURGICAL TREATMENT OF GLAUCOMA DR BOURDEAUX, Evreux

The author, after having proved that up to the present time surgical intervention alone is capable of stopping the progress of glaucoma and so changing the prognosis of this condition for the better, regrets that many oculists still cling to treatment with miotics, the action of which is practically nil. He describes an operative technic, to which he has given the name of sclerostomy, which has been proved by more than 60 trials to be simple, reliable and efficacious. Aside from 1 or 2 cases, in which owing to faulty technic when the operation was in an early experimental stage of elaboration, there was a too early filling up of and obstruction (*colmatage*) of the scleral ostium, the results were excellent in at least 90 per cent of the cases. Tension was reduced permanently to normal without any complications and generally with a truly astonishing improvement in the filtering function (*?pouvoir séparateur* [P. H. F.]) and with preservation or, in some cases, extension of the visual field.

In the 2 cases in which the operation was not successful a second operation, which is always feasible, remedied matters. With this simple procedure one may banish blindness due to glaucoma. The author pays a tribute to oculists like Felix and Henri Lagrange, Holth, Elliot, Rene Onfray, Schiotz and Baillat, whose theoretic and practical studies, research and clinical and operative experimentation have contributed so greatly to changing the formerly grave prognosis of what was so long considered an incurable malady.

PATHOGENESIS OF OCULAR DISEASES OF DENTAL ORIGIN DR RENE HERMANS, Brussels, Belgium

The author reviews the two theories which have been in conflict since the reports of Fromaget (*Société française d'ophtalmologie*, 1924) and those of Worms and Becher (*Société française de stomatologie*, 1925) and discusses the arguments which have been advanced in favor of one or the other theory and which have appeared in the literature of the last twelve or more years. His data deal with ocular disease due to congenital dental anomalies and with those following eruption, disease, conservative treatment or extraction of teeth. In some cases, owing to an extension of the pathologic process, the pathogenesis is obscure and one may be dealing with reflexes or with the transportation of germs by way of retrograde venous channels, by way of the sinuses or by way of the general circulation.

RESISTANCE OF CAPILLARIES TO PRESSURE IN OCULAR TUBERCULOSIS DR VAN LINT, Brussels, Belgium

The fragility of blood vessels which is brought out by compression of the aim is so generally augmented in ocular tuberculosis that it may constitute an important symptom. The author's research has made of an inapparent symptom a visible sign. This sign plays a part in diagnosis and gives definite and possibly valuable therapeutic indications.

OPHTHALMOLOGIC ASPECTS OF A CASE OF CHORDOMA (Lantern Slides)
DR MICHEL ROCHE, Thiers, and DR MARTIN, Lyon

A man of 76 complained of lowered vision and nasal obstruction. Biopsy of the growth in the nose proved it to be a chordoma. Vision in the right eye was $\frac{3}{10}$ and in the left eye $\frac{4}{10}$, with correction. There was beginning atrophy of the optic nerve as indicated by the temporal pallor of both disks. The retinal arterial tension was slightly raised (45 and 55 mm). The most interesting clinical symptom was partial horizontal hemianopia on each side with a definite notch (*encoche*) in the upper part of the visual field. The sixth nerve was also slightly affected, and there were some symptoms of involvement of the trigeminal nerve. The authors were able to find reports of cases of cephalic chordoma in which ocular symptoms had been noted. Their study of this clinical material shows that such tumors may run either one of two opposite courses. They may originate intracranially and reach the nasopharynx through the sphenoid sinus, or they may originate on the ventral side of the spinal cord, develop first in the posterior sinuses, the walls of which they erode, and thus become intracranial (*encéphaliques*). All pairs of cranial nerves are successively involved in the course of this neoplastic process as it progresses to invade the orbit and the globe, a course which is inexorable and resists all therapy.

OPTICOCIASMIC ARACHNOIDITIS DR J BOLLACK, DR M DAVID and
DR P PUECH, Paris

Data obtained from a study of 129 cases of this condition, reports of 63 of which were collected from the literature, the reports of the other 66 being unpublished so far, are presented. The patients were seen in the neurosurgical service of the Hôpital de la Pitié. In all of these cases surgical exploration of the region of the chiasm had been carried out. Opticochiasmic arachnoiditis, which might be styled leptomeningitis involving the chiasm and optic nerve, is a provisional term which allows one to group certain histologic and clinical facts which should be kept separate from those pertaining to other conditions of this region which are at present well classified, such as tumors and certain inflammatory lesions. These cases of opticochiasmic arachnoiditis are characterized clinically, above all, by a symptom complex of visual disturbances of varying aspect, course and character and histologically by several types of pathologic lesions, a knowledge of which has been gained only by exploratory operations on this part of the cranial cavity. The historical part of this research shows that the present conception of opticochiasmic arachnoiditis is based on additions to the knowledge of localized serous meningitis. On the other hand, much has been gained by the development of neurosurgical procedures, which have disclosed the existence of pseudotumorous lesions of the opticochiasmic region and have also made possible the surgical treatment of certain diseases of the optic nerve. A special part of this study is devoted to the normal anatomic structure of this region. The various theories of the structure and constitution of the normal arachnoid and the pia mater were studied in collaboration with Professor Oberling, and the partitioned (*cloisonné*) space of the opticochiasmic region was studied. The portion of the study on path-

ologic anatomy includes the macroscopic and microscopic aspects of the lesion. The state of the meninges, the cortex and the chiasm as found by the neurosurgeon at operation is described. The authors discuss adhesive, cystic and mixed adhesive and serous forms of *arachnoiditis* and present many original illustrations. The microscopic data include those collected from the literature in addition to the detailed report, illustrated with important photomicrographs, of 3 unpublished cases of Professor Obeiling, including the observations made at autopsy. Clinically, as stated previously, the visual disturbances are variable in onset, course and degree. Changes in the visual fields, too, are of different types. Their order of frequency appears to be as follows: central scotoma, 31 per cent, concentric contraction, 23 per cent, and temporal narrowing, 17 per cent. Limitation of the nasal field (7 per cent), horizontal contraction (5 per cent) and lateral hemianopia (5 per cent) are more rare. The changes in the fundi are also variable. Simple atrophy with sharply defined margins of the disks (*à bords nets*) was found in 38 per cent of the cases, postneuritic atrophy with irregular (*flous*) borders, in 16 per cent, papilledema, in 10 per cent, partial atrophy of the temporal segment, in 7 per cent, simple hyperemia, in 7 per cent, and partial horizontal atrophy, in 4 per cent. Extraocular symptoms indicating an extension of the disease process to neighboring regions or the association with encephalitis are represented mainly by headache, somnolence and vertigo and more rarely by syndromes involving the infundibulum and the region of the tuber cinereum, giving rise to anosmia. The absence of changes in the cerebrospinal fluid and normal roentgenograms of the skull and ventricles are of great diagnostic value. Synthetic study of the symptoms allows the grouping of clinical forms. The extraocular forms may be symptomatic, associated or consecutive to the progress of the disease (*évolutives*). The most important forms are purely ocular. There are three principal symptomatic types: (1) the syndrome of macular or axial neuritis, with lowered vision, a large central scotoma, often associated with changes in the peripheral visual field, and various changes in the nerve head, postneuritic atrophy being the most typical, (2) the chiasmic syndrome, with lowered vision, atypical and asymmetric alterations of the temporal visual field, rarely purely defined, and papillary atrophy and (3) the syndrome of simple atrophy of the optic nerve, with concentric contraction of the field of vision. Topographic groups include (1) a prechiasmic type, indicating a predominant involvement of the intracranial portion of the optic nerve marked by central scotoma and/or concentric contraction of the peripheral field with various changes at the disk, particularly atrophy with irregular margins (*à bords flous*), and (2) a purely chiasmic form with contraction of the temporal visual field and simple atrophy of the optic nerve.

A positive differential diagnosis is difficult because the symptoms are polymorphous, no one, taken alone, being pathognomonic, and because a similar grouping may be met with in other conditions. Positive evidence is given by a clinical course marked by successive attacks (*poussées*) and such etiologic factors as trauma and infection of the accessory sinuses, and the association of signs indicating an involvement of neighboring regions and by encephalitic or meningitic complications. Negative findings such as a normal cerebrospinal fluid and a normal

roentgenogram of the sella turcica, are significant. Ventriculography is indispensable for differentiation between opticochiasmic arachnoiditis and tumors of the hypophysial region. The only procedure used by the authors is injection of air by bilateral occipital trephine puncture. The lateral and the third ventricles are normal in position and contour. They are sometimes dilated in so-called associated posterior arachnoiditis. The brain may be so edematous in some cases that the potential (*virtuels*) ventricles allow no air to enter on injection. It flows back and only dilates the subarachnoid spaces. Differential diagnosis from non-tumorous growths and allied conditions is rendered difficult by the fact that opticochiasmic arachnoiditis is not a strictly defined disease and that the question of its existence as a clinical entity arises continually. The axial neuritic form may closely resemble the changes in the optic nerve in cases of multiple neuritis, optic neuromyelitis, acute encephalitis, retrobulbar neuritis due to sinus disease, certain forms of retrobulbar neuritis localized in the chiasm and even, to a certain degree, in cases of Leber's disease. The form of opticochiasmic arachnoiditis associated with simple atrophy of the optic nerve and concentric contraction of the field has certain clinical and histologic characteristics in common with the tabetic involvement. Such were noted in the course of operation in cases of syphilitic atrophy of the optic nerve. This may open a new field to neurosurgeons. The form associated with a chiasmic syndrome is often difficult to differentiate from that which syphilis may present when it involves the chiasm, the meninges of the base of the skull or the bony structures of neighboring organs. The tumorous forms with prechiasmic localization must be differentiated from other intracranial neoplastic formations which give rise to a syndrome of direct compression of one or both optic nerves, from an olfactory meningioma and especially from a meningioma of the lesser wing of the sphenoid bone. Posterior forms may suggest the formation of a tumor in the hypophysial region rather than an inflammatory process. Roentgenograms of the sella turcica, which show changes in case of an adenoma, glioma of the chiasm or craniopharyngioma, allow a rapid diagnosis. Differentiation is more difficult in the case of less common neoplasms which are not accompanied by marked lesions of the sella, such as suprasellar adenoma, meningioma of the tuberculum sellae and cholesteatoma. Ventriculography and exploratory operation must settle the diagnosis in these cases.

Opticochiasmic arachnoiditis represents 27 per cent of all of the neurosurgical diseases that occur in the hypophysial region. Sixty per cent of the cases occurred in adults between 30 and 40 years of age. The most common etiologic factors appear to be sinus disease, cerebral or meningeal infection and traumatism. Acute, subacute or chronic disease of the antrum is a common factor, especially when it presents in the flaring up of an old chronic process with repeated relapses (*poussées*) and is often quiescent (*en sommeil*) when the meningeal localization becomes apparent. The majority of the cerebro-meningeal infections are due to syphilis, tuberculosis and colibacillosis should also be kept in mind. Encephalitis and multiple disseminated sclerosis, with special localization, exclusive for a long time, may create a special type of opticochiasmic arachnoiditis. Acute articular rheumatism and various forms of intoxication, exogenous or endogenous, have

been noted. Traumatism is often considered an etiologic factor. The intensity, localization and nature of the injury do not seem to play a decisive role. The time of appearance of symptoms is variable. When the condition develops a long time after injury, medicolegal questions may arise.

The pathogenesis varies according to whether the syndrome follows a disease by way of the circulation, by contiguous infection or by traumatism. The lymphatics play a role in complications of rhinitis. The visual disturbances associated with arachnoiditis may be considered (1) as a contemporary but independent manifestation of the infection which has attacked the optic nerve and the localized meninges simultaneously or (2) as a consecutive and subordinate manifestation to a preceding arachnoiditis, which is the basic phenomenon. In such a case there might be direct action by way of an arachnoiditic cyst or connective tissue strand (*bride*). However, it would be more logical to postulate an indirect action from these formations by way of disturbances of circulation in the vessels of the chiasm or the optic nerve. The visual disturbances may also be considered (3) as the primary lesion, involving the optic nerve fibers, the arachnoiditis being merely additional evidence, a secondary reaction. These three possibilities are not rigidly exclusive mutually. They may possibly be combined in some cases more or less, and the opticochiasmic arachnoiditis would then be expressed as an attenuated inflammation of the entire region.

Whether treatment consists of medical measures, radiotherapy or operation depends on the stage of the disease, the rapidity of its course and the etiologic factors. Surgical intervention becomes necessary when in spite of milder methods previously applied vision is failing, although not completely lost, and the visual field is steadily contracting or/and the central scotoma is becoming larger, before too gross changes in the fundus make their appearance. Operative exposure takes place by the direct (*droite*) transfrontal intradural route. The operative mortality is low. Cure or improvement was obtained in 37 per cent of the cases. The best results are obtained in recent cases in which the lesion of the optic nerve has not appeared or has not developed to a marked extent and in cases in which the eyes are fairly sound, that is, not previously diseased, and have good vision, or vision which has only just begun to fail, and show no marked contraction of the peripheral field and above all, no marked central scotoma and a fairly normal appearing nerve head, or at least one which is compatible with a fair amount of vision. Early surgical intervention appears to be all important in the preservation of vision.

SYPHILITIC OPTICOCCHIASMIC ARACHNOIDITIS AND PYRETOTHERAPY DR JULES FRANÇOIS, Charleroi, Belgium

A syphilitic patient, under treatment for a year and a half showed bilateral papilledema and rapid loss of vision. Neurologic examination gave negative results. Cytologic and chemical changes of specific type were found in the cerebrospinal fluid. Energetic treatment with mercuric cyanide failed to prevent total blindness, and decompression by trephining gave no result. Exploratory intracranial operation revealed opticochiasmic arachnoiditis and restored the vision in the left eye to about $\frac{1}{15}$. The right optic nerve was completely atrophic. The condition remained

unchanged after three months, and then fever treatment with sulfur in oil was instituted. The left eye recovered vision of $\frac{2}{3}$ with a practically normal field, and this improvement has held for more than a year.

Third Session, Wednesday, June 30, 8 a. m.

THE BLOOD SUPPLY IN THE RETINAL VESSELS DR FRITZ, Brussels, Belgium

Progressive compression of the central retinal artery between the minimum and the maximum brings about an increasingly marked reduction in the retinal perfusion indicated by a segmentation of the blood column in the retinal vein, a *courant veineux granuleux* (CVG). The current is at first rapid and then slows, and finally it stops when the compression reaches the maximum (Mx). Its appearance may serve to estimate the volume (*débit*) of blood entering the eye by way of the retinal artery. If this supply is copious, the current reaches the point of column segmentation only slowly, and the CVG does not appear until the Mx is approached. If, on the contrary, the supply is low, slight reduction results in the CVG, which may take place near the Mn (minimum). A formula for the blood supply indicates the relation, to Mn and Mx, respectively, of the compression necessary to produce the CVG in accordance with the equation $D = k + k' \frac{CVG - Mn}{Mx - Mn}$, in which k and k' are constants. A scanty supply, such that the CVG appears spontaneously, lowers the index to the value of k. A copious supply, such that the CVG can be caused only by maximal compression, Mx, gives the index a value of k + k'. Normally, the CVG appears in the second half of the vessel collapse (average k + k' 0.7). Its appearance in the first half of the compression phase is pathologic and has been observed in certain forms of arachnoiditis with vascular involvement (*entreprise*). Study of the blood output is calculated to give more definite indications of the state of the cerebral circulation.

RETINAL ARTERIAL SPASM, FATAL FORMS LACK OF ACETYLCHOLINE DR S GENET and DR R CHARPENTIER, Lyon

Observation of a young man who died after an attack of angina pectoris showed the authors that the functional disturbances and the ophthalmoscopic signs do not aid in detecting which forms of retinal spasm are ominous. The data of a general physical examination are almost as inconclusive. The primary cause of these spasms may be found in an initial lesion of the nervous system or in the changes in the walls of the vessels. The authors also consider the possibility of a lack of acetylcholine as an etiologic factor. Physiologic research has shown that the nervous influx which brings about the contraction of the blood vessels is interrupted at the junction of the terminal nerve fibril with the muscle fiber by the formation of a "chemical transmitter" which, in the case of the parasympathetic system is acetylcholine. This product is, on the other hand, the most active agent in the treatment of vascular spasms. Subjects with this disturbance suffer from an absence of acetylcholine, and this can be supplied by small but repeated and continued doses of the drug.

OSCILLOMETRY OF THE ARTERIES OF THE LIMBS IN THROMBOSIS OF THE
CENTRAL RETINAL VEIN (Lantern Slides) DR DUBOIS-POULSEN,
Paris

Patients with thrombosis of the central retinal vein can be divided into three groups on the basis of a normal, an increased or a lowered oscillometric index. In the first group the vascular disturbance is limited to the eye. The second group consists of persons with hypertension whose arterial walls have become distended by high blood pressure. The lowering of the index in subjects of the third group, who have become aged and hypertensive, indicates arteriosclerosis. In young persons it gives evidence of an arterial process which, it seems, cannot be connected with Buerger's thromboangitis.

RELAPSING RETINAL HEMORRHAGE AND DYSCRINISM (Lantern Slides)
DR VALOIS, Moulins, DR JEANDELIZE, Nancy, DR DROUET,
Nancy, and DR LEMOINE, Nevers

No abstract was submitted

RETINAL DETACHMENT IN TWO BROTHERS, CURE BY THERMO-
PUNCTURE DR ANTOINE BUJADOUX, Lyon

This paper was not read

CHANGES IN THE FUNDUS OCULI OF RABBITS WITH RABIES DR C
DEJEAN, Montpellier

The infection of rabies has its site of election in the cerebral cortex. One should be able to study the changes in this tissue by ophthalmoscopic study of the retina, which is embryologically nothing but an islet of that cortex which has migrated to the orbit. In the eyes of rabbits inoculated with the stable (*fixe*) virus, a progressive disappearance of the corneal and pupillary reflexes, paralysis of the muscles of the lids and eye, clouding of the transparent media and, finally and principally, changes in the fundus oculi were observed. The papilla was congested and more or less swollen. The retina was increasingly hyperemic. The vessels were dilated, and the veins were swollen and tortuous. The white fan of medullated nerve fibers, extending horizontally from either side of the disk, was invaded by numerous little vessels, previously invisible. This intense congestion would seem to give one an accurate picture of what is taking place within the cranium, to judge by the postmortem observations.

DIATHERMOCOAGULATION (WEVE) FOR DETACHMENT OF THE RETINA
RETINAL TEARS WITHOUT DETACHMENT DR NICO TRANTAS,
Athens, Greece

Rather than to try to localize the tear by measuring its distance from the limbus, it would be preferable to measure from the black band of the ora serrata, which is visible externally, by means of the transilluminator applied at the equator as well as with the ophthalmoscope under digital pressure (A. Trantas). To keep the cornea transparent during the operation, the author recommends, provisionally, a conjunctival flap

prepared by uniting the extremities of the equatorial incision for the detachment operation with two vertical incisions extending to the limbus. This covering can be removed for an ophthalmoscopic check-up. To prevent the escape of vitreous when the needle penetrates the sclera for coagulation, he uses fixation threads passed into the episcleral tissues, which are drawn taut at the moment of penetration to avoid pressure on the globe. A small incision of the sclera with a cataract knife at the site of the needle puncture facilitates still further the entrance of the needle without too much pressure on the globe. Tears without detachment are frequent. The author has seen 5 typical cases in the last nine months.

BLINDNESS IN INFANTS, WITHOUT OPHTHALMOSCOPIC CHANGES
(Congenital Forms) DR L. GENET and DR ROSNOBLET, Lyon

A case of congenital blindness in a child 13 months of age is reported. No infectious cause could be found in the parents or in the infant, who showed no malformations and nothing to indicate organic disturbance. Comparison of these congenital forms with the infectious forms seen after birth in association with encephalitis or meningitis may give one reason to believe that conditions of a similar nature may appear in the course of intrauterine life. An abiogenesis of the visual paths is also to be borne in mind. There have been no autopsies performed to throw light on this question, and the prognosis is in doubt, as such patients cannot be easily followed. The diagnosis, which is difficult during the cradle age, is easier when the child has begun to walk.

SECONDARY DEVIATIONS IN VARIOUS TYPES OF STRABISMUS DR
MARQUEZ, Madrid, Spain

No abstract was submitted

BILATERAL PARALYSIS OF THE INTERNAL RECTUS MUSCLE AND OF THE
MUSCLES OF THE IRIS "LOBSTER EYES" DR FROGÉ, DR
POURSINES and DR JEAN CHINIARI, Beyrouth, Syria

No abstract was submitted

ANEURYSMAL DILATATION OF THE OPHTHALMIC ARTERY CLINICAL
COURSE (Lantern Slides) DR JULES FRANÇOIS, Charleroi,
Belgium

In the case reported, in spite of intracranial operation, vision continued to fail and the visual field to contract. The fundus remained normal. Four months later a detachment of the retina supervened, at first in the lower half, but later becoming complete. The pupil showed an excellent red reflex with transscleral illumination. Some weeks later glaucoma developed and progressed rapidly, necessitating enucleation. When the globe was sectioned, a melanosarcoma of the choroid was revealed.

STUDY OF HEMATOGENOUS TUMORS OF THE ORBIT (Lantern Slides)
DR PASCHKEFF, Sofia, Bulgaria

No abstract was submitted

ENORMOUS BONY TUMOR OF THE OUTER WALL OF THE ORBIT, THE GREATER WING OF THE SPHENOID BONE AND THE TEMPORAL FOSSA DR JULES FRANÇOIS, Chaieioi, Belgium

The condition reported was first noted seven years previously. There were direct protrusion of the globe without diplopia or limitation of ocular motility, bilateral papilledema and normal vision. The visual field was slightly limited on the temporal side, in the left eye only. A prominent bony tumor, 7 cm in diameter, was found in the region of the temporal fossa. Roentgenographic examination showed marked clouding in the greater wing of the sphenoid bone, the lower outer part of the frontal bone, the lower anterior portion of the parietal bone, the anterior part of the temporal bone and the upper part of the malar bone. A diagnosis was made of osteoma, possibly eburnated osteosarcoma of the outer wall of the orbit, originating in the greater wing of the sphenoid bone and encroaching on the orbit (proptosis), the cranial cavity (bilateral papilledema) and toward the outside (tumor of the temporal fossa). Only 4 similar cases are found in the literature.

HERNIAS OF ORBITAL FAT DR J BOURGET, Paris

No abstract was submitted

FOUR CASES OF THE LAURENCE-BARDET-BIEDL SYNDROME TREATMENT WITH TOTAL EXTRACTS OF ANTERIOR LOBE OF PITUITARY GLAND (Lantern Slides) DR PAUL PESME and DR GABRIEL HIRTZ, Bordeaux

The authors report the observation of the complete adiposogenital syndrome, monstrous obesity of the cerebral type with genital hypoplasia and marked retinal changes, in children. The disk was dirty yellow, with indistinct outlines and markedly contracted arterial network. The region of the posterior pole had a lead gray hue, with a normal peripheral zone. There was entire absence of pigmentary overloading. This retinal degeneration was accompanied by a high degree of amblyopia, marked nystagmus and hemeralopia. There were anomalies of a pituitary type, marked relaxation of the ligaments, conical digits and genu valgum. Other symptoms pointed to involvement of the region of the infundibulum and the tuber cinereum: amenorrhea, polyuria and the marked improvement following the administration of anterior pituitary extract in the only patient so treated. The hypophysial pathogenesis of the syndrome seems to apply not only, as generally accepted, to the adiposogenital syndrome but, as the authors believe, to the retinal changes as well. The malady remains, etiologically, in the rubric of congenital malformations with a recessive mendelian heredity.

INTRASCLERAL GRAFTS WITH TENDON FROM DEAD ANIMALS AFTER EVISCERATION OF THE GLOBE DR HENRY PROBY, Evian

The author makes use of the tendons of stillborn calves, which can easily be procured from the slaughter house. The grafts are preserved in 90 per cent alcohol for one month and then kept in sterilized tubes in 60 per cent alcohol. His procedure does not make use of tendons fashioned into a spherical mass but of simple strips of tendon placed

gun-barrel fashion in the interior of the globe. Like all "dead" grafts, according to Nagoette's studies, they "take" easily. Photographs were shown of the eyeball and of a patient operated on with success one year previously. The procedure allows one to avoid enucleation in cases of panophthalmitis. The author was led to adopt this modification by his studies and good results in the use of dead tendon grafts intranasally in cases of ozena. Lantern slides showed the mechanism and the different stages of the revivescence of the grafts.

SEA AIR AND OPHTHALMOLOGY DR. JEAN RATEAU, Bordeaux

A marine climate congests the spongy tissues, the organic parenchyma and the cicatricial tissue, likewise, it acts favorably in the treatment of ozena and "osseous" otorrhea and should be useful in the treatment of follicular and phlyctenular keratoconjunctivitis. On the contrary, it is unfavorable in cases of chronic glaucoma and torpid iritis. Rateau spoke in praise of the climate at the seashore of Royan, which is tempered by pine forests.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY

Fifteenth Meeting, Cairo, Egypt, Dec 8 to 14, 1937

REPORTED BY S. R. GIFFORD, M.D., Chicago

The congress was officially opened on Dec 8, 1937, in the auditorium of the Egyptian University by His Majesty King Farouk. Professor Nordenson, of Stockholm, Sweden, chairman of the International Council, and His Excellency the Egyptian Minister of Health made addresses of welcome, after which a few words were spoken by representatives of the various countries. Mr. Arthur Sinclair spoke for the British Empire, Dr. Bailliart for France, Dr. Park Lewis for the United States, Professor Lohlein for Germany, Professor Bardelli for Italy, Professor Marquez for Spain, Professor Koyanagi for Japan and Professor Szymanski, of Poland, for the countries not otherwise represented.

The scientific program, which will be abstracted later, included not only the program of the congress itself but that of the International Organization Against Trachoma and of the International Association for Prevention of Blindness, each organization being allowed a full half-day's session for its deliberations. Time was allowed for visits to the many interesting places in and about Cairo. In these arrangements the local committee, in cooperation with the Egyptian government, was both thoughtful and generous. Automobiles and excellent guides were provided gratis for every one making the excursions. Visits to the principal mosques, the old Coptic church, the Citadel and the Egyptian, Arabic and Coptic museums were made by wives and relatives during the scientific program. The same ground was covered by participants in the congress in two half-day excursions, which also included visits to the Giza Memorial Ophthalmic Laboratory, two ophthalmic hospitals, the general hospital, Kasr-El-Aini, and a mobile hospital unit under canvas. In addition to these trips, two half-days

were set aside for excursions by the whole membership of the congress, with their wives and relatives. One of these excursions included a visit to the pyramids of Giza, with time and opportunity for camel and donkey rides, a visit to the Sphinx and a visit to the interior of the pyramid of Cheops. This trip ended with tea on the terrace of the Mena House. The other excursion, held on the last day of the congress, was to the Nile Barrage, a large dam below Cairo, with lunch on a steamer and a visit to the ophthalmic hospital of Rod-El-Farag.

In addition to all these arrangements for the comfort and amusement of their guests, an elaborate and enjoyable social program was provided for the evenings. His Majesty King Farouk entertained the members of the congress and their guests at the Abdine Palace, where delegates of the various countries were received personally by him. An elaborate program of music and dancing was presented in the palace theater and, in addition, an old Arabic play dealing with the romance of a blind princess and the possible cure of her blindness, apparently by the power of love alone, without benefit of ophthalmology. This was followed by a buffet supper and reception. Elaborate receptions and buffet suppers were offered to the members of the congress by the prime minister and by the Ophthalmological Society of Egypt. The final social event was a banquet at the Semiramis Hotel, at which Professor Nordenson spoke and called for brief replies from delegates of the various countries. The Egyptian government and the local committee were offered many expressions of thanks for their hospitality.

A meeting of the council and a business meeting of the entire congress were held on the last day. It was voted to accept the invitation of the Austrian government to hold the next International Congress in Vienna. It is planned for July or September of 1941 or 1942. Professor Nordenson reported that in response to appeals by the International Council the German Fuhrer had graciously consented to make a change in the status of Professor von Szily, of Munster, Germany. Professor von Szily, who had been forced to retire about two years ago, was given the position of emeritus professor, which involves a definite benefit as regards salary and status, though as yet no teaching duties. Professor Nordenson expressed the appreciation of the council that this exception to the German law had been made in view of Professor von Szily's unusual scientific contributions.

Professor Nordenson was reelected chairman of the International Council for a term of four years. Professor Maix, at his request, retired as secretary and was replaced by Holger Ehlers, of Copenhagen, Denmark. Several members of the council retired and were replaced as follows: Walter Parker, United States, replaced by Arnold Knapp, Wagenman, Germany, replaced by Lohlein, Parsons, England, replaced by Sinclair, Wright, British Dominions, replaced by Tooke, Montreal, Canada, and Oguchi, Japan, replaced by Nakamura.

The number of duly registered members of the Fifteenth International Congress was approximately 650. Of these, only about 350 attended the meeting, including 30 Americans. As regards the social and archeologic opportunities offered by the congress, enthusiasm was unanimous. The perfect weather, which allowed a program of outdoor excursions to be carried out without any interruption, was naturally a source of especial pleasure to guests from northern climates. The

scientific program, especially the part devoted to arterial hypertension of the retina and the endocrinology of the eye, was, in general, of excellent quality. A number of speakers reviewed work which had previously appeared in ophthalmologic journals, but it seemed to be of value to have this material presented at such a congress. The free communications were somewhat uneven in value but included some valuable papers. Some of the scientific exhibits were excellent, especially those of the government ophthalmic hospitals and the Giza Memorial Laboratory and of Dr. Uribe Troncoso, of New York.

Scientific Program

The two official subjects of the congress were "Arterial Hypertension of the Retina and Endocrinology of the Eye." Of 115 communications listed in the program of the congress, 25 related to the first subject and 17 to the second. There were 26 communications not related to the special subjects, 19 papers for the session of the International Organization Against Trachoma and 12 for that of the International Association for Prevention of Blindness. The sessions were presided over by representatives of various countries appointed by Professor Nordenson, including Sir Stewart Duke-Elder, Dr. Harry Gädle, Professor Lohlein and Professor Weve.

Mr. A. F. MacCallan conducted the session of the International Organization Against Trachoma and Dr. Park Lewis that of the International Association for Prevention of Blindness. The languages employed were English, French, German and Italian. English was employed not only by the British and American speakers but by Snapper and Weve, of Holland, von Imré and von Rotth, of Hungary, Mulock Houwer, of Java, Feigenbaum and Shimkin, of Palestine, and some of the Egyptian essayists. Discussion from the floor was permitted only by those who had filed copies of their discussion a day in advance with the program committee.

The papers presented will be published in full in the transactions of the congress. A brief summary will be given here. A number of the papers listed in the program were not read because of the absence of their authors, and these will not be considered.

The first official subject, "Arterial Hypertension of the Retina," was opened by Dr. Henry P. Wagener, of Rochester, Minn., who presented material prepared by himself and Dr. Norman Keith on diffuse arteriolar disease and hypertension. They concluded that arterial hypertension is an expression of a diffuse disease or abnormal condition of the arterioles throughout the body. Narrowing of the arterioles is due to increased tonicity of the wall of the vessels, to spastic constriction or to a combination of the two. It is not known whether this constriction is due to a pressor substance acting on the arterial wall or to increased vasomotor stimuli. If the causative factor continues to operate, histologic changes develop in the arterioles of various tissues, including the retina. Both functional narrowing and structural changes in the arterioles are present in cases of so-called essential hypertension, and a determination of the relative dominance of functional or structural changes offers a basis for classification of essential hypertension with reference to the disease. Such a distinction may be made ophthalmo-

scopically The retinal changes seen are due to decompensation of the retinal circulation While persons with such arterial disease may show signs of glomerulonephritis, all of the ocular changes and the symptoms leading to a fatal termination may be present in patients with diffuse arterial disease without any inflammatory disease of the kidneys

Baillart, of Paris, France, emphasized the value of examination of the retinal vessels by his method of dynamometry Certain technical difficulties inherent in the method may and should be overcome by proper technic, since the method offers the only means of detecting changes in the tonus of the retinal vessels in the period of functional constriction, before structural changes have occurred

Koyanagi, of Sendai, Japan, discussed the histologic changes present in the eyes of patients with hypertension He emphasized, as in previous articles, the importance of changes in the chorioidal vessels and of resulting degeneration and abnormal secretion of the pigment epithelium in the production of most of the retinal changes, especially those at the posterior pole Many photomicrographic slides were presented to illustrate the changes described The foregoing three papers constituted the official reports to the congress on the subject of hypertension

Lauber, of Warsaw, Poland, spoke on arterial hypotony and retinal atrophy He emphasized the importance of arterial hypotony as a cause of deficient blood supply to the retina, since blood reaching the retina must overcome the intraocular tension Hypotony may be the result of syphilitic vascular changes or of a functional condition When loss of vision occurs under these conditions, especially if it is more marked in the eye with the highest intraocular tension, it is to be treated by raising the blood pressure, often a difficult procedure, or by lowering the intraocular tension by the use of miotics or cyclodialysis

Mylius, of Hamburg, Germany, discussed the significance of transitory arterial hypertension in the genesis of retinal changes He emphasized the importance of studying the retinal vessels at a stage before structural changes occur In preeclamptic patients, spastic contraction is commonly seen, while tetanic contraction is more rarely observed Both conditions disappear after birth when the blood pressure returns to normal Retinal hemorrhages and deposits occur only when prolonged contraction of the arteries is followed by passive dilatation of the veins The cause of the retinal changes is not a lack but an excess of blood, but of blood poor in nutritive properties Restoration to normal often occurs even under these conditions, less often permanent changes are left

Klar, of Katowice, Poland, discussed retinal arterial hypertension in cases of commotio cerebri One hundred and fifty persons with injuries to the head were examined Those with commotio cerebri showed regularly an elevation of retinal arterial pressure and a contraction of the peripheral fields, which is apparently due to malnutrition of the retina as a result of the local circulatory disturbance The presence of increased retinal arterial tension is a valuable diagnostic sign in cases of injury to the head

Sabbadini, of Rome, Italy, discussed autonomic hypertension, the lowest common denominator of various severe ocular complications He investigated renal function, sugar metabolism and blood pressure in cases of thrombosis of the retinal veins, chronic glaucoma, hemorrhagic

retinitis and early disturbances of retinal circulation. Elevation of diastolic pressure was constant, hyperglycemia was common, and the retinal vessels showed signs indicating a venous imbalance, which he believed to be responsible for the various phenomena observed. He employed in treatment iodine, methenamine and a hormonal substance to produce vasodilatation.

The address of Volhard on the importance of ocular examination for an understanding of hypertension and nephritis was given by Thiel, of Frankfurt on the Main, Germany, who discussed the subject from the ophthalmologist's point of view on the basis of Volhard's findings as an internist. Volhard insisted that all the retinal changes formerly called uremic may occur without any signs of renal insufficiency and solely as a result of increased blood pressure. Generalized contraction of the arteries, in fact, is the causative factor in nephritis. Of 98 persons with acute inflammatory nephritis who were seen within six weeks of the onset, Volhard was able to obtain a cure in all but 1 by measures directed only toward a reduction of the blood pressure. If the vascular contraction is allowed to persist longer, changes in the kidneys and in the retina occur, resulting in the picture known as albuminuric retinitis, which should be called angiospastic retinitis. Thiel supported Volhard's view that the ocular changes are the result not of renal insufficiency but of increased blood pressure. He distinguished between the changes seen in so-called red hypertension (essential hypertension) and those in malignant angioneurosis (malignant hypertension). In the former condition sclerotic changes in the retinal and choroidal vessels are characteristic. Hemorrhages and areas of degeneration are rare. Edema and the macular star are absent. Tortuosity of the small venules (Guist's sign) and arteriovenous compression (Gunn's sign) are present. The transition stage to pale, or malignant, hypertension is marked by contraction of the arterioles, peripapillary edema and poorly outlined gray-white deposits. In definite cases of malignant hypertension the typical picture of angiospastic (albuminuric) retinitis is present. In forms of nephritis which run their course without elevation of blood pressure (acute glomerulonephritis and nephrosis), normal fundi are the rule.

Igersheimer, of Istanbul, Turkey, reported on the latent processes in the visual pathways in cases of hypertension. The visual pathways of 22 persons who died of hypertension were examined histologically. Two of the patients had shown no subjective or ophthalmoscopic signs during life, but histologic examination disclosed marked atrophy in the entire course of the optic nerves. The periphery of the nerve was predominantly affected in 1 specimen and in the other, the central portions. The retinal arteries showed advanced changes, and the atrophy of the optic nerve was considered secondary to impairment of the retinal circulation. The patients who died of uremia presented the typical fundus picture of neuroretinitis with papilledema. Isolated areas of degeneration were found in the chiasm in both instances. Igersheimer considered the degeneration to be due to increased intracranial pressure. He had previously reported a case of choked disk due to tumor of the brain in which similar changes were observed (*Folia ophthalmologica* 2. 1, 1935).

Fritz, of Brussels, Belgium, discussed isoperfusion and anisoperfusion of the capillaries in cases of retinal hypertension. He defined isoperfusion as the condition in which all capillaries are uniformly filled with blood and anisoperfusion as the condition that is present when certain vessels are spastic while others remain normal. When the vessels are equally contracted, increased cardiac effort succeeds in supplying the tissues with sufficient nutrition, while when local differences of vascular tone are present certain areas may receive too little blood and other areas too much blood. In cases of hypertensive retinitis the areas in which angiospasm occurs will show atrophic areas (anisospastic retinitis). Where tonus is insufficient, on the other hand, hemorrhages and exudates will be present.

Sallmann, of Vienna, Austria, discussed the diagnostic significance of retinal changes in cases of hypertension. According to him, tortuosity of the small venules (Guist's sign) is not of diagnostic importance, as it may be present in normal persons. Arteriovenous compression is of more importance and depends, as Sallmann's anatomic investigations have shown, on sclerotic changes in venous walls, resulting in a change in position of the vessels. It is suggestive of central hypertension, according to Kahler's classification, in which the cerebral vessels are assumed to be involved. In cases of hypertension of peripheral origin changes in the retinal vessels are absent.

Horniker, of Trieste, Italy, discussed the diagnostic value of ophthalmodynamometry. He described the results of examination by this method of 212 patients with diseases of the central nervous system. He considered the method, when employed with clinical, ophthalmoscopic and campimetric examinations, to be of great value.

Keyes, of Cleveland, reported on the work of Goldblatt and himself on experimental hypertension. Changes in the eyes of monkeys subjected to occlusion of the renal arteries by a clamp were described and illustrated by numerous excellent slides. The material has been presented in the *ARCHIVES* (17: 1040 [June] 1937) in abbreviated form. Dr. Keyes' presentation aroused much interest among Europeans interested in hypertension.

Seil, of Jena, Germany, illustrated by slides and moving pictures the process of spontaneous retinal pulsation. The record was made on a patient with intraocular tension of 45 mm., with exposures at intervals of one-sixteenth second. It was seen that the collapse of the retinal veins coincided with the cardiac systole and the filling of the veins, with the diastole. This indicates that the retinal venous pulse is not transmitted directly from the heart but is the result of an increase in intraocular tension which occurs during systole and which produces collapse of the veins.

Busacca, of São Paulo, Brazil, reported on the cerebrospinal pressure in cases of retinitis. He performed occipital puncture in cases of hemorrhagic retinitis associated with hypertension of various types. In some cases the cerebrospinal pressure was normal, and in these no improvement in the fundus picture occurred. In other cases, however, in which neuroretinitis and swelling of the disk were present, removal of the cerebrospinal fluid caused improvement in the vision and the fundus picture, although this was only temporary. In cases of arterial

hypertension and increased cerebrospinal pressure, with only moderate edema of the disk and retina, the improvement was especially marked.

"Endocrinology and the Eye," the second official subject of the congress, was introduced from a general point of view by Professor Snapper, of Amsterdam, Netherlands. He emphasized the complex nature of endocrine disorders and the frequency with which hyperfunction or hypofunction of one gland is accompanied by or results in dysfunction of other glands. Illustrations are the common occurrence of gonadal and pancreatic insufficiency in cases of disorders of the pituitary gland and of pituitary dysfunction in cases of Addison's disease. A practical point worth remembering is Professor Snapper's opinion that 80 per cent of reports concerning the blood calcium content as an indication of parathyroid dysfunction are of no value, since not the total calcium content but the ultrafiltrable portion is the important constituent of the blood, and its determination requires careful control of the hydrogen ion concentration in serum and filtrates.

Von Szily, of Munster, Germany, discussed dysfunction of the thyroid gland and its relation to the eye. He also emphasized the frequency with which the picture of thyroid dysfunction is complicated by accompanying disorders of other glands.

He reviewed previous work on the pathogenesis of exophthalmos and other ocular changes in cases of hyperthyroidism. There seems to be evidence that hyperthyroidism alone will not account for the complete picture of progressive exophthalmos, especially the type showing progress after thyroidectomy. To explain this, existence of an x hormone secreted by the pituitary gland has been assumed which continues to be effective after thyroidectomy.

Von Imré, of Budapest, Hungary, discussed ocular diseases connected with gonadal dysfunction. He mentioned cases in which subconjunctival hemorrhage, conjunctivitis and phlyctenulosis occurred periodically in relation to menstruation. Filamentous keratitis due to atrophy of the lacrimal glands is recognized as commonly associated with the menopause. According to von Imré, keratoconus is often due to gonadal insufficiency and cases are reported in which extracts of a potent estrogenic or androgenic substance were effective in favorably influencing this condition. He stated that glaucoma is often connected with gonadal dysfunction. In 4 glaucomatous patients the production of estrogen was found to be abnormally low, while the amount of gonadotropic substance was normal.

Jeandelize and Drouet of Nancy, France, presented a report on the hypophysis and its relation to the eye. They reviewed the anatomic structure and the physiologic function of the gland as well as the pathologic changes which have been observed. Aside from the better known facts, they emphasized the effect of the gland on the production of pigment in the retina and the effect of light received by the retina on the function of the pituitary gland. The syndrome of Laurence-Biedl is considered as due to a lesion of the hypophysis, with resulting pituitary insufficiency. The gland may play an important part in recurrent retinal hemorrhages, and the signs of enlargement of the pituitary gland during pregnancy are well known. The influence of the pituitary gland on the production of cataract and glaucoma cannot as yet be determined.

Lo Cascio, of Naples, Italy, discussed the anatomic structure and physiologic function of the parathyroid gland in relation to the eye. Aside from the well known form of cataract occurring after parathyroidectomy, other forms of cataract associated with tetany were discussed. Among these are lamellar cataract in children with tetany, subcapsular cataract associated with the tetany in mothers and cataract associated with tetany of unknown origin. In rabbits submitted to parathyroidectomy the following changes were found: (1) a slight elevation of the p_H of the serum and crystalline lens, (2) a decrease in the ascorbic acid content of the aqueous and the lens, (3) a decrease in the glutathione content of the lens, (4) a decrease in the glycolytic power of the lens, (5) an increase in the calcium and ammonium content of the lens, (6) slight displacement of the isoelectric point of the lens to the alkaline side and (7) changes in the capsule of the lens comparable to those in senile cataract. It is likely that similar processes occur in the development of cataracta parathyreopriva in man. While the use of parathyroid extracts cannot clear opacities of the lens already formed, it can stop their progress.

In addition to the foregoing official reports, the following communications on the subject of endocrinology and the eye were offered:

Wibaut, of Amsterdam, Netherlands, reported on retinitis pigmentosa and internal secretion and treatment with estrogen. In addition to the fact that more men are affected than women (61/39), Wibaut stated that he had seen more cases in men than women (19/6) in which the course was unfavorable. He has seen other evidences of endocrine imbalance in his patients. He reported favorable results with the use of a potent estrogenic substance in both men and women affected by the disease.

Bietti and Rubegni, of Rome, Italy, reported the use of the interferometer for the estimation of endocrine function in ocular diseases. They employed the instrument of Hirsch on 100 patients with ocular conditions suggesting a possible endocrine origin as well as on a number of other persons. In cases of blepharochalasis, dysfunction of the thyroid gland was found, with some signs of hypophyseal and gonadal dysfunction, in cases of vernal catarrh, dysfunction of the adrenal glands and gonads, in cases of keratoconus, chiefly dysfunction of the thyroid gland and thymus, and less markedly of the gonads and the hypophysis, and in cases of tapetoretinal degeneration, dysfunction of the thyroid gland and the hypophysis. In cases of juvenile cataract the results were conflicting or negative.

De Cori, of Florence, Italy, reported on the action of vitamin C on human cataract. The results were almost entirely negative.

Pascheff, of Sofia, Bulgaria, discussed some ocular problems in the field of endocrinology. He reported a case of bilateral exophthalmos in which there were no other symptoms of exophthalmic goiter but almost complete loss of the corneas. He stated that certain forms of retinal degeneration must be related to the hypophysis and the hypothalamus, the center of the vegetative nervous system. He emphasized the association of retinal degeneration without pigmentation with adiposity, polydactyly and mental debility, of retinitis pigmentosa with infantilism and disturbances of the vegetative system and of acromegaly with glaucoma.

Blatt, Bratianu, Joviu and Milcou, of Bucharest, Rumania, discussed cataract in relation to dysfunction of the hypophysis. Thirty-seven patients with senile cataract were studied from an endocrinologic standpoint. The study included roentgenographic examination of the sella turcica, a basal metabolic test, chemical analysis of the blood, urinalysis and biologic tests for gonadotropic, luteinizing and estrogenic substances. All but 9 patients showed abnormalities of the sella turcica of various types. The luteinizing factor was absent from the urine of all patients, and the gonadotropic principle was present, as a rule, in reduced amounts. An increase of potassium and cholesterol in the blood was fairly constant. The authors expressed the belief that a causal relation between hypophysial insufficiency and cataract is likely.

Free communications included papers by the following men:

Lenz, of Breslau, Germany, presented a report on stereophotography of incipient senile cataract. Slides and photographs shown in the scientific exhibit demonstrated the faithfulness with which a record of early opacities of the lens can be made by the transmitted light of an arc lamp. The opacities show dark against the light background.

Shimkin, of Haifa, Palestine, reported on antepositio conjunctivae fornicis in cases of severe spring catarrh. In 29 cases of severe involvement he dissected all diseased tissue from the tarsus, which was found to be healthy. The upper fold was then dissected freely and sutured to the tarsus near the border of the lid, thus covering the healthy tarsus with relatively healthy conjunctiva. Results were satisfactory.

Taumi, of Tallinn, Estonia, reported on the operative treatment of astigmatism. Operation was performed on 21 persons with astigmatism greater than 3 D. In cases of astigmatism with the rule a portion of the cornea near the upper limbus was resected beneath a conjunctival flap and the incision was closed with aplanation sutures. In cases of inverse astigmatism a spade incision was made and enlarged with scissors. Remarkable results are claimed, but only a few patients were observed for more than a year.

Rauh, of Leipzig, Germany, discussed the experimental basis for the treatment of tetany cataract. Parathyroidectomized dogs and rats were treated with a form of irradiated ergosterol (A T 10). The blood calcium became normal under such treatment, and tetany and cataract were prevented. If treatment was begun in the early stages of cataract the opacities of the lens cleared almost entirely, while if the opacities were already extensive they remained permanently in a form resembling lamellar cataract.

Velhagen, of Halle, Germany, presented a film on examination of the eye. Tests for orthophoria, eversion of the lids, removal of foreign bodies, examination of the lacrimal passages, tonometry, focal illumination, slit lamp microscopy, skiascopy, ophthalmoscopy, photography of the fundus and dynamometry were covered in the films.

Muller, Bruning and Sohr, of Berlin, Germany, discussed graduation of the Bailhart and Sobański dynamometers on the human cadaver. By employing bodies immediately after death, they found that the scales in use with these instruments were unreliable. The age of the patient plays a part in the values found, a series of scales for various ages being necessary. It is proposed that centers be organized for the standardization of instruments now in use.

Buckleis, of Tübingen, Germany, discussed the causation of zonular cataract. He examined 70 children who had been under treatment for rickets and spasmophilia in infancy. None showed any signs of cataract. He stated that heredity is responsible in most cases of zonular cataract.

Richman, of Brooklyn, N. Y., presented results of an ophthalmoscopic study of newborn infants. Five hundred and thirty-one infants were examined, of whom 12.2 per cent showed retinal hemorrhages. The incidence was greater (17.5 per cent) in first children than in those born later and was greatest in those delivered by forceps (20.6 per cent). Colored drawings in the accompanying scientific exhibit showed the type of hemorrhages observed and in some cases their absorption at the time of later examinations.

Lindner, of Vienna, Austria, presented a clinical study of the vitreous. The importance of shrinkage and liquefaction of the vitreous was illustrated by observations on animals with an artificial fistula of the vitreous and on human eyes with detachment and other conditions, as observed with the plane contact glass and the angled microscope of the author.

Bietti and Rubegna, of Rome, Italy, presented a discussion on the blood calcium in cases of juvenile cataract. The total and the ultrafiltrable calcium contents were determined for a series of patients with no history of tetany, and tests of muscular irritability were also performed. The findings showed no important variations from normal.

Klar, of Katowice, Poland, discussed miner's nystagmus from the standpoint of etiology. The rarity of this condition in the Polish-Silesian coal region as compared with the incidence in other regions was explained by the author as due to the difference in the coal deposits. In this region the gases generated by the coal are much less harmful than in other regions, where carbon monoxide, methane and other gases are important factors in causing the condition.

Troncoso, of New York, presented a report on the anatomic structure and evolution of the iridocorneal angle in mammalia. The angle in rodents, ungulata, carnivoia and primates was described and illustrated. An accompanying scientific exhibit showed the conditions described most clearly by means of dissected specimens mounted and placed in the slit lamp beam. Schlemm's canal first appears in the primates.

Schupfer, of Florence, Italy, discussed the resolving power of the human retina. By means of a diffraction screen and monochromatic light of 5,400 angstroms it was found that the resolving power varied between 115 seconds and 36 seconds in various observers. In most cases it was below 60 seconds.

Nastri and Valeri, of Rome, Italy, reported on the cholesterol metabolism of patients with retinal lesions. Fifteen diabetic patients with retinal lesions and 9 without retinal lesions were examined. In 9 of the patients with retinal lesions the blood fats were increased, while only 2 of those without retinal lesions showed such an increase. The total cholesterol content was usually normal, but in patients with retinal lesions there was a tendency for the free cholesterol to increase and the cholesterol esters to decrease, as compared with the findings in patients without retinal lesions. The retinal blood pressure was elevated, as a rule, in the patients with retinal lesions.

Zeiss, of Wurzburg, Germany, presented an apparatus for measuring and recording disturbances of ocular motility. The instrument, a projection coordimeter, is made by Zeiss (demonstration).

De Cori and Wiechmann, of Florence, Italy, discussed the effect of the p_H on miotics and mydriatics. A shift in p_H to the alkaline side increased the speed, effectivity and duration of action both of miotics and mydriatics. The optimum p_H was found to be 8.2.

Duggan, of Bombay, India, reviewed the various ocular disorders observed in diabetes.

E. V. L. Brown, of Chicago, reported on the yearly changes in refraction from birth to beyond middle life. Eight thousand, eight hundred and twenty observations were reported on the eyes of 1,203 persons examined under atropine cycloplegia on several occasions one or more years apart. According to these observations, hyperopia increases from birth to the age of 7. Myopia develops and hyperopia decreases mainly between the ages of 8 and 13, the increase in refraction averaging 0.23 D per year. Between the ages of 14 and 20 myopia increases further but at a slower rate, seldom increasing at all after the age of 20. After this age the changes in refraction are slight, there being a slight increase up to the age of 33, then a slight decrease up to the age of 42, followed by a more regular increase up to the age of 51.

Pascheff, of Sofia, Bulgaria, discussed the corneal and scleral forms of vernal conjunctivitis. An attempt was made to classify the various changes observed, which the author prefers to call fibropapillary hyperplasias.

Teissler, of Prague, Czechoslovakia, described the use of a new type of contact glass made of pyroxylin. Such prostheses mold themselves to the globe to some extent and are worn by certain patients who do not tolerate glass prostheses. It cannot yet be stated whether their advantages are such that they will replace glass prostheses.

Gillessen, of Dusseldorf, Germany, reported on diabetic and tetanic cataract in relation to Krauss' theory of the muscle function of cortical cells. Experimental evidence was offered that the cortical fibers of the lens possess contractile power, as shown by the use of acetylcholine and histamine. Refraction was increased, as shown by skiascopy, after any effect of the ciliary muscle was excluded by atropine, the change amounting to 12 diopters. The knowledge of such contractile power in the lens explains certain phenomena of presbyopia as well as the process of tetanic cataract.

Hildesheimer, of Tel Aviv, Palestine, discussed the possibility of decreasing myopia by scleral excision with the electric knife and also the treatment of cataract with preparations of strontium. Photographic records showed apparent clearing of some opacities of the lens under treatment.

Demonstration Session

Lincz, of Budapest, Hungary, demonstrated a comparison microscope, with which the anterior segment of both eyes could be observed simultaneously.

Hughes, of New York, demonstrated by moving pictures a new method for rebuilding a lower lid.

Krausz, of Lodz, Poland, showed a combined short wave and ionization apparatus, which he employs chiefly in trachoma

Goldmann, of Berne, Switzerland, demonstrated an improved slit lamp

International Organization Against Trachoma

Jan 10, 1938

MR A F MACCALLAN, *Presiding*

The scientific meeting was open to all those attending the congress, a short business meeting for delegates and members of the organization being held after the program. Membership is open to all ophthalmologists by application to the secretary, Dr Wibaut, of Amsterdam, Netherlands

The annual membership fee is 1 pound, 5 shillings, which includes subscription to *Revue internationale du trachome*

In his presidential address, Mr MacCallan gave a brief history of the fight against trachoma in Egypt, the modern phase of which was begun by him with the foundation of the ophthalmic hospitals, the traveling units under canvas and courses in the treatment of trachoma for native physicians. The most recent accomplishment in the work was the founding of the Memorial Ophthalmic Laboratory at Giza

Thygeson, of New York, discussed the causation of trachoma by a virus. Work by himself and others was reviewed, indicating that trachoma is due to a virus which only passes filters with rather large pores. The elementary bodies found free and in the epithelial cells seem to represent the visible form of the virus, and their presence in filtrates is necessary for successful inoculations. Thygeson stated that he does not believe that the bodies described in the follicles by Busacca and others are parasitic in nature

Grueter, of Marburg, Germany, reported on the microstructure of the epithelial cell and the significance of such cells in the causation of trachoma. He described and illustrated structures in the cells which resemble the Halberstadter-Prowazek bodies and which he considered reaction products of the cells to various abnormal influences, including that of the invisible agent of trachoma

Von Roth, of Budapest, Hungary, reported the results of experimental work which confirm Thygeson's conception. In tissue cultures inoculated with Elford filtrates from active trachoma it was possible to demonstrate inclusion bodies in the surviving epithelial cells

Oguchi and Majima, of Nagoya, Japan, concluded that so-called paratrachoma, including inclusion blennorrhoea and swimming pool conjunctivitis, is a virus disease due to the Halberstadter-Prowazek virus, but that these bodies are not characteristic of true trachoma. When they are found in cases of trachoma it is considered that a mixed infection with the paratrachoma virus is present

Poleff, of Rabat, Morocco, from work with tissue cultures and the inoculation of animals, concluded the rickettsia bodies described by Busacca and by Cuenod are the cause of trachoma. He considered that these bodies, which are found in the follicles, probably represent the same virus as the Halberstadter-Prowazek bodies found in the epithelial cells

Major Stewart, of the Giza Memorial Laboratory, read the report of Julianelle and his associates on their work, the essentials of which have been published in American medical journals

Wilson, of the Giza Memorial Laboratory, reviewed the pathologic picture of trachoma. The lesions occurring in the stages of trachoma as classified by MacCallan were described and illustrated

Pascheff, of Sofia, Bulgaria, described the formation of follicles in a mucous membrane graft applied by the Denig technic for pannus. Portions were removed at two different periods and the formation of a large true folliculoma from a number of small follicles could be demonstrated

Michail, of Cluj, Rumania, reviewed recent observations made on the histologic picture and the pathogenesis of trachoma

Busacca, of São Paulo, Brazil, reviewed his recent work on the development of trachoma in the cornea. By marking areas studied with the slit lamp and removing these for section, he obtained a clear correlation of the histologic changes with the clinical signs

Mulock Houwer, of Batavia, Java, discussed acute trachoma on the basis of 12 cases. Typical of all cases was the presence of numerous fine infiltrates of the cornea and corneal edema with folds of Descemet's membrane. The conical lesions had disappeared when typical trachomatous changes in the lids developed

Sobby Bey and Samaan, of Cairo, Egypt, discussed the treatment of trachoma. All of the usual measures were employed, but especial value was given to the use of quinine, which has replaced chaulmoogra oil in recent years. Without apparently knowing of Selinger's work, Samaan developed an ointment containing both quinine alkaloid and copper for a year or more

Shimkin, Haifa, Palestine, reported on the use of auricular cartilage for the correction of severe entropion following tarsectomy. Good results were obtained in 5 cases

International Association for Prevention of Blindness

Dec 11, 1937

PARK LEWIS, M D, *Presiding*

MacCallan, of London, England, discussed a national policy to be adopted in a tropical country for the prevention of blindness. The same subject was discussed by Sadek of Tantah, Egypt

Maziny Bey, of Cairo, Egypt, discussed the social aspect of the prevention of blindness

Weve, of Utrecht, Netherlands, discussed the spread of blindness in the Dutch East Indies

Miyashita, of Tokyo, Japan, described work in the schools in Japan for children with defective sight

Pascheff, of Sofia, Bulgaria, described the struggle against blindness and the new law of public assistance in Bulgaria

Toulant, of Algiers, Algeria, discussed difficulties of the prevention of blindness in a tropical country

Park Lewis, of Buffalo, New York, discussed the responsibility of the ophthalmologist in the conservation of sight and the prevention of blindness

Bailliart, of Paris, France, discussed social service in an ophthalmic hospital

Carris and Merrill, of New York, discussed the role of the social worker in a program for the prevention of blindness

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W WHITE, M D, *Chairman*

Jan 17, 1938

RUDOLF AEBLI, M D, *Secretary*

SIMPLE SURGICAL PROCEDURE FOR RELIEF OF SPASTIC ENTROPION DR S A AGATSTON

Spastic entropion is frequently encountered in elderly persons who manifestly have no other complaint. The condition is caused by the combination of loose skin and a soft tarsal plate, by anophthalmos or by the protracted use of a bandage after a surgical operation or during prolonged chronic inflammatory conditions. The patients suffer great discomfort, principally on account of the lower eyelashes, which irritate the cornea, causing pain, photophobia and tearing.

To relieve this condition, many operations have been devised, and it is with hesitation that I add another to the long list. Among the accepted surgical procedures, I might mention the following techniques: Snellen's suture, Gaillard's suture, Ziegler's cautery puncture, Janson's three vertical scars, Graefe's excision of a triangular piece of skin, Flaxer's removal of cilia by splitting the gray line and the Jaesche-Ailt modification of Flaxer's operation.

The last-mentioned operation consists of removal of a horizontal crescentic piece of skin below the cilia, after which, instead of removal of the cilia, the ciliary margin is sewed to the lower border of the wound, the space between the cilia and the gray line being allowed to heal by granulation.

Other methods of treatment consist of the Hotz operation of removal of the fibers of the orbicularis muscle close to the inferior tarsus and of Hughes' injection of alcohol into the orbicularis muscle. Wheeler recently devised a satisfactory operation in which the fibers of the orbicularis muscle are freed and attached to the periosteum of the orbital margin or divided vertically and overlapped. Wilder's operation consists of a combination of canthoplasty and the Snellen-Streatfield operation. After doing the canthotomy, he horizontally cuts some of the fibers of the orbicularis muscle from the raphe of the muscle.

The operation which I wish to describe was first done on a patient who failed to respond to the injection of alcohol or to Gaillard's, Ziegler's or Hotz's operation.

The technic of the operation follows. A short canthotomy is done, followed by a U-shaped dissection of the conjunctiva at the outer canthus and dissection of the skin below the canthotomy incision, away from the orbicularis muscle. The orbicularis muscle is picked up at its attachment to the tarsus, a vertical incision is then made with scissors through the orbicularis muscle, detaching it from the tarsus. With a double-aimed silk suture, the two needles are inserted through the U-shaped conjunctival flap, and the needles are passed down and out at an angle of 45 degrees, to emerge 1 cm away from the cut end of the external canthus, fair traction is exerted, and the suture is tied. The conjunctival flap completes the canthoplasty and by downward traction holds the tarsus away from the cut edge of the orbicularis muscle.

The results, so far, have been satisfactory for from six to eight months. I have done only a few such operations, but in none of the cases has there been a recurrence in from two to eight months. The cosmetic results are also good.

COMPARISON OF OPERATIONS FOR PARALYSIS OF THE LATERAL RECTUS MUSCLE DR BRITTAN F PAYNE

The cases presented here demonstrated two types of operations performed for paralysis of the lateral rectus muscle. In the first case transplantation of the lateral halves of the tendons of the superior and inferior rectus muscles with recession of the medial rectus muscle was performed. In the second case a simple resection and recession gave excellent results. The condition in the latter case was more favorable for operation in that the paralysis had existed a shorter time and was specific in type.

CASE 1—Mr J V, an Italian aged 24, in January 1931 suffered from double vision of sudden onset, with a converging left eye. His family history revealed no evidence of squint or other ocular diseases. Results of the general physical examination were negative. The Wassermann reaction was negative. The neurologic examination gave negative results, with the exception of paralysis of the left abducens nerve.

Examination revealed vision to be 20/20 in the right eye and 20/70 in the left eye, which was improved to 20/40 + with glasses.

The left eye deviated nasally approximately 45 degrees, as measured on the perimeter. No abduction was present. The eye could be rotated vertically, but no amount of effort carried it to the primary position laterally. External examination and ophthalmoscopic examination gave essentially negative results. Refraction under cycloplegia showed slight hyperopia in the right eye and a small amount of myopic astigmatism in the left eye. An attempt to plot the fields for diplopia was unsuccessful. The visual fields showed concentric contraction for form in both eyes, with good central color perception and normal blindspots. With the aims of the orthoptoscope placed convergent at 35 degrees, second grade fusion was attained.

Owing to the fact that the patient suffered from discomfort due to the diplopia for six years, transplantation of the lateral halves of the vertical rectus muscles with advancement of the lateral rectus muscle was performed at the New York Eye and Ear Infirmary on Aug 13, 1937, local anesthesia being used.

The operation was as follows

An incision was made through the conjunctiva from 10 to 8 o'clock over the insertions of the superior, lateral and inferior rectus muscles. The tendon of the superior rectus muscle was exposed generously and held with a squint hook. A fine double-armed silk suture was inserted into the lateral half of the tendon near its insertion, which was then severed close as possible to the globe. The lateral half of the muscle was separated with scissors for 15 or 20 mm posteriorly. The same procedure was followed in the case of the inferior rectus muscle.

The lateral rectus muscle was exposed, and a fixation forceps was applied 8 mm posterior to its insertion. The muscle was severed just anterior to the clamp. The stump was split equally up to its attachment. The divided ends of the stump were fixed to the lateral halves of the rectus muscles, respectively. The lateral rectus muscle was brought forward over its former attachment and advanced by means of two double-armed sutures. The conjunctiva was closed with a running suture, and both eyes were dressed.

Recovery was uneventful, and at the end of three weeks the patient was able to rotate the eye approximately 25 degrees laterally. Left hypertropia of 5 prism degrees and esotropia of 7 prism degrees were present. Recession of the medial rectus muscle for 2 mm was performed about one month after the first operation.

Examination of the patient on Jan 8, 1938, revealed a good cosmetic result with abduction of 43 degrees, as measured on the perimeter and confirmed on the tropometer. The fields of monocular fixation were almost the same for the two eyes. Examination with the orthoptoscope showed that esotropia of 4 degrees and left hypertropia of 6 degrees persisted without prism correction. When the patient wore his glasses, which contained both lateral and vertical prisms, he attained good third grade fusion and was able to diverge 7 degrees and to converge 26 degrees. The operations improved the patient cosmetically and functionally, but he still has annoying diplopia in the upper and lateral fields of gaze. Fortunately, he is able to read comfortably and experiences no difficulty when the eyes are held in the primary position.

CASE 2—Mr J. A. complained of sudden diplopia, which occurred in January 1935. His past medical history was essentially unimportant but his blood showed a strongly positive Wassermann reaction. Anti-syphilitic treatment was advised, and after two years of continuous treatment his blood remained Wassermann fast. The diplopia persisted.

On first examination the patient's vision was improved to normal with a +1.00 cylinder, axis 90 degrees, in each eye. He had esotropia of approximately 50 prism degrees for distance and for near vision. He was unable to abduct the left eye beyond the primary position. General physical and neurologic examinations gave essentially negative results. The fact that the patient demonstrated some lateral rotation of the eye and occasionally brought it to the primary position accounted for the selection of a simple resection and recession operation.

The operation consisted of recession of the medial rectus muscle for 4.5 mm and resection of the lateral rectus muscle of the left eye for 5 mm. The operation was performed at the New York Eye and Ear Infirmary on Aug 20, 1937. Recovery was uneventful, and diplopia disappeared after the first dressing. A series of orthoptic exercises

were prescribed, and on examination on Jan 8, 1938, it was found that the patient had third grade fusion with an amplitude of 5 degrees base in and 35 degrees base out. No evidence of diplopia was found in any direction of the gaze. The patient had esophoria of 2 prism degrees for the near vision with the screen test and of 8 prism degrees with the Maddox rod, but was comfortable.

The cases presented, hardly comparable in all details, suggest conservative approaches to the problem. Under favorable circumstances a simple operation should precede more complicated measures. If transplantation of the tendons of the vertical rectus muscles is indicated, a recession of the antagonist muscle should precede the transplantation.

DISCUSSION

DR WILLIS S KNIGHTON I understood from Dr Key that when he performed his operation in which he joined the vertical rectus muscles to the lateral rectus muscle, he split the vertical rectus muscles back as far as possible. I should like to ask Dr Payne if he did the same thing.

DR BRITTAIN F PAYNE The superior and inferior rectus muscles were split for 15 to 20 mm posteriorly.

DR JOHN H DUNNINGTON I should like to know what effect the transplantation alone had on increasing the abduction. As I understand it, the recession followed a month or two later, so I am anxious to know if the abduction was increased much before the recession.

DR BRITTAIN F PAYNE Abduction was increased about 15 degrees more with the recession.

INTRAOCULAR MASS DR BERNARD FREAD

Five years before the present examination the parents of E. P., aged 8 years, noticed a peculiar "reflection" from the child's left pupil. The left fundus showed a large yellowish white area about the disk and amorphous white exudates above and below, there were also some hemorrhages. A diagnosis of Coat's retinitis was made. Physical examination showed the tonsils to be slightly infected. The reactions to the Wassermann and the tuberculin tests were negative.

The patient was seen several times during the next few years. No change occurred in the fundus, except that the hemorrhage was absorbed.

Recently the parents noticed that the left pupil was yellowish. The patient had no perception of light. The pupil was dilated, and there was a large yellowish mass protruding far anteriorly into the vitreous. The mass was globular and slightly pigmented, and there were numerous blood vessels in it. On transillumination less light passed through in the nasal part of the eye. The intraocular pressure was slightly sub-normal. A roentgenogram of the eye did not show any calcium granules.

The diagnoses considered were retinoblastoma and the end-result of Coats' disease.

DISCUSSION

DR FREDERICK H VERHOEFF I think that it is a little risky to express a definite opinion about a condition like this when one has seen the eye only once and has not been able to study it as long as one

would like. However, it seems to me that this projection into the vitreous cannot be an inflammatory mass. It is not essentially an inflammatory mass, because the vitreous is so clear. The projection is so sharply defined that it makes one think that it is a detached portion of the retina. My final conclusion is that the original diagnosis was correct, that there was retinitis with massive exudation, and now there is separation of the retina, including the involved retinal area. This is a common outcome in cases of Coats' disease. I shall be much surprised if on microscopic examination this is not found to be true. In such cases, however, when the condition is of long standing, the eyes show certain changes which may make it impossible for one to tell even microscopically what the original condition was.

I advise that the eye be removed at once, because I do not think that any one can be certain that it does not contain a retinoblastoma, because the eye is blind and because I feel sure that it will ultimately be necessary to remove it, anyway. If microscopic examination does not reveal a retinoblastoma, it may reveal what the condition is.

DR SIGMUND A. AGATSTON. I agree with Dr. Verhoeff in the explanation of this case, the fact that the eye is soft excludes the possibility of a neoplasm. It seems to me that atrophy is developing. The first step is detachment of the retina. The chances are that the retina will become detached on the other side, and the typical picture of an atrophic globe will be present. There is no doubt that if the eye is left alone it will atrophy, and I think that it might just as well be removed.

ANOMALOUS PROJECTION AND OTHER VISUAL PHENOMENA ASSOCIATED WITH STRABISMUS. DR F. H. VERHOEFF

This paper was published in full in the May issue of the ARCHIVES page 663.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

ALEXANDER G. FEWELL, M.D., *Chairman*

February, 1938

WARREN S. REESE, M.D., *Clerk*

ASSOCIATION OF AN ANNULAR BAND OF PIGMENT ON POSTERIOR CAPSULE OF LENS WITH A KRUKENBERG SPINDLE. DR WILLIAM ZENTMAYER

This article appears on page ?? of this issue of the ARCHIVES

DELAYED REMOVAL OF MAGNETIC FOREIGN BODIES FROM THE VITREOUS DR CHARLES R. HEED

Accurate localization and early extraction of magnetic foreign bodies before the advent of degenerative changes, which result in an over-

whelming percentage of all cases in which an ultraconservative policy has been pursued, are important. A scleral incision is advocated when the metal is located back of the lens, and the introduction of any magnet tips into the vitreous is not advised. The reports of 3 cases follow.

CASE 1—D. B., examined five days after the accident, had vision in the right eye of 20/100. There was an incomplete coloboma at 11 o'clock due to operation by the company physician the day after the accident for removal of particles of torn iris. A small wound of the cornea, the iris and the periphery of the lens with a star-shaped opacity posteriorly were visible. Extraction with a magnet through a scleral incision was done on March 11, 1937. Two months later vision was 20/20, and the eye was quiet. Ophthalmoscopic and slit lamp examinations exhibited less density of the opacity.

CASE 2—B. R. was examined on June 23, 1937, one hundred and twelve days after the accident. The pupil was dilated, and the iris was copper colored. A diagnosis of retinochoroiditis with siderosis was made. After localization, a metallic body, 1.5 mm in size, was extracted with a magnet through a scleral incision. At the patient's last visit, on July 20, vision equaled 20/30. No deposits were seen on the capsule, and a few opacities of the vitreous were present.

CASE 3—T. J. A., examined six days after injury, had vision, with correction for myopia, of 20/20 (partly) in the right eye and 20/200 in the left eye. A particle of metal from an iron pin or hammer had entered the left eye through the upper lid. A roentgenogram revealed metal in the vitreous. Vision became blurred in forty-eight hours after the injury, and severe pain developed three days later. There was a delicate haze in the vitreous, the disk became somewhat blurred, and there was no apparent retinochoroidal lesion or hemorrhage. Slit lamp examination showed marked wrinkling and folds of Descemet's membrane, many cells in the aqueous, no apparent change in the lens and an increase of cells in the vitreous. A diagnosis of iridocyclitis was made. At the Jefferson Hospital roentgenographic examination disclosed a spherical metallic foreign body, 2 mm in diameter, in close proximity to the ora serrata, 4 mm to the nasal side of the median line. Extraction with a magnet was done through a scleral incision, to the nasal side of the superior rectus muscle about 8 mm posterior to the limbus. There was no prolapse of the vitreous and no pigment. Severe pain was present for two hours. The patient slept well and was free from pain the following morning. He was discharged on Nov. 8, 1937, and examination showed vision of the left eye to be 20/50, with correction. Slit lamp examination revealed a few fine opacities of the vitreous. No hemorrhage was present, nor were there any visible retinochoroidal lesions. Descemet's membrane was clear and free from wrinkles, the capsule and lens were clear, and there were no cells in the aqueous. The report from the patient's physician two months later stated that vision equaled 20/20, with correction.

DISCUSSION

DR. H. MAXWELL LANGDON. Every one knows what happens to eyes in which foreign bodies have been allowed to remain. It is not known how long a foreign body may remain in the eye before a distur-

bance begins, but it is known that sooner or later it will occur and that frequently it involves the fellow eye. I am glad that Dr Heed urged that roentgenograms be made in every case in which a foreign body is suspected.

One patient, whom I saw about twelve years ago, had been to a clinic in one of the large hospitals, having felt something strike her eye after a needle broke from a machine on which she had been sewing. She suggested that a roentgenogram be made, but she was told there was no foreign body in her eye. Not satisfied with this, she went to a private physician, who also assured her that a roentgenogram was not necessary. By this time her employer had notified the insurance company, and she was sent to me. A roentgenogram showed a part of the needle, over $\frac{1}{2}$ inch (1.3 cm) long, in the eye. Fortunately, a magnet attracted the needle, and it came out through the posterior scleral wound. The result was satisfactory, and vision equaled $\frac{5}{9}$.

Another case which has always interested me was that of an employee in the shops of the Pennsylvania Railroad at Olean, N. Y. He felt something strike his eye while at work, and promptly a roentgenogram was made, which was said to be normal. The eye was free from inflammation, and the vision was normal, he was told that there was no foreign body in his eye. In a short time he returned saying that he had no pain and could see but that he was sure there was something in his eye, and the same procedure was repeated, again with negative results. He returned after an interval of time but was sent to Philadelphia. Dr Newcomet localized a very small foreign body, not as large as the head of a pin, which was easily removed with a magnet, and the patient returned to work. There never was any inflammation of the eye, and the vision had never been less than $\frac{6}{6}$.

Unless the foreign body is in the anterior chamber, I favor removal by the posterior route.

DR LEIGHTON F APPLEMAN. I agree with Dr Heed that foreign bodies should be removed as soon as possible after their presence within the eye has been established. It is not always possible to see a foreign body within the eye owing to hemorrhage, therefore a roentgenogram should always be made.

The amount of intraocular disturbance may vary considerably, depending on the position of the wound of entrance and the size and shape of the penetrating particle. Also the subsequent reaction will depend on whether infection has been introduced with the foreign body.

Clean magnetic bodies can often be removed through the wound of entrance, if this is not possible, an incision through the sclera may allow it to pass after the tip of the magnet is presented at the opening. Dr Heed is to be congratulated on the result obtained in the second case reported, in which the foreign body had been in the eye for two months. I emphasize the importance of making roentgenograms in all cases in which a history of injury is given and of removing the foreign particle as soon as possible.

DR WALTER I LILLIE. I am in accord with the procedure as to the time and method of removal of intraocular foreign bodies described by Dr Heed. In his 3 cases and in those just described by Dr Langdon the visual results were excellent. This does not always occur, even

though the foreign body is removed without incident within twenty-four hours. I removed an intraocular foreign body situated in the vitreous of the right eye of a young man. It had entered the vitreous through the lower nasal portion of the sclera, just posterior to the ciliary body. His convalescence was uneventful, and the eye is now normal except for a small area of healed choroiditis at the site of entrance and exit of the foreign body. His vision of $\frac{3}{60}$ can be improved to $\frac{6}{60}$ only with a + 50 sphere combined with a + 1.00 cylinder, axis 90. The visual field was interesting, as the patient could only see the 10 mm test object in the extreme opposite field from any avenue of approach. This, of course, does not conform with any type of change in the visual field of organic cause or with the visual field associated with amblyopia ex anopsia or hysteria. I am sure that the patient is a malingerer. Such a possibility should always be thought of in a case in which compensation is being sought.

LATE RESULTS FROM RESECTION OF THE CERVICAL PORTION OF THE
SYMPATHETIC TRUNK IN CASES OF RETINITIS PIGMENTOSA DR
E. B. SPAETH

A series of 7 cases were reviewed in which resection of the cervical portion of the sympathetic trunk had been done because of retinitis pigmentosa. Of these, 5 cases were discussed in detail. Five other cases of retinitis pigmentosa in which the patients were under observation for the same length of time but were not subjected to surgical intervention were also reviewed in an attempt to decide by comparison whether or not surgical treatment has any proper place in aborting the almost inevitable progressive degeneration which occurs in cases of retinitis pigmentosa. While no definite statement can or could be made, there are some indications present which seem to suggest that this form of therapy may be seriously considered in certain cases. These 10 cases, as presented, are to be again reviewed after two more years have elapsed.

DISCUSSION

DR WILLIAM ZENTMAYER. If the pathologic picture of pigmentary degeneration of the retina is what it is believed to be and the disease is of an abiotrophic nature, I should not expect more than a transient improvement in the vision from sympathectomy.

DR ALFRED COWAN. I should like to ask Dr. Spaeth whether he knows the result in those cases of retinitis pigmentosa in which Dr. Frazier operated at the University Hospital some years ago. Dr. Grant should know, because he worked with Dr. Frazier at that time.

DR WALTER I. LILLIE. Dr. Spaeth's observation of the appearance of either new vessels or dilatation of small arterioles near the disks after cervical sympathectomy is interesting. If the retinal arterioles have not undergone organic changes, dilatation occurs when a paralytic Horner's syndrome is produced. This has been observed in patients with Raynaud's disease after cervical sympathectomy, but the dilatation usually does not persist more than one year. As the changes are temporary, as mentioned by Dr. Zentmayer, the value of this surgical procedure for retinitis pigmentosa is questionable.

DR E B SPAETH The 2 patients operated on by Dr Frazier died These cases will be presented again after two years to determine further results and the fate of the new blood vessel formation on the nerve head

ALEXANDER G FEWELL, M D, *Chairman*

March 17, 1938

WARREN S REESE, M D *Clerk*

THE USE OF PAREDRIE IN CYCLOPLEGIA DR I S TASSMAN

Paredrine is a proprietary name for a sympathomimetic drug (4-hydroxy- α -phenyl- β -aminopropane, β -4-hydroxyphenylisopropylamine) the chemical structure of which is the same as that of benzedrine (α -phenyl- β -aminopropane, β -phenylisopropylamine) with the exception that it contains an added OH group Since paredrine is considered in general less toxic and less irritating to mucous membranes than benzedrine, although in other respects its action is generally the same, it was employed to produce cycloplegia in conjunction with the use of atropine sulfate and also with homatropine hydrobromide, similar to the method described by Beach and McAdams for use with benzedrine

After the instillation into the conjunctival sac of paredrine alone, the effects were observed with reference to the size of the pupil, the presence or absence of signs of any irritation of the eyes and the effect on the intraocular tension

The preparation employed was a 1 per cent aqueous solution of paredrine hydrobromide In 50 eyes which showed no signs of any previous inflammation and in which the reaction of the pupil was normal, the instillation of 1 drop of a 1 per cent solution of the paredrine hydrobromide produced dilatation of the pupil to 5 or 6 mm in forty minutes The maximum effect was noted in from fifty to sixty minutes This effect wore off in about three to four hours In no case was any sign of irritation of the eye noted

The intraocular tension of 25 patients was tested with the Schiotz tonometer both before and from forty-five to sixty minutes after the instillation of 1 or 2 drops of the solution of paredrine hydrobromide There was no increase in the tension in any case

The method employed in obtaining the cycloplegic effect was based on that described by Beach and McAdams After experimenting with solutions of different strengths, it was found that a 4 per cent solution of paredrine hydrobromide produced satisfactory results

In a series of patients ranging in age from 6 to 45 years, the results were studied after the instillation of atropine sulfate and paredrine hydrobromide in those patients below the age of 16 years and after the use of homatropine hydrobromide and paredrine hydrobromide in the older age group In the former, 1 drop of a 1 per cent solution

of atropine sulfate was instilled into each eye, while in the latter, 1 drop of a 2 per cent solution of homatropine hydrobromide was instilled. This was followed in three or four minutes by the instillation of 1 drop of a 1 per cent solution of paredrine hydrobromide. A second drop of the cycloplegic was instilled in some instances in order to insure the desired effect. There is no objection to the instillation of the second drop of either the cycloplegic or the solution of paredrine hydrobromide. The results obtained in producing cycloplegia in this way were compared with those obtained from the usual method of repeated instillations of a 1 per cent solution of atropine sulfate in the younger patients and a 2 per cent solution of homatropine hydrobromide in the older patients.

The cycloplegic effect was obtained in the average case in forty minutes, with the maximum effect present in sixty minutes. The visual acuity for distant and for near vision and the measurement of the accommodation both before and after instillations were recorded and compared by the two methods, and the results obtained in all respects were found to be uniformly the same.

Retinoscopic examination and subjective refraction revealed almost unvarying results from those obtained by the customary method of repeated instillations. The degree and axis of astigmatism could be determined with equal accuracy, and the results were uniformly alike by the two methods in all kinds and degrees of refractive errors. The cycloplegic effect from the use of homatropine hydrobromide and paredrine hydrobromide begins to wear off in from four to five hours and newsprint can be read in most instances in from seven to eight hours. Black glasses can be discarded on the following morning. Within eighteen hours all near work can be accomplished without difficulty, and the patient can resume the normal use of the eyes. After the instillations of atropine sulfate and paredrine hydrobromide the accommodation returns in from two to three days. Four days was the longest time the effect of the cycloplegia was noticed.

This method can be employed with satisfactory results in routine office practice especially, and it greatly reduces the loss of time to the patient by the shorter duration of the cycloplegic effect.

DISCUSSION

DR SIDNEY L. OLSHO. In my office I have heretofore employed ten instillations of homatropine hydrobromide at ten minute intervals. The frequency was to make certain of a thoroughly reliable cycloplegic effect. During the past several weeks, I have instead, much after the method recommended by Beach and McAdams, instilled into each eye at five minute intervals 1 drop of the following solutions: a 5 per cent solution of homatropine hydrobromide, a 1 per cent solution of benzedrine sulfate and a 5 per cent solution of homatropine hydrobromide. I have then waited one hour for the cycloplegic effect. To avoid having the drop squeezed out the patient looks down the upper lid is held up and the drop is allowed to flow over the eyeball from an insensitive point well above the cornea. Before the solution can be squeezed out, it has mixed with the tears. The second drop of homatropine hydrobromide might have been omitted but I was not making an experimental study but was employing the method for effect.

In most instances the patients were able to use the eyes for close work on the following day. Postmydriatic examinations are, in my opinion, advisable. I prefer ample time to elapse before prescribing glasses.

The cycloplegic effect seemed to me perfectly satisfactory with this newer procedure. The postmydriatic reduction from the total hyperopia corresponded to that made after thorough homatropinization. For those patients who were reexamined, the mydriatic and postmydriatic results seemed also to correspond to my expectations following use of homatropine hydrobromide.

VERTICAL PRISM VALUES IN COMMONLY USED BIFOCAL LENSES DR SIDNEY L. OLSHO

This article appears on page 95 of this issue of the ARCHIVES

OBLITERATION OF THE PERICORNEAL BLOOD VESSELS IN THE TREATMENT OF VARIOUS TYPES OF KERATITIS. FURTHER OBSERVATIONS DR TRYGVE GUNDERSEN

The literature regarding peritomy was reviewed. A carefully selected, but consecutive series of 36 cases of chronic corneal disease was studied. The condition in these cases had resisted the usual forms of treatment. The types of keratitis were grouped as follows: chronic dendritic keratitis, 6 cases; ocular rosacea, 7; tuberculous keratitis, 12; keratitis from trauma, 2; lipid interstitial keratitis, 2; trachoma, 5; and hypopyon ulcer, 1 case. The condition in 1 case was not classified.

Obliteration of the vessels was usually performed with a sharp needle attached to the diathermy unit. In a few instances cutting instruments were used.

Some patients have been followed for almost five years. The results obtained by the operation have been classified as follows: excellent, 12 cases; good, 9; fair, 9; and no improvement, 6.

Book Reviews

Text-Book of Ophthalmology. By Sir W Stewart Duke-Elder
Volume II Pp 2094, with 742 illustrations including 24 colored
plates Price, \$15 St Louis C V Mosby Company, 1938

An examination of Duke-Elder's long expected second volume affords English-speaking ophthalmologists new reasons for rejoicing. The fact that at last ophthalmologists are beginning to have a reference book which is in every sense of the word modern is some compensation for the fact that nothing comparable has been available in the past. A comparison of the two volumes which have appeared with older works makes it evident that they could not have been achieved by any one writing before the twentieth century or, indeed, before the second quarter of the century. Since no amount of revision can ever make an old book thoroughly modern, the advantage cannot be overestimated of having such a book prepared from the ground up at this time.

The qualifications of Duke-Elder for writing such a modern reference book are known to most ophthalmologists. On a basis of training and research in physiology and biochemistry, which resulted in his fundamental works on the aqueous and vitreous, the equipment of a clinical ophthalmologist has been built. If the first volume revealed especially his unusual training in basic sciences, the second shows equal training in the clinical field. The works of the past have been digested and put in their proper place with relation to the most recent achievements. The bibliographies which accompany the discussion of each subject are remarkably well selected and include important articles which appeared as late as 1937. In this volume, for example, the pathogenesis of nevi as discussed in the first volume is revised, in view of Masson's recent work indicating their origin from neuroectodermal elements in the sensory nerves.

In the first volume the anatomy, embryology, physiology and biochemistry of the eye were discussed, the second volume covers methods of examination, congenital anomalies, general pathology, bacteriology and immunology and diseases of the conjunctiva, cornea and sclera. This systematic arrangement necessitates frequent cross reference, since the anatomy, physiology and embryology of each portion of the eye are in different chapters. Buphthalmos, for example, is considered under congenital anomalies, while its treatment is reserved for full discussion in a later chapter on glaucoma. Heterochromia, melanosis retinae and lamellar cataract are also discussed as congenital anomalies. Such cross references are carefully indicated in footnotes, however.

The chapter on examination of the eye includes a consideration of such modern instruments as the Sander pupilloscope and the Friedenwald ophthalmoscope and the use of monochromatic light for examination of the fundus. Photography of the fundus is discussed, with recognition of its limitations with present equipment.

The chapter on congenital anomalies is exceedingly complete, the monumental work of Treacher Collins in this field being emphasized.

Many illustrations are taken from his work and that of other British workers in this field. It is impossible, in a short review, to do justice to the 200 pages of excellent material in this chapter.

In the chapter covering general pathologic and therapeutic considerations one is brought abreast with modern thought in heredity, disturbances of metabolism, nutrition and various types of degeneration. The section on senescence is of especial interest. A review of bacteriology is included, and it is in this section that the reviewer finds a few points deserving criticism. It is certainly not true, for example, that *Bacterium granulosis* is a variant of *Bacillus xerosis*, nor will many ophthalmologists agree that it is a cause of follicular conjunctivitis. It may be bacteriologically correct to call the bacillus of Morax and Axenfeld *Haemophilus lacunatus*, but its great differences from the other *Haemophili* should be emphasized. The importance of epithelial scrapings for early diagnosis is certainly deserving of discussion. The virus diseases are well discussed, especially herpes simplex and herpes zoster. The subject of diseases due to the inclusion bodies of Halberstadter and Prowazek is not, perhaps, made sufficiently clear. The future may justify the author's inclination to favor the theory of the production of trachoma by *Rickettsia*, but the fact that Thygeson has obtained the same results with trachoma as with inclusion blennorrhoea, indicating the etiologic importance of inclusion bodies in both diseases, should have been noted. A mistake in referring to a work of Day and myself is noted, the article having dealt with conjunctivitis due to the meningococcus and not to *Bacillus influenzae*.

In the chapters on the conjunctiva, cornea and sclera one is constantly surprised at the freshness with which the various subjects are treated. Here is no dry compilation of facts from the literature but a careful analysis of facts with the object of arriving at conclusions in agreement with modern conceptions of pathology. The sections on healing of corneal wounds, corneal precipitates and edema of the cornea are written with the interest of one who might have made each subject his hobby. The author is interested in allergy not only as the probable cause of phlyctenulosis and vernal conjunctivitis but as the cause of forms of inflammation, such as transient periodic episcleritis and rosacea keratitis, in which a sensitization to bacteria or then toxins seems likely.

The discussion of therapy is, as a rule, complete and practical. In the treatment of serpent ulcer the value of paracentesis is brought out but no mention is made of delimiting keratotomy. The procedure is mentioned, however, as of value in the treatment of Mooren's ulcer. One wonders on what experience the author's conviction is based that secondary glaucoma is the most important cause of failure in treating serpent ulcer.

The illustrations of the book are excellent, including 742 halftones and 24 colored plates. The histologic changes of the diseases considered are especially well illustrated.

On the whole, this is a work of which English-speaking ophthalmologists have reason to be proud. It is devoutly to be hoped that the author's energy may not have been completely dissipated by this effort, so that the projected third and fourth volumes, which will complete the work, may not be too long delayed.

SANFORD R. GIFFORD

Contribution From the Eye Clinic of the Odessa Medical Institute
in Honor of the Director, Prof. V. P. Filatov (Honorary Scientific
Worker of the U. S. S. R.), on His 60th Birthday. Pp 503
Odessa Odessa Medical Institute 1936

These neatly published papers of high scientific and clinical value (two parts) illustrate the many phases of investigation and the indefatigable work which has been done for the past eighteen years at the Odessa Eye Clinic by Filatov and his assistants

In the first part a review is given by Filatov on the clinical and surgical activities of the clinic and on the organization and the instructional work of the chair of ophthalmology his second article on medical ophthalmology is exceedingly interesting Seven articles in this part are devoted to the problem of glaucoma The Eye Clinic has 75 beds 15 of which are reserved for patients who are to undergo corneal transplantation A great deal of experimental work is being done at the clinic on corneal transplantation and on aniline dyes The clinical and surgical activities are devoted to glaucoma its early diagnosis and the follow-up study of the patients corneal transplantation, retinal detachments (121 operations were performed for detachment from 1929 to 1935) plastic operations Filatov's modification of Golovin's orbitosinal-extraorbital exenteration the control of trachoma (repeated expressions) treatment of blepharitis with brilliant green (tetraethyldiaminotriphenylmethane sulfate) and physical therapy of the eye Two hundred and forty papers were published from 1917 to 1936

The outstanding article on glaucoma is that by Kalfa on elastotometry or the measurement of intraocular tension of tonometers of various weights He deals with the history and the theory of tonometry elastotometry and the reaction of the eyeball at various ages and the regulation of the intraocular tension by the reflex of the choroid Kalfa advances a new theory of the etiology of glaucoma namely, the disturbance of the normal function of the choroidal reflex leads to the change of the elastotometric curve and to its wider amplitude particularly in the early stage of glaucoma The uveal reflex is the chief regulator of the intraocular tension Various factors such as high arterial pressure changes in the blood chemistry and an increase of secretion lead to changes in the neural vascular apparatus of the choroidal reflex it becomes inhibited and the tension cannot be regulated properly A number of curves of intraocular tension of normal and glaucomatous eyes tables diagrams, and histories of patients illustrate this serious and thorough article Kalfa considers Maklakov's tonometer one of the best on the market The other articles on glaucoma deal with various methods of diagnosis in the early stage of glaucoma and various surgical procedures for glaucoma adapted by the Odessa Eye Clinic

The second part is devoted mostly to keratoplasty Up to 1935 300 corneal transplantations were performed by Filatov in the clinic corneas of cadavers were used for the transplant in 95 of these cases Filatov's prophylactic spatula conjunctival flap corneal sutures and various trephines are described A number of the cases in which operation was successful (about 57 per cent) are illustrated by photographs The experimental work deals with the instrumental trauma of the transplant and with the tissue cultures of conserved and dried corneas and their

viability Several photomicrographs demonstrate the histologic structure of conserved corneas

An exhaustive bibliography on keratoplasty for from 1814 to 1935 is dedicated by Brodsky and Shmulian to Filatov It is arranged in chronologic and alphabetical order and is divided into decades Filatov recommends it to all "transplantatois of the cornea" Each article is followed by an abstract in the German language

OLGA SITCHEVSKA

The Patient and the Weather By William F Petersen, M D, Department of Pathology and Bacteriology, University of Illinois College of Medicine, with the assistance of Margaret E Milliken, S M, Volume IV, Part 3 Organic Disease, Surgical Problems Price \$10 Pp 651 Ann Arbor, Mich Edwards Brothers, Inc, 1938

In the preceding volumes of this exhaustive monograph the author presented disease-dysfunction-disintegration-organic alteration (pathologic change) as a problem from which the factor of environment is inseparable, in particular, that of meteorologic change If this is decisive or at least of influence in the precipitation of the actual clinical event, it must be even more significant for the underlying fault, the pathogenesis It is to be regretted that fundamental work in the relation of constitution and meteorologic environment to surgical conditions has been practically ignored in the English and the American literature In a chapter devoted to ophthalmologic episodes one learns that the attention of the oculist has naturally been centered wholly on the eye and that the background of constitution, of phase fluctuations in the general organic status and in its adaptation to the environment, has been disregarded A study of cases of intraocular hemorrhage, optic neuritis, chorioretinitis, aneurysm, retinal detachment and especially of glaucoma appear to Petersen to make evident a simple relation between the precipitation of acute vascular episodes in the eye and the meteorologic environment Polar or tropical fronts and pressor episodes may combine to produce pathologic changes in the eyes or at least to precipitate a clinical crisis The large volume contains many pertinent and more or less convincing case histories, each of which is accompanied by a complete climatologic and meteorologic graph The author is of the opinion that intraocular hemorrhage is associated with relatively high systolic and diastolic blood pressure and sympathicotonic accentuation (ARS phase), while thrombosis and/or glaucoma, either spontaneous or induced by mydriatics, occurs with the corrective or overcorrective (COD) phase when the current is sluggish, the blood pressure levels relatively low, capillaries and venules dilated and the endothelium "unduly permeable and adhesive"

PERCY FRIDENBERG

Diseases of the Nervous System in Infancy, Childhood and Adolescence

By Frank R Ford Price, \$8 50 Pp 953, with illustrations
Springfield, Ill Charles C Thomas, Publisher, 1937

This is a sound book on the neurology of the developing nervous system It has the orthodox first chapter on methods of examination This includes data on roentgenography and the newer technics of the injection of air, iodized poppyseed oil 40 per cent and colloidal thorium dioxide, with a critique of their limitations and dangers

The chapter on anatomy and physiology is concise and clear and stresses the developmental point of view, so important in a genuine comprehension of neurologic manifestations, particularly in the ripening nervous system of children.

The clinical descriptions follow under various etiologic classifications, the section on the prenatal heredofamilial group being naturally somewhat longer than in a textbook on the nervous system of the adult. The separate disease entities are detailed under each etiologic classification. The descriptions are brief and clear. They include a consideration of the etiologic factors, the pathologic picture, the symptoms and signs, the diagnosis, the prognosis and the treatment. The presentation of the last phase is particularly good. When there is a good therapeutic regimen, it is given in detail. When there is none, the book is not burdened with futile suggestions. One hundred and thirty-three case histories are included in the descriptions. These are well chosen and help to sharpen the clinical pictures. A useful bibliography follows each heading. There is no attempt made to cover the psychiatric disorders of childhood.

The book is obviously based on a wide personal experience and is no mere pastiche. It can be recommended as a reliable textbook covering the immense field of neurology as applied to the infant, the child and the adolescent.

S. C. BURCHELL

Primer Congreso argentino de oftalmología. Vol. 1. Sociedad argentina de oftalmología. Pp. 446. Buenos Aires. Imprenta Frascoli y Bindi, 1937.

The first Argentine Congress of Ophthalmology was held in Buenos Aires on Oct. 27-31, 1936. The Argentine Society of Ophthalmology has now issued a report of this congress, and the first volume is published. The committee consisted of Dr. Carlos S. Damel, president, and Dr. Gunther von Grolman, secretary. Delegates from all the countries of South America were present, and their addresses are published.

The official subjects of the congress were lacrimal passages, spring catarrh and roentgenography in ophthalmology.

The lacrimal passages were most completely treated by a number of writers, in 242 pages, from every point of view from embryology to treatment. The subject is well illustrated, and a complete bibliography is appended.

The subject of spring catarrh was treated by Prof. Carlos Westkamp.

The papers on roentgenography in ophthalmology included a consideration of the use of this form of examination in the study of the orbit, intraocular foreign bodies, the optic foramen, the lacrimal passages, fractures of the skull, the nasal sinuses, cerebral tumors and the teeth. Good illustrations accompany the articles. There are in addition a number of interesting clinical reports.

ARNOLD KNAPP

A Textbook of Ophthalmology. By Sanford R. Gifford, M.A., M.D., F.A.C.S., Professor of Ophthalmology, Northwestern University Medical School, Chicago. Cloth. Price, \$4 net. Pp. 492, with 249 illustrations. Philadelphia. W. B. Saunders Company, 1938.

Among the problems encountered in writing a short textbook are the proper selection of material and its presentation in a well balanced

manner. It is self evident that nothing is more important than an adequate description of the methods of examination, as a knowledge of such methods combined with practical training forms the basis of all ophthalmologic experience. The selection of the rest of the material depends on what the writer has found to be of greatest importance to the beginner and to the general practitioner. These problems have been well solved by Dr. Gifford. The style of writing is clear and precise, and the descriptions are simple and adequate. The etiologic factors of the conditions described have been approached from a scientific standpoint, and the suggestions for treatment are practical and excellent. The author's personality appears on each page, so that the book is far from a mere digest. The makeup of the book is excellent. Some colored drawings have been used, and there is nothing which will so well depict a condition of the fundus. The illustrations in black and white are not as successful and some have lost clearness in the process of reproduction. This is a fault which can be easily corrected in a future edition.

The book can be recommended as an excellent introduction for the student in ophthalmology, who can be assured that the teachings imparted are sound and practical.

ARNOLD KNAPP

Studies on the Physiology of the Eye By J. Grandison Byrne. Price, 40 shillings. Pp. 440, with 52 figures and illustrations. Reissue with supplement. London: H. K. Lewis & Co., Ltd. 1938.

The first edition of this book appeared in 1933 and was reviewed in the ARCHIVES (10:721 [Nov.] 1933). The present edition is essentially the same except for a new index and the addition of a supplementary chapter entitled "The Effect of Stimulation of the Cortex Cerebri upon the Effector Mechanisms Which Mediate Movements of the Iris and Membrana Tympani" (reprinted from *J. Nerv. & Ment. Dis.* 85:528, 1937).

In my opinion there is no necessity for altering the criticisms and conclusions which were presented in some detail in the review of the original edition.

W. F. DUGGAN

Bulletin of the Ophthalmological Society of Egypt Vol. XXX. Thirty-Fourth Session 1937. Pp. 272. Cairo: Imprimerie Misr, S. A. E., 1937.

This bulletin consists of a review of the proceedings of the thirty-fourth session of the Ophthalmological Society of Egypt. The principal part of the program was a symposium on glaucoma, which treated the incidence, early diagnosis and surgical and medical treatment. In addition, there are well illustrated reports of clinical cases of interest. A few obituary notices and a report on the library concludes the volume.

ARNOLD KNAPP

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ARCHIVES OF OPHTHALMOLOGY

VOLUME 20 (old series Volume 77) AUGUST 1938

NUMBER 2

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DISTURBANCES OF THE VERTICAL MOTOR MUSCLES OF THE EYES

A BIELSCHOWSKY, M D

HANOVER, N H

I PHYSIOLOGIC ACTION OF THE VERTICAL MOTOR MUSCLES

The disturbances occurring in the sphere of action of the vertical motor muscles of the eyes present such numerous and manifold problems to the ophthalmologist and so often give rise to unexpected and unpleasant therapeutic failures that many ophthalmologists dislike to meddle with such conditions and refer the patients to other specialists from the very beginning. There are two reasons for this. While the function of the lateral muscles of the eyes is easily comprehensible under normal and abnormal conditions and the rules for diagnosis and appropriate treatment of their disturbances may be expressed in a few sentences, the correct understanding of even the normal function of the vertical motor muscles presupposes an accurate knowledge of their anatomic and especially of their physiologic characteristics, which can be appreciated only by some one who is thoroughly familiar with the fundamental laws of the movements of the eyes. Accordingly, pathologic conditions of the vertical motor muscles are much more complicated than anomalies of the lateral muscles. This is true especially in cases in which disturbances of heterogeneous origin coexist, producing symptoms which to one without adequate clinical experience seem contradictory and present the greatest difficulties in the analysis of the disturbance and may lead to wrong diagnosis and therapeutic failures.

At the present time four separate groups of disturbances of the vertical motor muscles are known, which are entirely different with reference to their origin. Before discussing these in detail, I shall summarize briefly the most important anatomicophysiologic conditions from which the normal functions of the vertical motor muscles and their disturbances are derived.

Each eye has at its disposal two pairs of vertical motor muscles. Each pair consists of a rectus muscle and an oblique muscle, which cooperate

From the Dartmouth Eye Institute, Dartmouth Medical School

Read at the meeting of the Western Ophthalmology Society, Los Angeles, Jan 26, 1938

both in supraversion and in infraversion. This cooperation brings about vertical movements of the eyes from the primary position without a simultaneous torsion, that is, without changing the position of the retinal meridians relative to each other. A torsional deviation takes place only in those extreme diagonal positions of the eyes which do not occur in ordinary vision because owing to compensatory movements of the head all extreme movements of the eyes are avoided. Moreover, the cooperation of a rectus muscle and an oblique elevator or depressor muscle makes it possible to maintain a certain amount of elevation and depression of the eyes in the entire field of fixation. For example, the superior rectus muscle, the muscle plane of which forms an angle of about 27 degrees with the visual line in the primary position, can effect the maximum of elevation only if the visual line is turned out by 27 degrees. If the visual line could be turned in by 63 degrees, it would be perpendicular to the muscle plane of the vertical rectus muscles and could not be elevated at all from this position, even by a maximal contraction of the superior rectus muscle. The only effect of the contraction in this case would be a torsion of the eye (inward rotation) about the visual line as axis. This holds good, analogously, for the depressing function of the inferior rectus muscle, the muscle plane of which is coincident with that of the superior rectus muscle. In other words, the vertical rectus muscles are preferably elevators and depressors of the abducted visual line, their participation in the vertical movement of the adducted eye being less the farther the visual line deviates from the muscle plane of the vertical rectus muscles. Approximately the reverse applies for the function of the oblique muscles. Since their muscle planes are vertical to the visual line when the eye is abducted by 40 degrees, the oblique muscles have absolutely no elevating or depressing effect in that position but rotate the eye solely about the visual line as axis. On the other hand, these muscles bring about purely a vertical movement when the visual line lies in their muscle plane, i. e., is adducted to the maximum (50 degrees).

Antagonistic torsional components of a rectus and of an oblique elevator (depressor) muscle compensate each other in such a way that an oblique position of the retinal meridians does not occur. Similarly, the diminution of the elevating or depressing component of one muscle during lateroversion is compensated by the accretion of the elevating or depressing function of the cooperating muscle in this very movement. This applies, *mutatis mutandis*, also to the increase and the decrease, respectively, of the torsional effect of those muscles in a change of the position of the eyes in the lateral direction. The action of the vertical motor muscles in the different visual directions is based on these anatomomechanical arrangements. However, the vertical motor muscles not only are connected with each other for the purpose of executing parallel vertical movements but are also engaged, though to a considerably

lesser extent, in other combinations, in which they are governed by other centers. The elevator muscles of one eye, together with the depressor muscles of the other eye, are innervated to move the eyes in opposite vertical directions to produce positive and negative vertical divergence, just as the lateral muscles of the eyes must bring about, besides dextroversion and levoversion, the convergence and the divergence movement. In a third combination the vertical motor muscles are governed by the vestibular apparatus, and effect, when the head is tilted to one shoulder, the parallel rotary movement of the eyes toward the opposite side about the visual lines as axes. This rotary movement of the eyes requires the cooperation of both the superior muscles of one eye and both the inferior muscles of the other eye. Rotation of the eyes toward the right, which takes place when the head is tilted toward the left shoulder, is performed by the inferior rectus and inferior oblique muscles of the right eye and by the superior rectus and superior oblique muscles of the left eye, because the first pair has an outward rotating component and the latter an inward rotating component. Since the aforementioned muscles, aside from their rotating component, are antagonists, the superior rectus muscle, for example, having an elevating and adducting action and the superior oblique muscle a depressing and adducting effect, there will occur merely a torsion of the eyes about the visual lines but no change in the position of the latter, provided both muscles are innervated simultaneously and are functioning normally. The use of this parallel rotary movement for diagnostic purposes in cases of pathologic involvement of the vertical motor muscles will be discussed later. In a fourth combination the vertical motor muscles effect a rotary movement in opposite directions, namely, a conclination (intorsion) or disclination (extorsion) of the vertical meridians. The innervation required for that rotation is brought about only through corresponding fusional stimuli, for example, that presented by two identical objects which are fused in the stereoscope or the haploscope and are rotated in opposite directions. The eyes follow this displacement within certain limits, so that the images will remain on corresponding retinal areas, and binocular single vision will thus be maintained. A fifth kind of innervation of the elevator muscles must be mentioned, because it is important for diagnostic purposes. The impulse to close the lids provokes a simultaneous upward movement of the eyes (Bell's phenomenon). There are cases of oculomotor disturbances in which the eyes are not only incapable of spontaneous elevation but fail completely to react to vestibular and fusion innervations, and only the prompt execution of the elevation together with closure of the lids will show that the nuclei as well as the nerves of the elevator muscles are absolutely intact but severed from the supranuclear pathways, with the single exception of the pathway for Bell's phenomenon.

In the examination of persons with oculomotor disturbances the following fundamental laws of the movements of the eyes must always be considered

1 All innervations which take place during the normal visual act, the spontaneous as well as the fusion innervations and those reflex-like innervations due to optic, acoustic and tactile excitations, and finally also the true reflex innervations of vestibular origin, always flow equally to those groups of muscles of both eyes which are associated in the execution of certain movements, the innervation causes at the same time the contraction of the agonists and the relaxation of the antagonists

2 From this it follows that it is impossible to innervate one muscle or one eye alone or to send a stronger innervation to one eye than to the other. All the muscles of both eyes always participate in each movement. One half experiences an increase of tonus and the other half a decrease

This fundamental law of ocular movements, established and formulated by E. Hering, has often been misunderstood and its strict validity doubted because equal innervation is not identical with equal movement of the eyes. If when looking at a distant object in the median plane one's attention is attracted by a near object located in the right visual line, only the left eye will make an adduction movement, and the right visual line will maintain its position unchanged. Although the movement is unilateral, the bilateral equal innervation of the eyes in such cases can be demonstrated unequivocally. Not only is there a symmetric increase of accommodation and a symmetric contraction of both pupils, but both eyes undergo the slight torsion about the visual lines as axes which is always connected with the fixation of a near object. Moreover, at the instant of the fixation of the near object it will show an apparent movement toward the right, indicating that the "double" eye has now received the impulse for dextroversion, which cannot become manifest in the right eye merely because in this eye the effect of the impulse for dextroversion is compensated by the simultaneous convergence impulse, whereas both impulses induce the left eye to move in the same direction, i. e., to the right. Against the validity of Hering's law cases have been reported in the literature in which the patients were able voluntarily to turn one eye alone either in the horizontal or in the vertical direction. I have checked up on a number of such cases and have not found any in which the unilateral movement could not be attributed to bilateral equal innervations analogous to the previously mentioned unilateral adduction movement. Voluntary unilateral divergence or vertical movements may be performed under the following conditions. There must be a horizontal or a vertical divergence, respectively, as the position of rest of the eyes, which can be kept latent by a well developed fusion mechanism. If such persons relax the

innervation correcting the anomalous position of rest, one eye will move outward and upward or downward, respectively, while the other eye remains focused on the fixation object. But if the patient will now direct his attention to the double images which have become apparent, the fusion tendency will induce the deviated eye to return to binocular fixation so that the double images will be fused. Some time ago I checked up on an especially instructive case of arbitrary unilateral vertical movements. I was able to show that the patient had an old—possibly a congenital—paresis of the trochlear nerve. He had learned to let the paralyzed eye move upward and back again to the horizontal plane, but the latter movement was accomplished only when the compulsion to fusion could become effective. If the eye in question was covered or the patient's head was tilted toward the paralyzed side, whereby a maximum deviation occurred, the squinting eye could not be brought back from its deviation. On the other hand, the patient could not turn the eye from the normal position to an upward deviation when his head was tilted toward the nonparalyzed side, thereby completely disburdening the paretic muscle. Therefore, the only voluntary factors in such cases are the disregarding as well as the cognizance of the impressions which are transmitted by one eye while the fixation with the other eye is maintained. The fusion mechanism takes care of everything else automatically.

Formerly, many a wrong diagnosis was made because in the examination of persons with disturbances of motility decisive importance was attributed to the functions of the individual muscles as they were derived from their topographicoanatomic conditions, one did not take into consideration the fact that a single muscle never executes an ocular movement separately, even if it had the power to perform the intended movement. On the basis of the fact that the isolated action of the inferior rectus muscle may bring about depression, adduction and torsion, it was assumed that there was paresis of this muscle if one eye could not move downward and inward. This assumption did not take into consideration the fact that the depressing function of the inferior rectus muscle becomes increasingly less the more the visual line is turned in. To turn the eye down and in, three muscles cooperate as agonists: the two depressor muscles and the internal rectus muscle, the principal part of the two depressor muscles being played by the superior oblique muscle, the depressing component of which grows with the increasing adduction of the visual line. A considerable restriction of the field of fixation inward and downward, therefore, points not to weakness of the inferior rectus muscle but to weakness of the superior oblique muscle. This restriction is a regular characteristic of paresis of the trochlear nerve.

II DISTURBANCES OF THE VERTICAL MOTOR MUSCLES

The disturbances of the vertical motor muscles may be separated into the following five groups (1) pure concomitant vertical deviations, (2) vertical deviations of parietic origin, (3) deviations that show the features of unilateral or bilateral overfunction of the inferior oblique muscle, (4) dissociated vertical deviations and (5) vertical deviations showing the combined characteristics of several of the other groups

CONCOMITANT VERTICAL DIVERGENCE

The first group comprises the vertical deviations showing the characteristics of pure concomitant squint. Above all, the angle of squint is independent of the direction of gaze and of the transition of the fixation from one eye to the other. The diagnosis is relatively simple if one has to deal with a permanent deviation. In many cases of this type the deviation is composed of a horizontal and a vertical component, these must be examined separately in order to determine whether the vertical component is not perhaps based on an atypical secondary effect of the horizontal muscles or whether it presents the characteristics of one or more of the other forms of vertical deviation. This may be established by comparative measurement of the angle of squint in the primary position as well as in lateroversion, supraversion and infraversion and finally in diagonal positions of the eyes. This is comparatively easy if double images can be brought into the consciousness of the patient and are localized according to the normal (innate) retinal correspondence. In cases of anomalous retinal correspondence one has to make objective measurement by means of one of the methods which are not to be discussed here. In cases in which the visual acuity of each eye is fairly equal the double images must be studied by putting the colored glass first before one eye and then before the other eye. If the results show differences in the amount and type of deviation which recur in repetitions of the test and these differences are not due to paresis, a characteristic of which is a difference between the primary and the secondary angle of squint, then these differences point to a dissociated disturbance of the vertical motor muscles. But if repeated measurements and comparison of the results show approximately the same degrees of deviation in all directions of gaze, it is fairly certain that there is a purely concomitant disturbance. In such cases the double images display no obliquity, which would point to a parietic vertical deviation and would be different in the different parts of the field of fixation. There is likewise lacking the influence of the tilting of the head to either side on the amount of the vertical divergence, which is so characteristic in many cases of vertical deviations of parietic origin.

Considerably more difficult are the examination and analysis of the latent, or only periodically manifest, vertical deviations. In such cases only a series of comparative measurements of the angle of squint will disclose with any degree of certainty the nature of the deviation. The principal difficulty is due to the fact that in such cases the innervation maintained by the fusion mechanism and compensating the vertical divergence cannot be immediately and completely relaxed when fusion is suspended. The suspension of fusion by means of the Maddox rod or a dark red glass before one eye does not suffice for the complete relaxation of the compensatory innervation which maintains an increased tonus of the elevator muscles of one eye and of the depressor muscles of the other eye any more than placing convex glasses before the eyes of a youthful subject with hyperopia induces him to give up promptly and completely the increased tonus of the ciliary muscle. Not only is relaxation caused by the breaking up of the fusion innervation incomplete, but when the patient is tested at different times it is also inconstant, so that if the test is repeated several times under the same conditions the amount of deviation will be found to be greater at one time and smaller at another. The continuous fluctuations of the compensatory innervation may occasionally simulate a paretic disturbance, since different degrees of deviation are found in the different directions of gaze. Such an error may be prevented only by repeating the measurements several times, thus an entirely dissimilar behavior of the vertical divergence may frequently be found when the subject is changing the direction of gaze. In such cases a careful determination of the vertical fusional amplitude is a valuable aid in rendering a reliable diagnosis. Take, for example, the case of a positive vertical divergence, which is usually kept latent through a negative vertical divergence innervation that causes an increased tonus of the right depressor and the left elevator muscles. If prisms of increasing strength are placed base down before the right eye, or base up before the left eye, the positive vertical divergence amplitude will be found to be considerably greater than normal, because the active positive vertical divergence innervation is required to set in only when, after the relaxation of the compensatory (negative vertical divergence) innervation, the eyes have taken their position of rest. The negative vertical divergence amplitude, on the other hand, is considerably diminished or reduced to zero, because the innervation to the negative vertical divergence must first overcome the positive vertical divergence, that is, the patient's position of rest, before it can bring the eyes into the opposite (negative) vertical divergence. With a series of tests which effect a positive vertical divergence, it is possible, as a rule, to bring about a complete, or at least a more extensive relaxation, of the compensating innervation, than by merely suspending fusion. Such examinations, of course, require time, patience and an adequate method of procedure (either a phorometer or a double

prism apparatus, a haploscope or similar apparatus) Despite every precaution and frequent repetition of the test, it is not always possible to distinguish whether one has to deal with a pure hyperphoria, i e., an anomalous position of rest, based on anatomicomechanical factors, or whether the vertical divergence is due to abnormal innervations, which may become manifest temporarily in cases in which there is a normal position of rest as well as in those in which the anomaly of innervation is associated with a true hyperphoria After discussion of the remaining forms of vertical deviations, I shall cite examples of such disturbances which may cause serious diagnostic and therapeutic difficulties

PARETIC VERTICAL DIVERGENCE

Vertical deviations of paretic origin do not require detailed discussion here so far as typical cases are concerned Whether an elevator or a depressor muscle is paralyzed is easily determined if the raising or the lowering of the eyes increases or decreases the vertical divergence Whether a rectus or an oblique muscle is paralyzed will be decided by ascertaining whether the vertical divergence increases either in abduction or in adduction of the paretic eye and decreases in the opposite direction Obliquity of the double images, as will be demonstrated presently, does not play a decisive role in the diagnosis Of even less importance for the differential diagnosis is the lateral distance of the double images which may be present in addition to vertical divergence One must not expect that in every case of paralysis of the trochlear nerve, for instance, there must be an uncrossed diplopia in addition to the vertical diplopia, owing to the loss of the abducting component of the superior oblique muscle In 25 per cent of the cases of paralysis of the trochlear nerve either the double images show no lateral distance or they are crossed This is due to the great frequency of exophoria as a position of rest, by which when it becomes manifest after onset of the paresis the slight convergence resulting from the absence of the abducting component of the superior oblique muscle will be compensated or over-compensated Diagnostic difficulties may arise in a later phase of paretic vertical deviation, namely, at the time when the paretic muscle has wholly or partially regained its function, but the vertical deviation continues as a result of a secondary contracture of the antagonist of the paretic muscle The deviation then no longer shows the characteristic signs of its paretic origin but will become more and more similar to the concomitant type This happens rather frequently also in the atypical secondary phase of paresis of the abducens nerve, which may appear as concomitant convergent strabismus when neither a distinct motor defect nor an increase or decrease of esotropia can be ascertained in lateroversion Paresis of the vertical motor muscles in

the atypical secondary stage shows an analogous behavior so far as the vertical divergence does not increase or decrease when the eyes are raised or lowered, thus one will not know whether an elevator muscle of one eye or a depressor muscle of the other eye had been originally paretic. But one does find regularly in such cases a considerable difference of the vertical divergence in lateroversion, according to whether the deviation is caused by a disturbance of balance between the vertical rectus muscles or between the oblique muscles. Take, for example, a case of typical paresis of the right fourth nerve in which the paresis has subsided considerably, the positive vertical divergence, however, continues, because it is maintained by the secondary contracture of the right inferior oblique muscle. It changes neither in supraversion nor in infraversion but increases considerably when the patient is looking toward the left, so that the right eye is adducted and its vertical position now depends predominantly on the oblique muscles. In dextroversion the vertical divergence recedes considerably, because the oblique muscles have no influence on the vertical position of the right eye while it is abducted. An analogous behavior of positive vertical divergence in the different directions of gaze could also be caused by paresis of the left superior rectus muscle in a later atypical stage. In such a case the vertical divergence would also attain its maximum in levoversion, during which the left eye is abducted, and its minimum in dextroversion, in which the vertical position of the left eye is influenced only in a minor degree by the vertical rectus muscles. Nor would the obliquity of the double images furnish a reliable clue as to whether the right superior oblique or the left superior rectus muscle is the cause of the vertical deviation. The obliquity merely shows whether there is disclination or conclination (extorsion or intorsion) of the vertical meridians.

Since both of the muscles under discussion are inward rotators, paresis of each of them causes a pathologic disclination. I want to emphasize the fact that it is by no means always the image seen with the paretic eye which seems oblique to the patient, quite often he will see either both images slanting or even the image of the nonparetic eye obliquely and that of the paretic eye straight, the latter results if the paretic eye happens to be the dominant eye. In such cases vertical and horizontal contours, although imaged on oblique sections of the retina (oblique meridians) of the paretic eye, will be seen vertically and horizontally, but then the corresponding images, although lying on the vertical and the horizontal meridian of the other eye, respectively, must appear obliquely, according to the angle of disparity between these meridians and the oblique meridians bearing the same images in the paretic eye. There are two possibilities for deciding which is the paretic eye in such cases. One is the head-tilting test. If the head is tilted toward the right shoulder a vestibular innervation for a parallel rotary

movement of the eyes toward the left side will be elicited, the right eye will receive the innervation for an inward rotation and the left eye, for an outward rotation. If the balance of the right oblique muscles is disturbed, owing to paresis of the superior oblique muscle, the superior rectus muscle, when innervated together with the superior oblique muscle for an inward rotation will produce an upward deviation of the right eye, while the left eye, in which the two lower muscles (the inferior rectus and the inferior oblique muscles) cooperate in the outward rotation, the lowering component of one muscle being compensated by the elevating component of the other, will perform merely a rotary movement about the visual line as axis. When the head is tilted toward the left, the right superior oblique muscle does not become active, the result is a decrease or complete disappearance of the vertical deviation. Since the right superior oblique muscle must participate as an inward rotator in the rotary movement to the left and the left superior rectus muscle functions as an inward rotator to the right, the results of the head-tilting test are different according to whether the vertical divergence is due to the paresis of one or the other muscle.

For an exact investigation of the influence which the position of the head exerts in certain cases of vertical deviation, one may use a simple apparatus constructed on the principles of the Helmholtz *Visierzeichen* (fig 1). While the patient's head is fixed by taking the little plate at one end of the rod between his teeth, he looks at a horizontal black strip on a piece of white cardboard fixed to the other end of the rod 30 inches (75 cm) away. The rod is put through a short tube, so that when the patient tilts his head it rotates around the same axis and through the same angle as the head. This insures the maintenance of the direction of gaze during the tilting of the head, since the cardboard with the fixed strip keeps pace with the movement of the head in respect to both the amount and the direction.

Another method which may be used to determine whether the vertical deviation is due to paresis of the oblique depressor muscle of one eye or to the elevator rectus muscle of the other eye is as follows. When one looks in a dark room at a faintly glowing filament, viewed monocularly through a tube, it will as a rule appear vertical and horizontal, respectively, to the observer only when its image lies on the vertical and the horizontal meridian, respectively, of the retina concerned. In paresis of a muscle with a rotating component, these meridians are positioned obliquely, consequently the glowing filament must be given a correspondingly oblique position in order to appear vertical or horizontal to the paretic eye of the patient. If the two eyes, which have been tested consecutively, show essential differences in the localization of the vertical and the horizontal meridian, one must derive therefrom to which eye the paretic elevator or depressor muscle belongs.

OVERFUNCTION OF THE INFERIOR OBLIQUE MUSCLES

The third group of disturbances of the vertical motor muscles is closely related to the atypical vertical deviations of parietic origin just discussed but is nevertheless distinguished from the latter by certain peculiarities. In its purest and simplest form, this type of disturbance presents the following characteristics. When the patient is looking straight ahead the visual lines are parallel, and even when the compul-

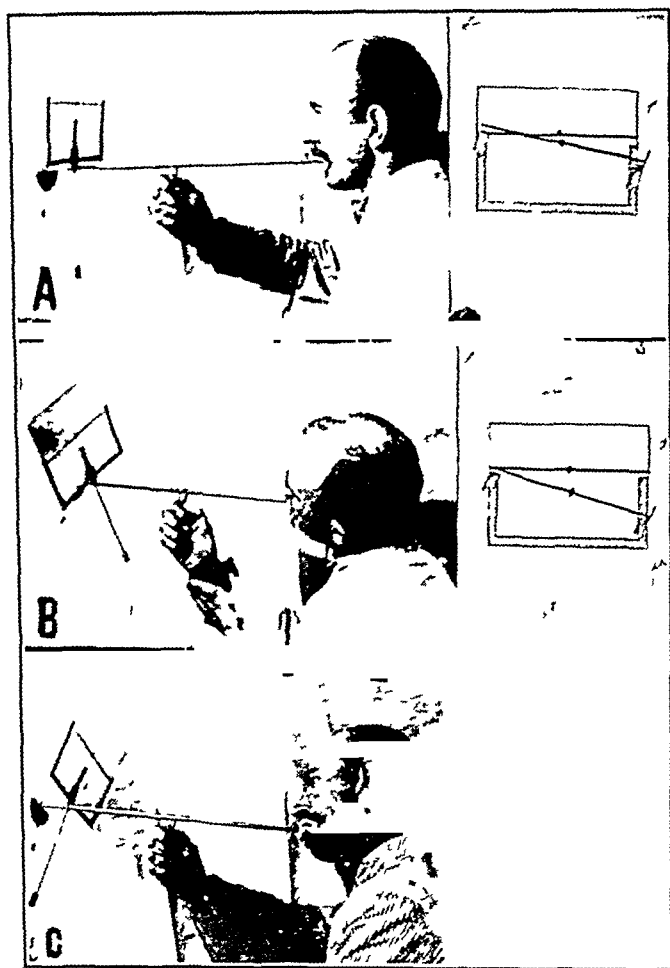


Fig 1—The head-tilting test as demonstrated by patients with palsy of the left trochlear nerve. *A*, two images of the fixed black strip are seen (reproduced on the right side of the photograph) while the patient's head is erect. In *B*, the vertical distance between the two images is increased by tilting the head toward the left shoulder. In *C*, the tilting of the head toward the right shoulder helps to secure binocular single vision.

sion to fusion is eliminated they show no, or only unimportant, latent disturbances of balance. But if the eyes follow an object moving laterally in the horizontal plane, the adverting visual line will deviate upward, occasionally by a considerable amount (fig 2). If the disturbance affects both eyes, the left eye will deviate upward in dextroversion, and the right eye will deviate upward in levoversion. When the fields of

fixation of either eye are tested, it will be seen that each field is considerably enlarged in the upper nasal quadrant without being restricted in the upper temporal quadrant. The limits of the lower halves of the field of fixation are either normal or slightly extended in the lower temporal quadrant. The behavior of the double images corresponds to the objective findings. Then obliquity points to a disclination of the vertical meridians, it is absent in some cases of congenital origin. The head-tilting test sometimes gives negative results, occasionally, however, in cases in which the deviation is manifest only when the patient is looking toward one side the results are positive, since the test shows a disturbance analogous to the atypical secondary stage of paresis of the trochlear nerve discussed previously. Frequently such an upward squint of the adducted eye during lateroversion will be found combined

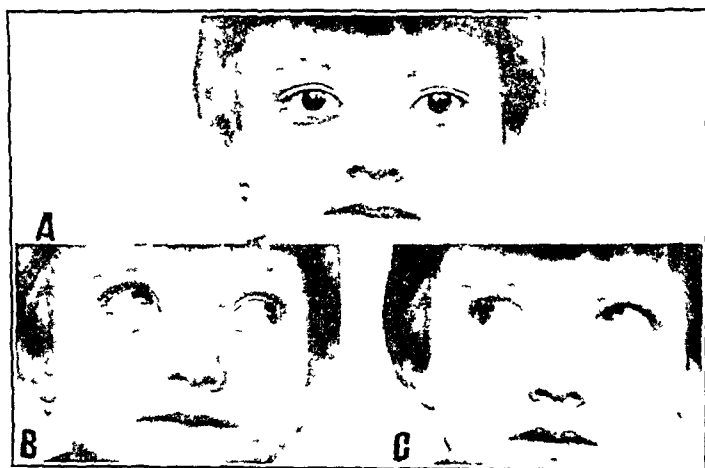


Fig 2—*A*, patient with binocular fixation in the primary position of the eyes. In levoversion the right eye deviates upward, as in *B*. In dextroversion the left eye deviates upward, as in *C*.

with either convergent or divergent strabismus, in the primary position of the eyes no vertical deviation, or only a minor one, is to be found in addition to the horizontal deviation (figs 3 and 4).

The anomaly of the vertical motor muscles just discussed is either unilateral or bilateral. It shows the features of an overfunction of the inferior oblique muscles, because of its far reaching conformity to the atypical secondary phase of paresis of the trochlear nerve, in which only the overfunction of the inferior oblique muscle, owing to a secondary contracture, may cause the deviation. But a primary overfunction of the inferior oblique muscle certainly does occur. I have seen it arise after radical operations on the frontal sinus, although paresis of the superior oblique muscle is found much more frequently in these cases. If in such operations the trochlea is not brought back

to its right place through an exact suture of the periosteum but remains dislocated, the function of the oblique muscles must be disturbed, at least temporarily. Usually there is a weakening of the function of the superior oblique muscle, but occasionally the function of the inferior oblique muscle is increased even without the weakening of its antagonist. This is explained by the fact that the action of the inferior oblique muscle is checked by a fascial ligament which is inserted in the trochlea. Its influence would be either lost or reduced if the trochlea were dislocated or the ligament itself were injured. C. H. Sattler also reported, after me, on the primary overfunction of the inferior oblique muscle after operations on the frontal sinus. In such cases the genesis and nature of the anomaly are vouched for. While the analogy of the



Fig 3—*A*, a patient with convergent strabismus of the right eye. The left eye is looking straight ahead. In dextroversion the left eye goes up while the right visual line remains in the horizontal plane, as in *B*. In levoversion the right eye goes up while the left eye is turned out, as in *C*.

symptoms in the much more numerous cases in which the anomaly is function of the inferior oblique muscle, there are no reliable data concerning the genesis, and especially concerning the fact whether it is a primary or a secondary disturbance, i. e., whether or not it developed in the course of a paresis of the trochlear nerve which existed originally but was cured. It might also be conceivable that the upward movement occurring simultaneously with the adduction of the right eye in a lateroversion impulse may be due to an asymmetric (oblique) insertion of the internal rectus muscle, as was mentioned by Cords on the strength of several cases in which he operated. In the numerous cases of apparently congenital overfunction of the inferior oblique muscle in which

the anomaly of the muscle was accompanied by convergent strabismus, I have found only occasionally a slight asymmetry of the insertion of the internal rectus muscle, so that it could not be held responsible for the extreme upward movement of the eye during the inward movement

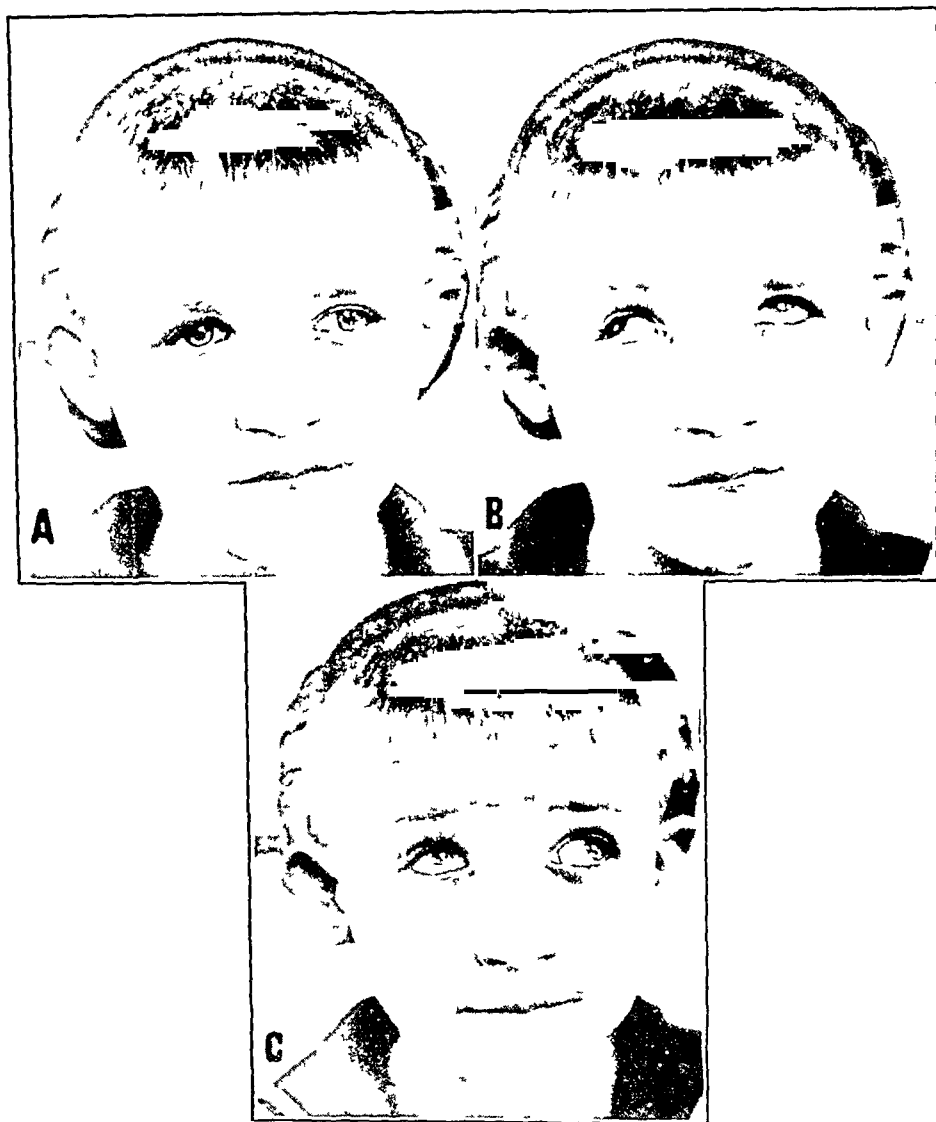


Fig 4—*A*, patient with divergent strabismus of the left eye. The right eye is looking straight ahead. In dextroversion the left eye goes up while the right visual line remains in the horizontal plane, as in *B*. In levoersion the right eye goes up while the left visual line remains in the horizontal plane, as in *C*.

In some cases the absence of certain characteristics which are never lacking in a true overfunction of the inferior oblique muscle argues in favor of the possibility that the type of overfunction of the inferior oblique muscle noted may be caused by other factors. That is to say, while there is always a meridional disclination in cases in which the

balance of the oblique muscles is disturbed by overfunction of one muscle or a weakness of the other, the disclination is surely absent in some cases of apparently primary overfunction of the inferior oblique muscle and there is likewise lacking the influence of the head tilting on the amount of the vertical divergence. Moreover, the position of the eyes when the subject is looking straight ahead is often perfectly normal in cases of primary overfunction of the inferior oblique muscle, this is never found in cases of secondary contracture of the inferior oblique muscle in which the contracture was preceded by paralysis of the antagonist. Aside from the few instances in which an oblique insertion of the internal rectus muscle may lack the aforementioned characteristics of a secondary anomaly, it is not yet known what is at the bottom of the primary overfunction of the inferior oblique muscle. On the other hand, there are many cases of apparently primary overfunction which bear all the essential characteristics of the aforementioned overfunction. This applies particularly to the congenital disturbances of this type, i. e., those already observed in earliest childhood and often brought to the attention of the physician because of the habitual torticollis existing since the second or the third year of the child's life. Of course in such cases the secondary, i. e., the postparetic, nature of the muscular disturbance cannot be excluded.

For therapy it is fairly unimportant whether overfunction is primary or secondary. What is important is merely the proof that one has to deal with such an overfunction, for example, of the right inferior oblique muscle, and not with a paretic weakness of the left superior rectus muscle. The latter is often assumed erroneously in cases of this kind, because when one looks up and to the left the left eye lags behind the right. If, as is often the case, the right eye is the dominant eye, its motility is decisive for the innervation of the two eyes. And when the function of its inferior oblique muscle is increased abnormally, the right eye will require a much smaller innervation to reach the limits of the upper nasal quadrant of the field of fixation than is required by the left eye with the normal superior rectus muscle in order to reach the normal limit of the upper temporal quadrant with a parallel movement. But if the fields of fixation of the individual eyes are examined separately, one eye will show no constriction of the field outward and upward, but there will be considerable extension of the field of the other eye when one looks up and inward. Moreover, in the majority of cases under discussion the increase of the vertical divergence when the head is tilted toward one side and the decrease of the vertical divergence when it is tilted toward the other side are decisive for the diagnosis of a disturbance of balance among the oblique muscles. The decision is important for the therapeutic procedure. The weakening of the excessively strong inferior oblique muscle through myectomy is considerably simpler and

more amenable to treatment than the strengthening of the function of the superior rectus muscle of the other eye by advancement or resection, which, in addition, has the disadvantage that as a rule it leaves the patient with a cosmetically disfiguring enophthalmos and slight ptosis. I have obtained most gratifying results in many cases with myectomy of the inferior oblique muscle. After binocular vision had been made possible with the head in the normal position, the habitual torticollis eventually disappeared, as did also the disfiguring upward squint when the eyes looked sideways. When the vertical divergence was associated with a considerable divergent or convergent strabismus, I found it expedient to remove first the lateral deviating component, after which, occasionally, a further operation became unnecessary. If the vertical divergence is also present in the lower half of the field of fixation, myectomy of the inferior oblique muscle may occasionally not remove it in this field, although it will remove the vertical divergence in the horizontal and the upper field. In such cases the muscular balance of the eyes can be restored by careful retroplacement of the inferior rectus muscle of the other eye, on the strength of the same deliberations which are decisive for using this operation in cases of inveterate palsy of the trochlear nerve.

DISSOCIATED VERTICAL DIVERGENCE

The most interesting disturbances of the vertical motor muscles, because their genesis and symptomatology are so fundamentally different from those of the disturbances so far discussed, are the so-called dissociated vertical deviations. Their peculiarities are most marked in cases of alternating or double hyperphoria, as is shown by the patient in figure 5. Ordinarily he had binocular fixation. Only occasionally, when he was tired or when he gazed abstractedly ahead, one or the other eye went up. With the diplopia test the image of the right eye at one time and that of the left eye at another time would be seen below the other one, according to whether the red glass was held before the right eye or the left. The characteristic difference between such a vertical deviation and those discussed previously is obvious. These deviations are either concomitant or of parietic origin or they are due to an overfunction of one of the vertical motor muscles. The joint feature of all these deviations is that when the eye which had been deviated upward was directed to the object which was first fixated by the other eye, the latter deviated downward. This alternation between upward and downward squint, which is caused by the transition of fixation from one eye to the other, is based on the law of the equal innervation of both eyes. To look at an object lying in the horizontal plane with the upward squinting eye, an infraversion impulse is called

for, which makes the eye which had first been in a horizontal position deviate downward. In contrast to this behavior, in the cases now under discussion, if the eye which is deviated upward is induced, by covering the other eye, to focus on the fixation object, the covered eye will first make a slight downward movement accompanying the parallel movement of the other eye, but immediately after the latter has become fixed an isolated upward movement of the covered eye will take place. Such anomalies were previously attributed to a mechanical superiority of the elevator muscles over the depressor muscles. This conception is erroneous. If the assumed superiority of the elevator muscles were the same in both eyes, as for example in cases of associated paresis of the depressor muscles, the eyes would be brought to a horizontal position

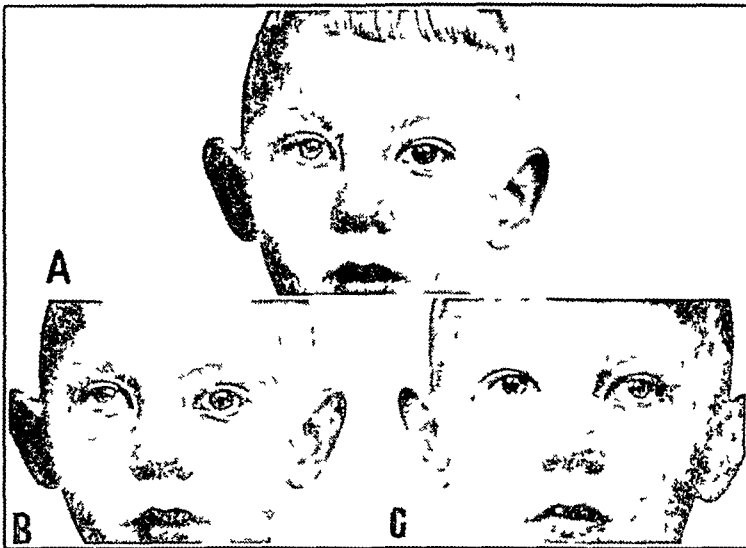


Fig 5—Patient with alternating hyperphoria. The visual lines either are parallel (A) or are deviated upward alternately (B and C).

by an increased infraversion impulse. An alternating upward deviation would not take place. And if the superiority of the elevator muscles were different on the two sides, for example, if the right depressor muscles were weaker than the left ones, the right eye with the weaker depressor muscles would be deviated upward, and if it were brought to the horizontal position by a depression impulse, the latter would bring the other eye into a downward deviation. It is easy to prove that the hypothesis of a superiority of the elevator muscle is untenable. If in a case of alternating hyperphoria, while the right eye is fixing a light located in the horizontal plane and the left eye being deviated upward, a darkening glass is held before the fixing eye, an isolated downward movement of the upward squinting eye will take place which will bring it down to, or even below, the horizontal position, while the fixing eye maintains its position unaltered. This one-sided vertical movement of

the deviated eye can be observed frequently, even without the darkening of the fixating eye. While this eye maintains constant fixation, the other eye, which is deviated upward behind a screen, will make at irregular intervals vertical movements of different extent, so that at one time a greater degree of upward deviation may be observed and at another time there may be a lesser degree of upward deviation or at times even a slight downward deviation. Such behavior, of course, cannot be reconciled with the assumption of an elastic superiority of the elevator muscles or of parietic disturbances among the elevator and depressor muscles. The alternating hyperphoria and the aforementioned one-sided vertical movements must be attributed to intermittent and alternating innervations for positive and negative vertical divergence, which are independent of the will and fluctuate in intensity. The ascertainment that in cases of alternating hyperphoria positive and negative vertical divergence movements may be elicited to more than an average extent by adequate fusion stimuli argues in favor of this assumption. It is known that normally not more than 3 or 4 prism diopters may be overcome by a vertical divergence innervation, furthermore, it is known that in concomitant hyperphoria the amplitude of either positive or negative vertical divergence is enlarged, whereas the opposite movement is either restricted or zero, according to whether it is a right or a left hyperphoria. In alternating hyperphoria, however, vertical prisms of 10 prism diopters and more in either direction may be overcome by corresponding vertical divergence innervations. This behavior refutes most impressively the hypothesis of a bilateral superiority of the elevator muscles over the depressor muscles as the basis of alternating hyperphoria. The increased amplitude of both positive and negative vertical divergence which manifests itself in the capacity to overcome unusually strong prisms in front of either eye, base down as well as base up, can be due only to the fact that with this test the one-sided vertical movements are brought about by positive and negative vertical divergence innervations. The movement is one-sided, because, as was discussed previously, an innervation to a parallel vertical movement caused by the fixation effort is combined with the fusion innervation which drives the two eyes in opposite directions, the latter compensating the effect of the former innervation in one eye while increasing it in the other eye.

Where these abnormal excitations originate is still entirely obscure. Only the following fact is known. The fusion mechanism, so long as its action is normal, keeps the tendency to alternating hyperphoria latent, just as it does in the cases of ordinary heterophoria. If, however, the fusion mechanism is weakened, for instance when fatigue sets in, or is lost entirely owing to blindness or amblyopia of one eye, the abnormal vertical divergence innervation becomes manifest and produces either alternating hyperphoria or in cases of unilateral amblyopia one-sided,

irregular upward and downward movements of the amblyopic eye. In cases in which there are no complications the direction of gaze has no influence on the anomaly under discussion.

This anomaly is often combined with concomitant squint (fig. 6). The squinting eye then shows in addition to the lateral deviation component an inconstant vertical deviation component, the extent and direction of which may be influenced, to a certain degree, by the occlusion of the squinting eye as well as by darkening the fixing eye by a colored glass. Such cases have been misinterpreted in various respects. It was believed that the isolated upward deviation of the squinting eye when it is covered and its isolated downward movement when it is uncovered had to be considered as a fusion movement, although the isolated movement of the squinting eye did not do away with the horizontal deviation. It was assumed that a binocular visual act existed on the basis of an

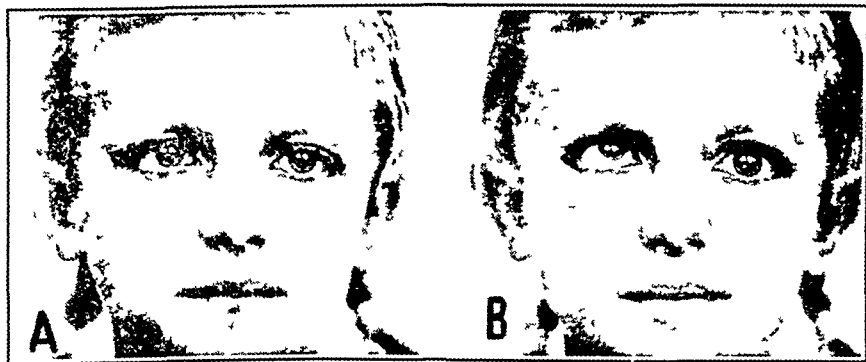


Fig. 6—*A*, patient with convergent strabismus of the right eye. This is the same patient shown in figure 3. In *B*, the right eye is turned up and in, the left eye remaining in its primary position.

anomalous retinal correspondence adapted to the horizontal deviation of the squinting eye and that, owing to a subsequent change of the anatomic position of rest, a vertical deviation had arisen in addition to the horizontal deviation, but had been kept latent during vision with both eyes. More thorough investigations refuted this assumption. It could be shown that the downward movement of the squinting eye took place also when the fixing eye was darkened by a colored glass, although the squinting eye remained covered. This, of course, precluded the influence of the binocular effect of a fusion innervation. One was forced to the same conclusion by the observation that even in cases of extreme amblyopia of the squinting eye which precluded the possibility of a fusion innervation, covering the squinting eye brought about or expedited its upward movement, uncovering the squinting eye or darkening the fixing eye caused the isolated downward movement of the former. Some authors interpreted the isolated downward movement of the squinting eye when the fixing eye was darkened

as a movement connected with the reaction of the pupils to light, due to an abnormal extension of the nervous excitation in the nuclear region. If that were actually the case, then the pupillary dilatation, occurring not only during the darkening of the fixing as well as the squinting eye but being greatest when both eyes are darkened, would have to bring about the one-sided downward movement. That never happens. The darkening of the amblyopic eye has the opposite effect of darkening the fixing eye. The former effects or expedites the isolated upward movement of the squinting eye and the latter effects or expedites the isolated downward movement, while a uniform darkening of both eyes, which causes a still greater dilatation of the pupils, is followed by no change whatever in the deviation of the squinting eye or only by those slight vertical movements which may also be observed when both eyes are uncovered. Moreover, I was able to prove that the one-sided vertical movements also take place when a restriction of the pupils, resulting from a stronger illumination, is brought about instead of a dilatation. In place of the covering screen I brought a mirror into the median plane of the head, rotating it about its vertical axis until it barred the fixated object from the amblyopic eye and at the same time exposing it to a stronger light by reflecting into the eye the light from a window. In spite of the bilateral pupillary constriction which this produced, the same upward movement of the squinting eye took place as when the squinting eye was covered by a screen, which brought about a pupillary dilatation. This, and other tests, made it likely that an abnormal excitability of the centers controlling the antagonistic vertical movements remains latent so long as both retinas receive homogeneous excitations, while the one-sided weakening or exclusion of the sensorial impressions makes the excitation of that vertical divergence center manifest, which causes the upward deviation of the eye concerned or the downward deviation of the other eye, according as the fixation impulse occurs in the one or the other. That the one-sided movements of amblyopic eyes are based on the same innervational anomaly as alternating hyperphoria is evident from the fact that when the amblyopic squinting eye is covered it goes upward, when it is uncovered, or the fixing eye is darkened, it goes downward—the same behavior as is observed in cases of alternating hyperphoria when one eye is covered or the other is darkened. The upward squint of the better eye in cases of unilateral squint cannot, of course, be demonstrated if the squinting eye has no central fixation.

If attention is paid to possible dissociated disturbances of the vertical motor muscles during examination of persons with heterophoria and strabismus, one will be surprised how frequently they occur combined with concomitant deviations. I have been able to ascertain such a combination in more than 41 per cent of all cases of congenital or acquired amblyopia of one eye and am convinced that the tendency to dissociated

vertical movements exists even in persons with binocular vision much more frequently than one would suppose, only, it is a great deal more difficult to demonstrate the disturbance so long as it is kept latent by the compulsion to fusion

The practical and clinical importance of the dissociated vertical deviations is, first of all, that they do not lend themselves to any optical or operative therapy such as would be considered in cases of concomitant or parietic deviations. I have seen cases of dissociated vertical deviations in which, owing to misinterpretation of their peculiarity, tenotomy of both superior rectus muscles had been performed. Thus the parallelism of the vertical meridians of the retinas, which in all probability existed previously, was transformed into a disclination, which expressed itself in that contours were seen in double images crossing each other at acute angles. The alternating hyperphoria which manifested itself temporarily and alternately continued and differed from the primary condition only so far as the vertical movements issued from a lower position of rest, and, although they did not extend so far upward, the downward extension was correspondingly greater. The only possible way of reducing or checking these deviations is through strengthening or relieving the fusion innervation. The stronger the fusion mechanism, the longer and the more easily it will keep the tendency to dissociated vertical movements latent. I have reported on the operative results in cases of divergent squint in which the squint was combined with vertical deviations of the dissociated type. After binocular single vision had been restored by correcting the lateral deviation, the formerly marked dissociated vertical movements disappeared completely and could no longer be elicited, even by a darkening of one eye. It goes without saying that in cases of unilateral amblyopia in which the amblyopia cannot be corrected, the dissociated anomaly continues even after the correction of the coexisting lateral deviation.

Alternating hyperphoria is much rarer than the combination of the dissociated anomaly with the other disturbances of muscular balance of parietic and nonparietic origin. Figure 7 shows a patient with paralytic divergence combined with dissociated vertical squint. The right eye is amblyopic and demonstrates alternately pure divergence, divergence and upward deviation and divergence and downward deviation. The vertical upward and downward deviations of the right eye are added at irregular intervals to the stationary divergence, but they may also be elicited by covering the right eye or darkening the left eye.

The complication of all kinds of squint with dissociated anomalies may, occasionally, render the analysis of the cases difficult. This applies especially to cases in which the dissociated disturbance is combined with a deviation of the concomitant or parietic type. Wrong diagnoses may be avoided by making systematic tests of the kind and amount of

the vertical deviation during fixation with the right and the left eye in all parts of the field of fixation. It may happen, for example, that in a case of paralysis of the right trochlear nerve it will be found that the right eye is considerably higher when the red glass is placed before the right eye. If it is placed before the left eye, there will be found either no vertical deviation or even a slight negative vertical divergence. I have seen such cases in which the diagnosis of paralysis of the

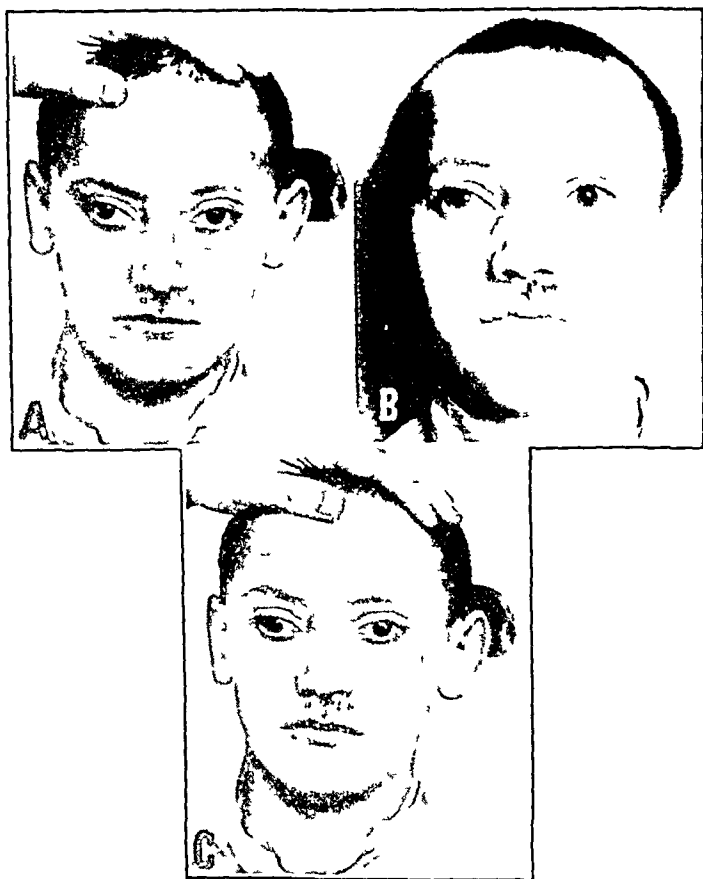


Fig 7—*A*, patient with divergent paralytic strabismus of the right eye. In *B* the right eye is deviated up and out. In *C*, the right eye is deviated down and out.

trochlear nerve was established unequivocally on the strength of other signs and in which the peculiar behavior of the double images when tested with the dark red glass became intelligible only after I had recognized the complication of the paralysis, with a dissociated disturbance of the vertical muscles. In the case of paresis of the right trochlear nerve, if the red glass is placed before the right eye the positive vertical divergence decreases because the dissociated component is added to the parietic component. But if the red glass is placed before the left eye, the parietic positive vertical divergence is

either partially or completely compensated or even overcompensated by the dissociated upward movement of the left eye, owing to the darkening of that eye

What I have been able to say here concerning the peculiar dissociated disturbances of the vertical motor muscles is, of course, merely an outline of their most important and, in the majority of typical cases, regularly recurring symptoms. For many details which in some cases render the picture even more diversified, I must refer to my article on the dissociated vertical movements (1931) ¹

III DIFFICULTIES IN DIFFERENTIAL DIAGNOSIS

In conclusion, I should like to make just a few remarks concerning the difficulties often encountered in separating the two groups of disturbances of the vertical motor muscles according to their different origin. One group comprises the concomitant deviations and those of parietic origin, including the cases in which there is an overfunction of the inferior oblique muscles. The second group comprises the deviations based on dissociated vertical divergence innervations. Their principal characteristic is the great instability of the deviation in contrast to the relatively great stability of the vertical divergence of the first group. But there are many exceptions even to the principal characteristic. In the discussion of concomitant hyperphoria I have already pointed out that the compensatory innervation maintained by the compulsion to fusion does not relax completely when the latter is suspended, but only partially, and in the individual investigations the relaxation is greater at one time and less at another. The concomitant nature of the deviation may be recognized and its spastic origin probably excluded by a careful measurement of the fusional amplitude, through which an extensive relaxation of the compensating innervation may be achieved. But the more thoroughly one examines the individual patients with motor disturbances and the more experience one gains in this field, the more one is convinced that frequently anomalies of the position of rest or parietic disturbances are complicated by abnormal excitations of the centers controlling the nonparallel movements. It is a well known fact that convergent strabismus represents only exceptionally a pure anomaly of the position of rest but is caused to a greater or lesser extent by an increased tonus of the convergence innervation. It is known how difficult it is to separate these two etiologic components and thus to create the reliable foundation for a rational therapy. Far less familiar is the combination of spasms of vertical divergence with deviations apparently based on an anomalous position of rest. The nonrecognition of the

¹ Bielschowsky, A. Die einseitigen und gegensinnigen ("dissoziierten") Vertikalbewegungen der Augen. *Arch. f. Ophth.* **125**: 493, 1931

neivous (spastic) component may cause entirely unexpected operative results if not complete failure

I should like to illustrate this with the following examples

CASE 1—A patient suffered for many years from severe headaches and, as he called it, "unsteady vision," which apparently was due to diplopia. I found a negative vertical divergence of 9 degrees and made a careful recession of the left superior rectus muscle, with a safe-guarding suture. Four days later there remained only a minimal overeffect in the form of a positive vertical divergence of 1 degree. In the course of the next day this increased to 9 degrees. I felt obliged to advance the retroplaced left superior rectus muscle. For eight days the result was excellent. Not a trace of the vertical divergence was demonstrable. Three days later the patient returned and complained of occasional diplopia, which he could overcome, but not without getting severe headaches. Examination showed a negative vertical divergence of almost the same amount as had been present before the beginning of the treatment. I sent the patient, who had a well developed neuropathic constitution, to the mountains for four weeks. When he came home he had no further complaints and no vertical divergence, nor did the vertical divergence return in later years. I shall postpone the epicrisis of this case until the other cases have been discussed.

CASE 2—A 27 year old engineer, intelligent, but with a severe neuropathic constitution complained of headaches and a most annoying feeling of sluggishness (dulness), which paralyzed his capacity to work. I found a greatly fluctuating positive vertical divergence. After relaxation of the compensating innervation, an apparently pure concomitant positive vertical divergence of 15 (arc) degrees became manifest. The recession of the right superior rectus muscle reduced the vertical divergence to 1 degree, but in the course of the next two weeks it increased again to approximately its former level. After a prolonged period of observation, during which there was no important change, I decided, on the strength of deliberations into which I need not enter at this time, to perform a recession of the left inferior rectus muscle. The immediate result was a slight overeffect (negative vertical divergence), which increased in about a week to 12 degrees. Since I had been anxious to graduate the effect of the recession carefully, I could not convince myself that the negative vertical divergence was the result of an excessive weakening of the left inferior rectus muscle. My doubts proved to be justified when I had both eyes of the patient bandaged for several days and then examined him again. I found an almost complete vertical balance, the position of the eyes fluctuating between 1 degree of positive vertical divergence and 1 degree of negative vertical divergence. After the patient had his eyes uncovered during the day, he again showed 12 degrees of negative vertical divergence in the evening. Nevertheless, he was able to bring about at the double prism apparatus 7 degrees of positive vertical divergence without the least difficulty, which seems inconsistent with the assumption that the 12 degrees of negative vertical divergence which he had demonstrated formerly with fusion suspended had to be attributed to the weakening of the left inferior rectus muscle. I discharged him after giving him a weak prismatic spectacle and directions for fusion exercises. He felt well for several months, but finally returned, because his condition had become considerably worse. The negative vertical divergence amounted to 14 degrees and appeared to be of a purely concomitant nature. The advancement of the left inferior rectus muscle resulted in a positive vertical divergence of 10 degrees, which still continued ten days later—an unusually powerful effect of a simple advancement. Two weeks later a negative vertical divergence of 6 degrees was

again found, which, however, already changed during the examination and the constant exclusion of the fusion tendency to a positive vertical divergence of 2 degrees. In the further course the eyes always oscillated by several degrees about the normal position in one direction or another, finally, the diplopia disappeared, and the patient was free from complaints and has remained so.

An analogous behavior could be observed in a third case.

CASE 3—A 45 year old man complained of diplopia, which presented no parietic characteristics but an extensive instability. The maximum positive vertical divergence which could finally be determined amounted to 18 degrees. After recession of the right superior rectus muscle there was absolute balance of the vertical motor muscles for five days. On the sixth day there was a negative vertical divergence of 10 degrees, which did not increase during elevation of the eyes, as would be expected in a case of weakness of the right superior rectus muscle, and even decreased temporarily to zero when fusion was excluded permanently. I had the patient under observation for seven months and found, with the same method of examination, 25 degrees of negative vertical divergence at one time and none whatever at another time. However, the patient's discomfort finally induced me to make a careful advancement of the right superior rectus muscle. Within the first week after the operation the slight overeffect, which is customary in an advancement, was reduced to 2 degrees of positive vertical divergence. In the second week it rose to 18 degrees. On no other occasion have I ever found that the mechanical effect of a simple advancement accomplished a change of position of approximately 43 (arc) degrees, from 25 degrees of negative vertical divergence to 18 degrees of positive vertical divergence. The fact that this enormous effect developed only during the second week after the operation is just as extraordinary, because the effect of an advancement is always greatest directly after the operation and then decreases. Evidence that the excessive effect of the advancement was not due to mechanical causes, i. e., to the change in the mechanical conditions of the position of the eyes, could be adduced from the fact that during the examination of the vertical fusional amplitude an absolutely identical and abnormally high positive and negative fusional amplitude was found. The patient was able to overcome 16 prism diopters base up or base down before one eye as well as before the other. It may be noted that in a few months all his troubles disappeared without recourse to any further measures, and after five years he showed only a slight fraction of his original vertical divergence.

I believe that the behavior in these cases can be interpreted only in one manner, that is, that the vertical divergence was due, at least to a great extent, to spasms first of one and then of the other vertical divergence innervation, which I had not been able to ascertain before the treatment or which possibly developed only in the course of the therapeutic procedure. All 3 patients had the following signs in common. The high amount of postoperative deviation existed only when both eyes were used and disappeared when one or both eyes were bandaged which, of course, excluded all innervations produced by the fusion mechanism. Moreover, contrary to heterophorias of concomitant or parietic origin both antagonistic innervations were increased beyond the norm and, what is more, the increase was approximately identical. The marked neuropathic constitution of the patients further supports

the assumption of the spastic nature of the temporary increase of the vertical divergence, which cannot be explained by mechanical factors

I must confine myself here to this outline without entering into the details of the analysis. The foregoing observations warrant merely the issuance of the warning that in cases of vertical deviations one should always bear in mind the possible complications with spastic vertical divergence innervations and try to follow their signs in order to prevent unexpected and undesired operative results.

PERIMETRIC STUDIES IN SYPHILITIC OPTIC NEUROPATHIES

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A study of syphilitic optic neuropathies was undertaken in 1931 jointly by the division of syphilology of the medical clinic and the department of ophthalmology of the Johns Hopkins Hospital and University. This investigation included repeated special clinical and ophthalmologic studies on a large group of syphilitic patients over a period of years, repeated perimetric studies and determinations of light sense, and a comparison of the effects of various types of treatment on primary syphilitic atrophy of the optic nerve. There are available for correlation, therefore, accurate clinical records and data on the visual fields of a large number of syphilitic patients with disturbances of the optic neuro-pathways who were followed for years during the progression or regression of the disease. This paper is a report of the changes in the visual fields occurring in association with syphilitic optic neuropathies.

HISTORICAL SURVEY

PERIMETRIC FINDINGS IN CASES OF PRIMARY ATROPHY OF THE OPTIC NERVE

In 1903 Uhthoff¹ summarized the results of his own and other investigators' perimetric studies in cases of tabetic atrophy of the optic nerve. He concluded that, while there was no defect in the field absolutely typical of syphilitic atrophy of the optic nerve, two general types could be recognized. The first type was characterized by a more or less equal reduction of form and color sense and of peripheral and central acuity. The capacity to perceive red and green was lost early, the perception of blue and yellow was retained somewhat longer. This

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This work was supported in part by a grant from the John and Mary R. Markle Foundation.

1 Uhthoff, W. Die Augenveränderungen bei den Erkrankungen des Nervensystems. In Graefe, A. and Saemisch, E. T. Handbuch der gesamten Augenheilkunde, Leipzig: Wilhelm Engelmann, 1909, vol 9 pt 2, chap 22 p 192.

type of defect was considered by Uhthoff to be due to an involvement of the entire cross section of the nerve. In the second type the defective regions of the field were sharply delimited from normally functioning regions. The changes consisted in sector-shaped defects or marked concentric contraction with good visual acuity and color sense, indicating a complete sparing of the nerve fibers supplying the central portion of the retina. The sector-shaped defects might or might not be connected with the blindspot. Defects starting as central scotomas were included in this group. Central scotoma occurred in only 2 per cent of cases in Uhthoff's own series and almost always in conjunction with a peripheral defect. Hemianopia, both homonymous and heteronymous, was reported. The homonymous type was believed by Uhthoff to be due to complicating factors other than tabes. He expressed the belief that apparent heteronymous hemianopia is not a true hemianopic defect due to a chiasmal lesion but merely a symmetric defect of the second type, it having been noted by many authors that the changes in the fields in the two eyes are often the same, starting, for example, in both temporal fields or in both upper fields.

Ronne² in his studies of the visual fields of a number of patients with tabetic atrophy of the optic nerve, found frequent nerve fiber bundle defects. He expressed the belief that the particular bundle of nerve fibers affected was more or less a matter of chance. Frequently a nasal bundle was involved and a temporal field defect produced. The papillomacular bundle might be affected and central scotoma produced, or occasionally there might be involvement of a paracentral bundle, producing an arcuate field defect somewhat similar to that seen in association with glaucoma. He noted that the scotoma associated with tabetic atrophy of the optic nerve was usually broader than that associated with glaucoma, and only seldom were other bundles entirely unaffected. He explained the infrequent involvement of the central field as probably due to the greater supply of nerve fibers to this region. Ronne noted a certain tendency toward symmetry of the fields in the two eyes but stated that this was not striking. He did not find homonymous defects in cases of tabetic atrophy of the optic nerve. The fields might by chance resemble those of hemianopia, but careful tests with test objects of graded sizes showed the defects not to be true hemianopia. He pointed out that true hemianopia is characterized by a sharp defect

2 Ronne, H. Ruht die Optikusatrophie durch Tabes von einem Leiden der Ganglienzellen oder der Nervenfasern her? *Arch f Ophth* **72** 481, 1909, Gesichtsfeldstudien uber das Verhaltnis zwischen der peripheren Sehscharfe und dem Farbensinn, speziell die Bedeutung derselben fur die Prognose der Sehnerventrophie, *Klin Monatsbl f Augenh* **49** 154, 1911, Ein Fall von Sehnerventrophie bei Tabes mit einseitiger nasaler Hemianopsie, *ibid* **50** 452 1912

across the vertical meridian and by the same limits in this region for all sizes of test object. He expressed the belief that the occurrence of nasal steps and the absence of hemianopic defects argue for a lesion starting somewhere in the optic nerve and not in the chiasm, the tracts or the ganglion cells of the retina.

Fuchs³ reported some supposedly atypical field defects in cases of tabetic atrophy of the optic nerve. The total number of patients studied was not stated. He found no less than 30 cases of central scotoma and expressed the belief that the incidence of this condition is greater than the estimate of 2 per cent made by Uhthoff. Fuchs found central scotoma in cases of tabes to be nearly always bilateral. In the beginning it was central, but later it extended to the temporal side and became cecocentral. At first the scotoma was for color only, especially for red and for green. As it progressed, the peripheral field became contracted. Fuchs observed 6 cases of bitemporal hemianopia. He stated the belief that its bilateral occurrence could not be an accidental result of lesions located symmetrically in the two optic nerves, for he did not find binasal defects. He concluded, therefore, that in these 6 cases of bitemporal defect the lesion was located in the chiasm.

Langenbeck⁴ studied the visual fields in 130 cases of tabetic atrophy of the optic nerve. In 89 of these the changes were characteristic of Uhthoff's first type. There were reduced acuity, partial or complete loss of color sensation and peripheral contraction, not necessarily concentric. In many cases the changes in the fields for color indicated the future course of changes in the field for form. In 27 cases the fields corresponded to Uhthoff's second type. They showed sector-shaped defects or (in 7 cases) marked concentric contraction, with well preserved function in the remaining part of the field. In 14 cases there was central scotoma, which was in no way like that seen in cases of retinobulbar neuritis. In 2 cases there was a defect somewhat similar to the nasal steps described by Ronne, but its boundary did not exactly follow the horizontal meridian. There were many instances of symmetric defects in the two eyes but no instances of true hemianopic defects.

Staigardt⁵ reviewed the previous reports and emphasized the great diversity of defects in the fields in cases of tabetic atrophy of the optic nerve. He expressed the belief that, in addition to the various forms of peripheral contraction, two other types of defect, namely, central

3 Fuchs, E. The Field of Vision in Tabetic Atrophy of the Optic Nerve, *Tr. Am. Ophth. Soc.* **12** 718, 1911.

4 Langenbeck, K. Die Gesichtsfeldformen der tabischen Sehnervenatrophie, *Klin. Monatsbl. f. Augenh.* (pt. 2) **50** 148, 1912.

5 Staigardt. Ueber die Ursachen des Sehnervenschwundes bei der Tabes und der progressiven Paralyse, *Arch. f. Psychiat.* **51** 711, 1913.

scotoma and hemianopia, must be included. He stated that, although occasionally there were symmetric defects in the two eyes, in general this was not the case, he mentioned instances in which one eye was severely affected while the other was completely normal. He stated that he did not believe, therefore, that all the various reported cases of heteronymous hemianopia should be considered as instances of symmetric defects in the two eyes, or that homonymous defects could be attributed in all cases to a complicating cerebral or basal lesion.

Igersheimer⁶ reported observations which indicated that changes in the fields may precede pallor of the disks. In a number of cases in which the older perimetric methods were used, he found questionable early defects associated with normal or nearly normal nerve heads. He was able to make prolonged observation in only 1 of these cases, in which at the first examination there were complete atrophy of the optic nerve and total blindness in the left eye. The right disk showed only a doubtful pallor, but there were marked defects in the peripheral field. The central visual acuity and the central color vision were entirely normal. One year later the patient reported by letter that he was totally blind in this eye. Modern perimetric methods proved more fruitful. When a tangent screen and small test objects were used in the examination, many tabetic patients with normal fundi showed nerve fiber bundle defects extending from the blindspot. A majority of these defects diminished or disappeared after antisyphilitic treatment. In 1 case, however, the defect disappeared before any treatment was given.

The more recent articles contribute little further to the knowledge of field defects in cases of atrophy of the optic nerve. Paton⁷ reported perimetric studies in 8 cases to illustrate the variability in the defects. Arlt⁸ examined a group of 53 patients and found that 85 per cent had contracted fields for form and color and reduction in acuity, 9 per cent had sector-shaped defects and 6 per cent had central scotomas. John,⁹ in a study of the frequency of tabetic atrophy, gave incidental data on the frequency of central scotoma before and after the introduction of arsphenamine. In 1905 none of the 22 patients studied had central scotoma. In 1910 2 of 24 patients had central scotoma. In 1920 and in 1925, after the introduction of arsphenamine, central scotoma was found in 2 of 19 patients and in 4 of 18 patients, respectively. Thus, in the entire group of 83 patients there were 8, or about 10 per cent, with central scotoma.

6 Igersheimer, J. Syphilis und Auge, Berlin, Julius Springer, 1918.

7 Paton, L. Tabes and Optic Atrophy, Brit J Ophth 6 289, 1922.

8 Arlt, E. Behandelte und unbehandelte Fälle von tabischen Sehnevenatrophie Ztschr f arztl Fortbild 19 367 1922.

9 John, I. Ueber die Häufigkeit der tabischen Optikusatrophie und ueber die bisherigen Behandlungsmethoden, Ztschr f Augenh 69 283, 1929.

Goldberg¹⁰ in an attempt to determine the initial field changes in cases of tabes, made perimetric studies of 75 tabetic patients, most of whom had no subjective symptoms of visual failure. Eight patients showed field changes, bitemporal in 7 cases. These were interpreted by Goldberg as due to lesions in the neighborhood of the chiasm.

Rutherford¹¹ reported studies of the visual fields of 50 patients with neurosyphilis of different types. Atrophy of the optic nerve was present in 7 of the 100 eyes and papilledema in 2. The other eyes presumably showed normal fundi, although this was not stated in the paper. The studies of the visual fields were made with a tangent screen at a distance of 1200 mm and a 1 mm white test object (equivalent to approximately $\frac{1}{20}$ degree, or 3 minutes, in angular measure). The peripheral limits for this test object were recorded at intervals of 15 degrees around the circumference, and the blindspots were mapped. The author expressed the belief that the normal field for the 3 minute white test object should extend to 30 degrees in each of the four principal meridians. In 84 of the 100 eyes the limits of the field were less than this in one or more of the principal meridians. The blindspot was enlarged in 31 eyes. No statement was made as to the size of the blindspot considered normal for this test object. The central visual acuity ranged from 6/6 to 6/21—. There was no relation between the extent of the field and the degree of visual acuity. The author made no mention of central or of paracentral scotoma. The most marked defects occurred in the 7 eyes in which there was atrophy of the optic nerve.

PATHOLOGIC PICTURE OF PRIMARY ATROPHY OF THE OPTIC NERVE

The various pathologic studies of syphilitic atrophy of the optic nerve have given somewhat conflicting results. These have already been outlined in detail by Moore¹². For our purposes they may be summarized as follows:

Stargardt³ formed the following conclusions from extensive pathologic studies in 24 cases of atrophy of the optic nerve in association with dementia paralytica, the tabetic form of dementia paralytica and tabes. The lesion in the nerves consists in a primary peripheral and interstitial neuritis with secondary degeneration of the nerve fibers. The most frequent sites of the exudative process are the intracranial and the foraminal segment of the nerve, and next in frequency is the chiasm.

10 Goldberg, F. Ueber initiale Veränderungen des Gesichtsfeldes bei Tabes im Zusammenhang mit der Frage der primären Lokalisation der Läsion der Sehbahnen, *Sovet vestnik oftal* **6** 508, 1935.

11 Rutherford, C. W. Central Visual Fields and Pupils in Neurosyphilis, *Tr. Am. Ophth. Soc.* **33** 81, 1935.

12 Moore, J. E. The Syphilitic Optic Atrophies with Especial Reference to Primary Optic Atrophy, *Medicine* **11** 263, 1932.

The orbital portion of the optic nerve, the tract and the lateral geniculate body are only rarely affected. In cases of paralysis the exudative process spreads from the brain to the optic pathways. In cases of tabes there is an isolated process in the optic nerve or the chiasm which may spread to the brain.

Wilbrand and Saenger¹³ suggested that in cases in which there is a relatively early decrease in central acuity with only slight peripheral contraction the degenerative process might begin in the orbital part of the optic nerve, where the papillomacular bundle is peripherally located.

Igersheimer¹⁴ summarized the results of his pathologic investigations as follows. Histologic evidence of atrophy in the nerve may occur in cases in which the ophthalmoscopic examination shows no abnormality. In the beginning the degenerative process is always at the periphery of the optic nerve. Inflammatory changes in the connective tissue do not necessarily occur. The findings in 2 cases (in 1 of which there were central scotoma and degeneration of the papillomacular bundle) show that the process can be distal or retrobulbar. The most frequently occurring form is diffuse. Another form, never before described, has as its characteristic sign a circumscribed focus.

Wagner von Jauregg¹⁵ criticized the previous pathologic studies and expressed the belief that valid conclusions could be drawn only from a study of cases of pure tabes when atrophy of the optic nerve is in an early stage.

Although the majority of the pathologic studies support the view that the atrophic process starts in the marginal fibers of the intracranial portion of the optic nerve, distal to the chiasm, Stargardt's findings indicate that it may start at the chiasm, and Igersheimer's, that it may start in the orbital section of the optic nerve. As pointed out by Wagner von Jauregg, however, little is known of the pathologic changes characteristic of early uncomplicated tabes.

PRESENT STUDY

MATERIAL

The subjects for this study were 433 syphilitic patients. At the first examination 145 of these were discarded on account of some other complicating ocular disease, such as syphilitic choroiditis or glaucoma. There remained 288 patients for study. Of these 288 patients, 185 showed normal vision, visual fields and fundi on repeated examinations, and 103 showed evidences of involvement of the optic

13 Wilbrand, H., and Saenger, A. *Die Neurologie des Auges*, Wiesbaden, J. F. Bergmann, 1913, vol. 5, chap. 22, p. 543.

14 Igersheimer, J. *Ueber der Opticusprozess bei Tabes und Paralyse*, *Deutsche med. Wchnschr.* **52** 943, 1926.

15 Wagner von Jauregg, J. *Ueber tabische Optikus-Atrophie und deren Behandlung*, *Ztschr. f. Augenh.* **61** 127, 1927.

neuro-pathways Of the 103 patients with such involvement, 41 were examined only once and then disappeared from observation, while 62 were observed over periods varying from one month to seven years

METHODS

The ophthalmologic studies consisted of examination of the external eyes, of the pupillary reactions and of extraocular movements, measurement of the tension, ophthalmoscopic examination, determination of the corrected visual acuity, tests of the light sense, and perimetric studies The results of the tests of the light sense will be reported in a separate paper

All the objective examinations of the eye were made by a single examiner (A C W) The following terms are used in this report in clinical evaluation of the degree of change in the disks from normal to complete primary atrophy: normal disks, questionable pallor, early primary atrophy, moderate primary atrophy, and advanced primary atrophy All the subjective tests, i e, determinations of acuity, of light sense and of visual fields, were likewise made by one examiner (L L S) The Ferree-Rand perimeter and tangent screen were used in charting the visual fields In order to determine quantitatively the severity of the defects in different parts of the field, two or more test objects were used, selected from a graded series of white, blue and red test objects of 3, 1 and $\frac{1}{2}$ degrees The 1 and the $\frac{1}{2}$ degree white test objects were particularly useful in detecting slight defects in the far periphery, and the 1 degree colored objects, in testing the central and the midperipheral region The 3 degree white test object was used in those cases in which marked defects were found with the 1 and the $\frac{1}{2}$ degree white object The $\frac{1}{2}$ degree blue and red test objects were used in detecting early defects in the central and the paracentral field in cases in which fields were normal for 1 degree white and colored objects

Throughout this paper the region in which a given test object appeared weaker or dimmer than in the surrounding regions is designated as a relative scotoma for that particular test object The term absolute scotoma is used to designate an area of complete blindness for a particular white or colored test object For example, in a given region there might be relative scotoma for a $\frac{1}{2}$ degree white and absolute scotoma for a 1 degree red object or relative scotoma for a 1 degree red and absolute scotoma for a $\frac{1}{2}$ degree red object Because of the greater reproducibility of the limits of an absolute scotoma, a test object sufficiently small to demonstrate an absolute scotoma was used whenever possible, rather than a larger object which could show only a relative scotoma

RESULTS

The patients on whom these studies are based fell into four general clinical groups (a) patients with primary atrophy of the optic nerve (56), (b) patients who had normal fundi but nevertheless showed defects in the visual fields similar to those found in patients with frank primary atrophy of the optic nerve (12), (c) patients with normal fundi or such lesions of the fundi as retinal arteriosclerosis or questionable pallor of the disks but with the common symptom of homonymous defects in the fields (12), and (d) patients with either active optic neuritis or a condition of the fundi indicative of a previous inflammatory process in the optic nerves (23)

The results of the clinical and perimetric studies on these different groups of patients are presented in four separate sections

(a) *Patients with Clinically Diagnosed Syphilitic Primary Atrophy of the Optic Nerve*—There were 56 patients with primary atrophy of the optic nerve among the 106 syphilitic patients with involvement of the optic neuro-pathways. The changes in the fields of these 56 patients were of four distinct types, as follows: (1) concentric peripheral contraction of the field (in 7), (2) localized defect in the peripheral field (in 19), (3) central or cecocentral scotoma with a normal peripheral field (in 8), and (4) central or cecocentral scotoma with a contracted peripheral field (in 22)

Type 1 The first type is characterized by an approximately concentric contraction of the field. In the early stages the defect is limited to the far periphery, the fields for colors and the central visual acuity are normal while the fields for $\frac{1}{2}$ degree and 1 degree white test objects show a moderate concentric contraction. The nerve head at this stage may show slight pallor or may look entirely normal. As the field defect progresses gradually inward, the fields for colors, particularly for red, show moderate concentric contraction. The nerve head at this stage shows slight or moderate pallor, but the central visual acuity remains normal. In the advanced stages the limits for a 1 degree white object show marked contraction, the field for a 1 degree red object may be reduced to a small central patch or be lost altogether, the visual acuity is moderately reduced and the nerve head shows definite pallor.

Seven of the entire group of 56 patients with primary atrophy of the optic nerve showed this type of field defect. The findings for these 7 patients are summarized in table 1. To illustrate the various stages of this form of defect, selected perimetric charts for these 7 patients are given (figs 1-3). The fields in cases 1, 2 and 3 (fig 1) illustrate an early stage, which may be present before there is ophthalmoscopic evidence of atrophy of the optic nerve. In case 1, for example, at the time of the first perimetric test both disks appeared normal, four months later the right disk showed slight pallor but the left was still normal. In case 2 there was definite pallor of the right disk but only questionable pallor of the left. In case 3 at the time of the first perimetric tests the right disk showed questionable pallor and the left was normal. At the time of the second examination, six months later, there was moderate primary atrophy of the right disk, the left was still normal. The fields for the second date are shown in figure 1.

A somewhat more advanced stage of concentric contraction is illustrated in case 4. The fields (fig 2) were plotted on a tangent screen of 30 degree radius and are shown on large scale charts covering only this portion of the field. With the right eye a $\frac{1}{2}$ degree red test object

TABLE 1—*Patients with Concentric Peripheral Contraction of Fields (Type 1)*

Case Number and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields
1				Tabs	Followed for only 3½ mo decrease in fields
11/24/34	O U Moderate concentric contraction	O U Normal	O U 20/15		
5/12/35	O D Increased contraction O S No change (fig. 1)	O D Early pallor O S Normal	O U 20/15		
2	O U Slight concentric contraction (fig. 1)	O D Early pallor O S Questionable pallor	O U 20/15	Diffuse meningo vascular	Seen only once
3				Diffuse meningo vascular	Decrease in fields, followed by improvement
5/2/34	O U Moderate concentric contraction	O D Questionable pallor O S Normal	O U 20/15—1		
11/15/34	O U Increased contraction (fig. 1)	O D Moderate primary atrophy of optic nerve O S Normal	O U 20/15—1		
11/6/36	O U Improvement	O D Moderate primary atrophy of optic nerve O S Pallor of peripheral rim of nerve tissue temporally and above	O D 20/15—3 O S 20/15		
4	O U Marked concentric contraction (fig. 2)	O U Advanced primary atrophy of optic nerve	O D 20/15 O S 20/30	Dementia paralytica	Seen only once
5	O D Blind O S Marked concentric contraction (fig. 5)	O D Advanced primary atrophy of optic nerve O S Moderate primary atrophy of optic nerve	O D Blind O S 20/30	Tabs	Seen only once but reported totally blind 4 yr later
6	O D Perception of light only O S Marked concentric contraction (fig. 3)	O U Advanced primary atrophy of optic nerve	O D Perception of light only O S 20/70	Dementia paralytica	Seen only once
7				Tabs	Decrease in fields between 1932 and 1936, questionable improvement in 1937
1/30/32	O D Blind O S Marked concentric contraction	O U Advanced primary atrophy of optic nerve	O D Blind O S 20/50+2		
1/25/35	O S Greater contraction	O U Same	O S 20/50—1		
2/14/36	O S Greater contraction (fig. 3)	O U Same	O S 20/50+2		
10/4/37	O S Questionable improvement	O U Same	O S 20/50		

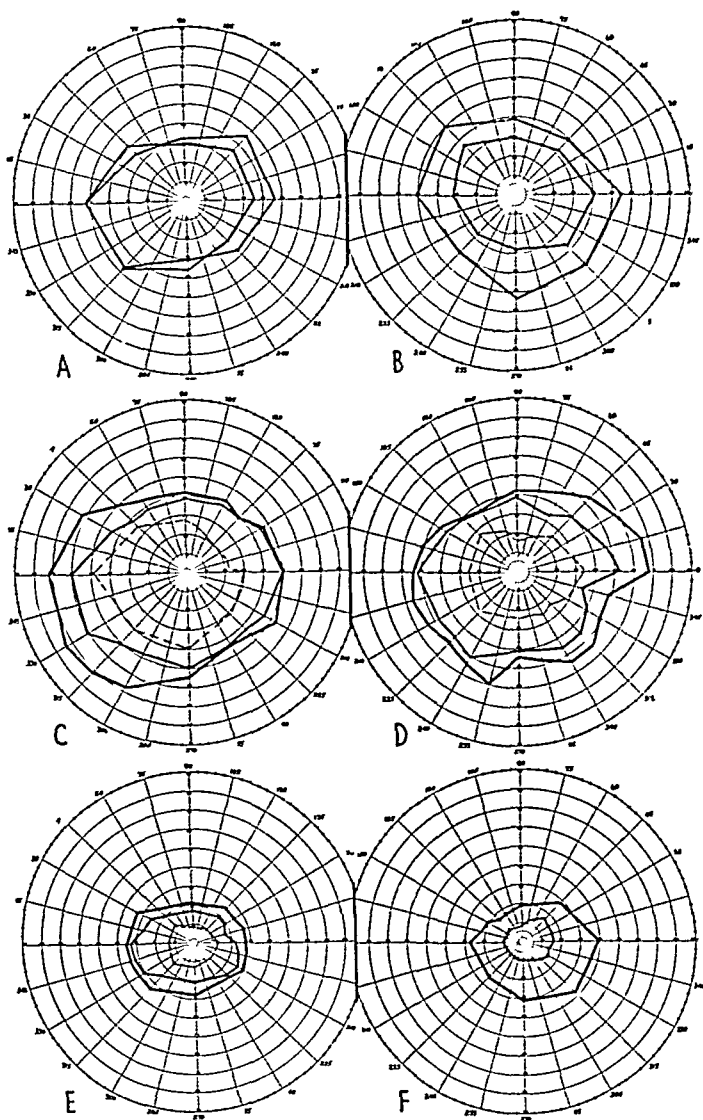


Fig 1—Early stage of concentric contraction (type 1) *A* and *B* (case 1) show the left and the right visual field, respectively, for a 1 degree white test object. The larger fields in *A* and *B* are those mapped on Nov 27, 1934. The smaller fields, those mapped on March 12, 1935. The visual acuity of both eyes was 20/15 and the fundi were normal at the time of the first determination. At the second examination the right disk showed slight pallor. *C* and *D* (case 2) represent the left and the right visual field for 1 degree red (broken line), $\frac{1}{2}$ degree white (inner solid line) and 1 degree white (outer solid line) test objects. There was questionable pallor of the left disk and only primary atrophy of the optic nerve. Visual acuity for both eyes was 20/15. *E* and *F* (case 3) represent the left and the right visual field for 1 degree red (broken line), 1 degree white (inner solid line) and 3 degree white (outer solid line) test objects on Nov 15, 1934. The left eye showed a normal fundus, and moderate primary atrophy of the optic nerve was present in the right. Vision for both eyes was 20/15—1.

was seen in a small area extending from 2 to 4 degrees from the fixation point, showing that in spite of the marked contraction the foveal area was still relatively intact. In the left eye the limits of the field for a $\frac{1}{2}$ degree red object, though wider, approached the fixation point in the upper and the temporal meridian. The visual acuity was 20/15 in the right eye and 20/30 in the left. Both disks showed advanced primary atrophy of the optic nerve.

In cases 5, 6 and 7 the vision in one eye was reduced to light perception or to total blindness. In the better eye there was moderate reduction of visual acuity, to 20/30, 20/70 and 20/50, respectively. The disks showed advanced atrophy, and the fields for a 1 degree white test object were markedly contracted in every case. In cases 5 and 7, a 1 degree red object was seen in a small central area. In case 6, a 3 degree red test object was not seen anywhere as red. The patient, it should be noted, had a lower visual acuity than did the 2 who still

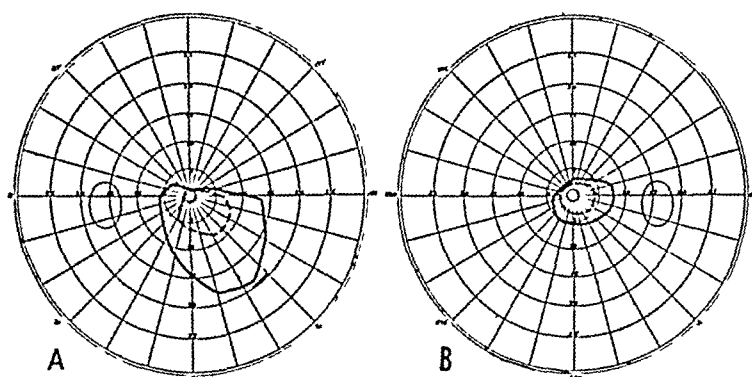


Fig 2 (case 4)—Visual field of (A) left and (B) right eye, showing advanced stage of concentric contraction, with preservation of good central vision (type 1) in a case of advanced primary atrophy of the optic nerve. The broken line bounds the field for a $\frac{1}{2}$ degree red and the solid line for a 1 degree white test object. Visual acuity for the left eye was 20/30 and for the right 20/15.

retained some perception of color at the fovea. In case 7 the fields at three different times are given, to show the gradual decrease during four years (fig 3). During this time the visual acuity remained unchanged.

On account of the late involvement of the fovea with this type of field defect, the central visual acuity gives little or no indication of the degree of involvement of the optic nerve. The findings in cases 1 and 3 indicated that fairly marked field defects can be present before there is ophthalmoscopic evidence of atrophy of the optic nerve. When first observed, the disks in case 1 were classed as entirely normal, but there was concentric contraction of the visual fields. Three months later the right eye showed definite pallor, and the visual field was further reduced. In case 3 there was questionable pallor of the right disk and

a normal left disk at the time of the first examination, the visual fields were contracted. Six months later the atrophy was definite in the right eye, and the visual field was further reduced. Two years later there was improvement in the fields of both eyes, but the nerve heads were essentially unchanged.

Type 2 A second and more common type of field defect is that characterized not by concentric contraction but by a defect particularly marked in some one section of the field. Table 2 summarizes the find-

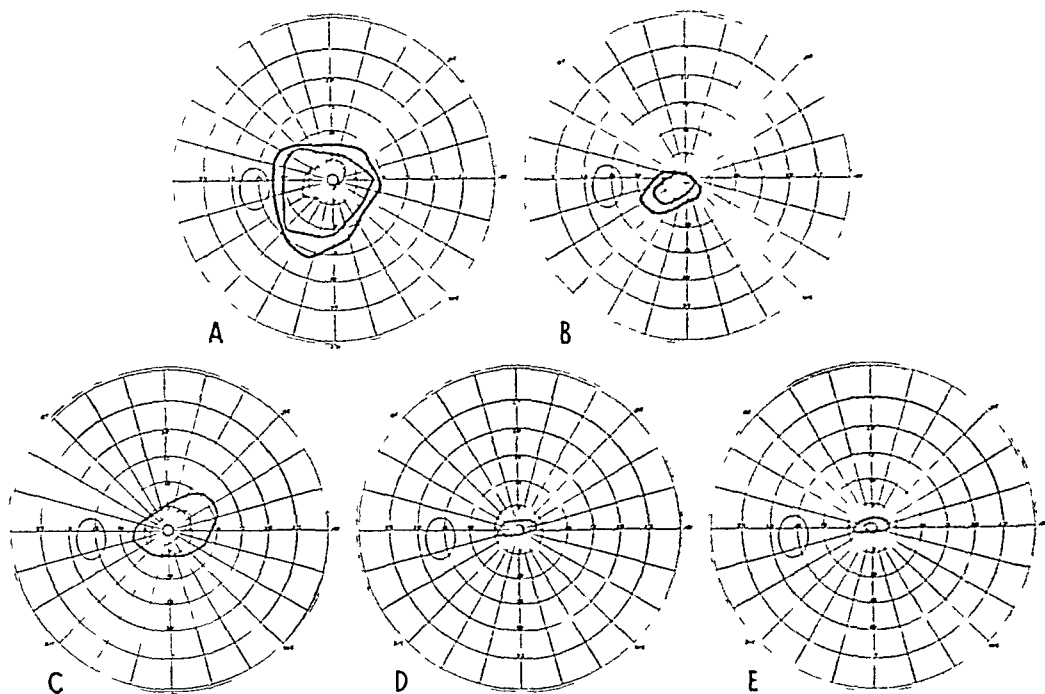


Fig 3—Advanced stage of concentric contraction, associated with reduced central vision (type 1). *A* (case 5) shows the left visual field for 1 degree red (broken line), 1 degree white (inner solid line) and 3 degree white (outer solid line) test objects in a case of moderate primary atrophy of the optic nerve. Vision was 20/30. *B* (case 6) shows the left visual field for 1 degree white (inner line) and 3 degree white (outer line) test objects in a case of advanced primary atrophy of the optic nerve. Vision was 20/70. *C*, *D* and *E* (case 7) show the left visual field in a case of advanced primary atrophy of the optic nerve as of Jan 30, 1932 (*C*), Jan 25, 1935 (*D*), and Feb 14, 1936 (*E*). The solid line bounds the field for a 1 degree white test object and the broken line that for a 1 degree red test object. Vision at the first and the third determination was 20/50 +2 and at the second determination 20/50 —1.

ings for the 19 patients whose fields were of this type¹⁶. As with type 1 the central visual acuity may be normal in the early stages when

¹⁶ In 10 of these, as has been indicated in the table, the classification as to type of defect in field was somewhat uncertain because of poor cooperation and unsteady fixation or because of advanced defects in field.

the foveal region is not involved, and there may likewise be only slight pallor of the disk. In general, however, reduction of acuity occurs at an earlier stage than with type 1, because the localized field defect progresses inward and involves the central region sooner than does a defect characterized by concentric contraction.

Selected charts illustrating various stages and forms of this type of defect are shown in figures 4 and 5. The fields in 4 cases (8, 9, 10 and 11) in which there were early field changes and normal visual acuity are shown in figure 4. In case 8 the right eye had a normal disk and a normal visual field. The left eye showed early atrophy of the optic nerve. The lower half of the visual field showed contraction for both white and red test objects, while the upper half of the field was entirely normal. Under treatment this patient ultimately showed complete restoration of the visual field. In case 9 the right eye showed only a slight cut in the lower nasal field and the left eye a more advanced defect limited chiefly to the nasal field. There was bilateral early atrophy of the optic nerve. In case 10 both eyes showed almost total loss of the upper portion of the field for a 1 degree white test object and moderate contraction in the lower portion. The disks showed moderate pallor. In case 11 the right eye was blind. The field defect in the left eye was limited entirely to the upper temporal quadrant. In this quadrant the limits for a 1 degree red test object came close to but did not involve the fixation point. Visual acuity was consequently not reduced. Examination of the fundi showed advanced atrophy of the optic nerve on the right and early atrophy on the left.

The fields in cases 13, 14, 15 and 16, shown in figure 5 for one eye only, illustrate a more advanced stage. The localized defect extended from the periphery and involved the foveal region, with a resultant reduction in acuity. In case 13 the defect for a 1 degree white test object in the upper field extended to the fixation point, and neither the 1 degree blue nor the 1 degree red test object was seen in this region. The visual acuity was 20/100. In case 14 the defect for the 1 degree white and for the 1 degree blue object were limited almost entirely to the nasal field. A 3 degree red test object, however, was not seen anywhere as red. The vision was 10/200. There was bilateral advanced atrophy of the optic nerve. The fields in case 15 were somewhat similar. Complete loss of vision for all colors, however, indicated a greater involvement of the temporal field than was present in case 14. In case 16 the fields showed a partial recovery of function in the foveal region a year after the first determination. In May 1933 the 1 degree blue test object was seen only in a small paracentral area of the temporal field. The visual acuity was 20/70 + 2. In April 1934 there was a slight increase in the field for the blue test object, which was seen at

TABLE 2—*Patients with Localized Defects in Peripheral Fields (Type 2)*

Case No and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
8 3/19/31	O D Normal	O D Normal	O U 20/15	Incipient dementia	Complete recovery	
12/21/31	O S Defect in lower field	O S Early primary atrophy of optic nerve	O U 20/15	dementia paralytica		
1/18/35	O D Normal	O U Same	O U 20/15			
9 1/18/35	O S Improvement	O U Same				
	O U Normal (fig 4)					
	O D Slight contraction in lower nasal field	O U Early primary atrophy of optic nerve	O U 20/15	Dementia paralytica	No change when retested 7 mo later	
	O S Contraction, especially of nasal field (fig 4)					
10 1/18/35	O U Contraction, chiefly of upper field (fig 4)	O U Moderate primary atrophy of optic nerve	O U 20/15	Tabs	Seen only once	
11 1/18/35	O D Blind	O D Advanced primary atrophy of optic nerve	O D Blind	Tabs	Seen only once	
	O S Contraction in upper temporal quadrant (fig 4)	O S Early primary atrophy of optic nerve	O S 20/20			
12 1/18/35	O D Advanced contraction of temporal field	O U Moderate primary atrophy of optic nerve	O D 20/200	Diffuse meningo vascular	Followed for 11 mo only	Successful tests showed decrease in fields
	O S Contraction, especially of temporal field		O S 20/30	Tabs	Followed for 2 yr, no change	
13 1/18/35	O D Normal	O D Normal	O D 20/15			
	O S Contraction, especially of upper field (fig 5)	O S Advanced primary atrophy of optic nerve	O S 20/100			
14 1/18/35	O D Contraction, especially of nasal field	O U Advanced primary atrophy of optic nerve	O D 5/100	Diffuse meningo vascular	Seen only once	
	O S Blind (fig 5)		O S Blind			
15 1/18/35	O D Contraction, especially of nasal field	O U Moderate primary atrophy of optic nerve	O D 14/200	Dementia paralytica	Seen only once	
	O S Light perception in temporal field only (fig 5)		O S Light perception			
16 5/1/33	O D Contraction, especially in nasal field	O U Advanced primary atrophy of optic nerve	O D 20/70+2	Dementia paralytica	Improvement	
	O S Questionable light perception in lower temporal field		O S Questionable light perception			
1/20/34	O D Improvement in field for blue	O U Advanced primary atrophy of optic nerve	O D 20/40-1			
	O S No change		O S Questionable light perception			

17	O D Advanced contraction perception O S Questionable light	O U Advanced primary atrophy of optic nerve	O D 20/100 O S Questionable light perception	Diffuse meningo vascular	Became blind	Advanced defect in field, difficult to classify as to type
18	O D Blind O S Advanced contraction, mostly temporal	O U Advanced primary atrophy of optic nerve	O D Blind O S 12/200	Diffuse meningo vascular	Followed for 1½ yr, successive tests showed decrease in fields Became blind	Advanced defect in field, difficult to classify as to type
19	O D Advanced contraction, mostly nasal O S Questionable light perception	O U Advanced primary atrophy of optic nerve	O D 6/200 O S Questionable light perception	Dementia paralytica		Advanced defect in field, difficult to classify as to type
20	O D Blind O S Advanced contraction in nasal and in upper field	O U Advanced primary atrophy of optic nerve	O D Blind O S 1/200	Tubes		Advanced defect in field, difficult to classify as to type
21	O D Questionable light perception O S Advanced contraction, mostly of temporal field	O U Advanced primary atrophy of optic nerve	O D Questionable light perception O S 5/200	Congenital	Followed for only 1½ mo, fields became worse Seen only once	Advanced defect in field, difficult to classify as to type
22	O U Advanced contraction, greatest in lower field	O U Advanced primary atrophy of optic nerve	O D 20/70-2 O S 20/30-1	Tubes		
23	O D Advanced contraction, greatest in lower and lower temporal field O S Blind	O U Advanced primary atrophy of optic nerve	O D 10/200 O S Blind	Diffuse meningo vascular	Followed for 1½ yr, no change	Fields suggest altitudinal hemianopia, similar to those in case 10 except that lower involved
24	O D Advanced contraction, particularly in upper field O S Same as O D	O U Moderate pallor and waxy appearance of nerve heads	O D 20/100 O S 20/200	Clinical syphilis of central nervous system, unclassified (no spinal puncture)	Followed for 2 yr, no change Seen only once	Fields difficult to classify as to type because of advanced defect
25	O D Moderate contraction, mostly of temporal field O S Same as O D	O U Early primary atrophy of optic nerve	O D 20/10+2 O S 20/30-2	Tubes		Fields difficult to classify as to type because of advanced defect
26	O D Light perception in upper nasal field only O S Marked contraction, leaving only upper nasal field for 3° white	O U Advanced primary atrophy of optic nerve	O D Light perception O S Counting fingers at 6 in (15 cm)	Diffuse meningo vascular	Followed for only 8 mo, no change Followed for 6 yr, between 1/21/31 and 5/5/36 gradual decrease, questionable improvement on 3/13/37	Perimetric data unreliable because of unsteady fixation and poor cooperation Difficult to classify as to type because of advanced defect, might be type 1

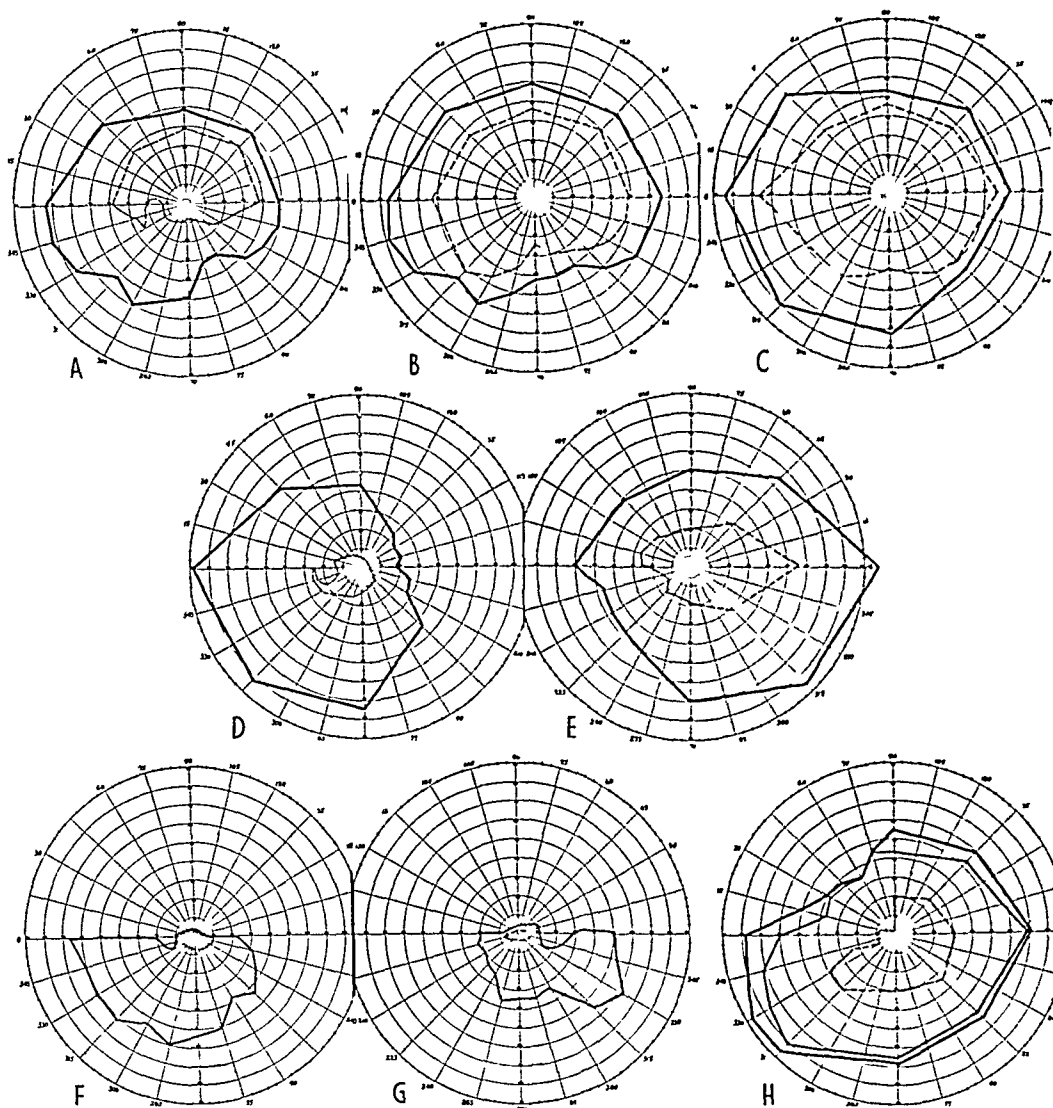


Fig 4—Various stages of localized peripheral defects, associated with normal central vision (type 2) *A*, *B* and *C* (case 8) show the left visual field in a case of early primary atrophy of the optic nerve on March 19, 1931, Dec 21, 1931, and Jan 18, 1935, respectively, for 1 degree white (solid line) and 1 degree red (broken line) test objects. Vision remained 20/15 throughout the period of observation. *D* and *E* (case 9) show the left and the right visual field, respectively, in a case of early primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white test object and the broken line that for a 1 degree red test object. Vision in both eyes was 20/15. *F* and *G* (case 10) show the left and the right visual field in a case of moderate primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white test object and the broken line, that for a $\frac{1}{2}$ degree red test object. Vision for both eyes was 20/15. *H* (case 11) shows the left visual field in a case of early primary atrophy of the optic nerve for 1 degree red (broken line), $\frac{1}{2}$ degree white (inner solid line) and 1 degree white (outer solid line) test objects. Vision was 20/20.

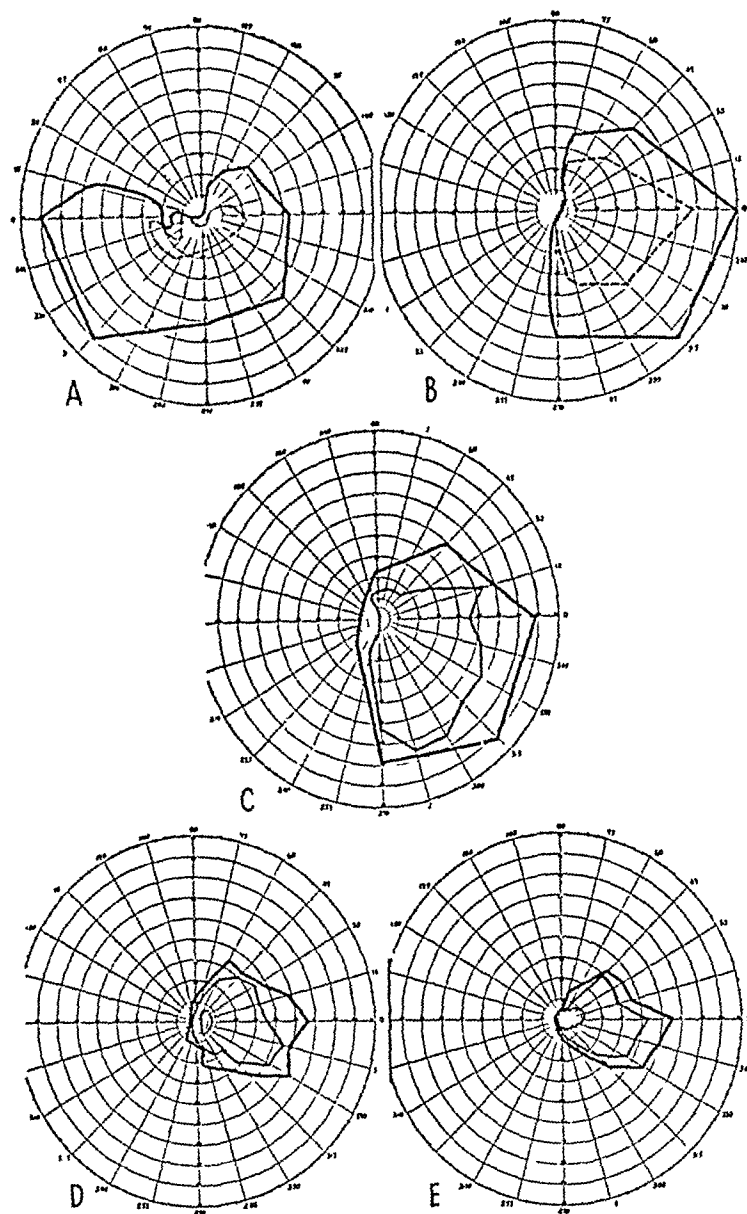


Fig 5—Localized defects, associated with reduced central vision (type 2) *A* (case 13) shows the left visual field in a case of advanced primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white test object and the broken line that for 1 degree red and blue objects. Vision was 20/100. *B* (case 14) shows the right visual field in a case of advanced primary atrophy of the optic nerve, the solid line bounding the field for a 1 degree white and the broken line that for a 1 degree blue test object. A 3 degree red test object was not seen. Vision was 5/100. *C* (case 15) shows the right visual field in a case of moderate primary atrophy of the optic nerve for a 3 degree white (outer line) and a 1 degree white (inner line) test object. A 3 degree blue and a 3 degree red test object were not seen. Vision was 14/200. *D* and *E* (case 16) show the right visual field in a case of advanced primary atrophy of the optic nerve on May 1, 1933, and April 20, 1934, respectively. The outer solid line bounds the field for a 1 degree white, the inner solid line that for a $\frac{1}{2}$ degree white and the broken line that for a 1 degree blue test object. A 3 degree red test object was not seen on either occasion. Vision at the first determination was 20/70 +2 and at the second 20/40 —1.

that time in the central region, and there was a corresponding improvement in visual acuity, to 20/40—1

As indicated in the illustrations and in table 2, the most marked defect may occur in any part of the field. It should be noted, however, that almost invariably the same region is affected in the two eyes. This tendency is illustrated in figure 4 (cases 9 and 10) and is evident from a study of the table.

Type 3 The third type of field defect differs markedly from the two previously considered. Central or cecocentral scotoma is associated with normal or nearly normal peripheral limits for a 1 degree white test object. Eight of the 56 patients had this type of defect. In a majority of these patients the disks showed atrophy of the papillomacular bundle or of the temporal portion. In 2 cases, however, although the perimetric studies indicated that the lesion involved chiefly the papillomacular bundle, localized temporal atrophy of the nerve was not observed on ophthalmoscopic examination. The visual acuity in cases of this type is involved early.

The fields in 3 cases (27, 28 and 29) illustrating this type of defect are shown in figure 6. In case 27 bilateral cecocentral scotomas were found with the 1 degree and the $\frac{1}{2}$ degree white test object. Only the smaller of these test objects showed involvement of the foveal area. The visual acuity was 12/200 in the right and 20/200 in the left eye. The peripheral fields of both eyes were normal for a 1 degree white test object. Attempts to map the fields for 1 degree blue and red objects gave variable and inconsistent results but indicated a probable slight involvement of the peripheral field. Both disks showed atrophy of the papillomacular bundle. In case 28 there was a unilateral defect only, consisting of a paracentral scotoma for a $\frac{1}{6}$ degree white test object and a larger cecocentral scotoma for a 1 degree red test object. No scotoma was demonstrable with a $\frac{1}{2}$ degree white test object. The visual acuity was 20/70—. The peripheral fields for 1 degree white, blue and red test objects were entirely normal, indicating that the lesion was confined to the papillomacular bundle. The disk, however, showed moderate generalized atrophy. In case 29 the perimetric findings showed early involvement of the right eye and a more advanced defect in the left. Results for two different dates are shown in the illustration. On the first date the left eye showed a cecocentral scotoma for $\frac{1}{2}$ degree white and 1 degree red objects. The scotoma for the white test object did not extend to the fovea. The left eye showed only a relative cecocentral scotoma for a $\frac{1}{2}$ degree red object, but this did not involve the foveal region. The visual acuity was 20/15—1 in the right eye and 20/20—1 in the left. Both nerve heads showed early atrophy of the papillomacular bundle. The results of the perimetric examination made

TABLE 3—*Patients with Central or Cecocentral Scotomas and Normal Peripheral Fields*

Case No and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
27	O U Cecocentral scotoma (fig 6)	O U Moderate atrophy of papillomacular bundle	O D 12/200 O S 20/200	Diffuse meningo vascular	Seen only once	
28	O D Cecocentral scotoma O S Normal (fig 6)	O D Moderate atrophy of atrophy of optic nerve O S Normal	O D 20/70— O S 20/15	Diffuse meningo vascular	Seen only once	
29	O D Cecocentral scotoma, fovea not involved O S Cecocentral scotoma involving fovea O U Scotoma 1 worse (fig 6)	O U Early atrophy of papillomacular bundle	O D 20/15—1 O S 20/20—1	Diffuse meningo vascular	Seen only once	
30	O U Central scotoma for 1° red	O U Advanced atrophy, especially of papillomacu lar bundle	O D 20/15—3 O S 20/40+3	Diffuse meningo vascular	Scotoma became worse between 12/3/31 and 5/23/36, and 1/5/37	
31	O D Blind (glaucoma) O S Central scotoma for 1½° white	O U Questionable atrophy of papillomacular bundle	O U 20/20—3 (uncorrected)	Tubes	Seen only once	
32	O U Cecocentral scotomas for 3° and 1° white	O D Advanced glaucoma O S Early primary atrophy of optic nerve	O D Blind (glaucoma) O S 15/200	Tubes	Followed for 4½ yr, no change	Poor cooperation, unsteady fixation
33	O D Cecocentral scotoma for 1° white O S Cecocentral scotoma for ½° white	O U Moderate atrophy of papillomacular bundle	O D 9/200 O S 6/200	Tubes	Followed for 8 mo only, no change	
34	O U Relative cecocentral scotomas for ½° blue and 1½° of questionable congenital red green color blindness	O U Advanced atrophy, possibly more marked in bundle O U Early pallor, especially of papillomacular bundle	O D 5/200 O S 10/200	Tubes	Followed for 5 yr, no change	
		O D 20/10 O S 20/70 (opacities in lens)	Congenital		Followed for 7½ yr, no significant change	Reduced vision, due in part to deposits of pigment on ante rior lens capsules, probable congenital red green color blindness

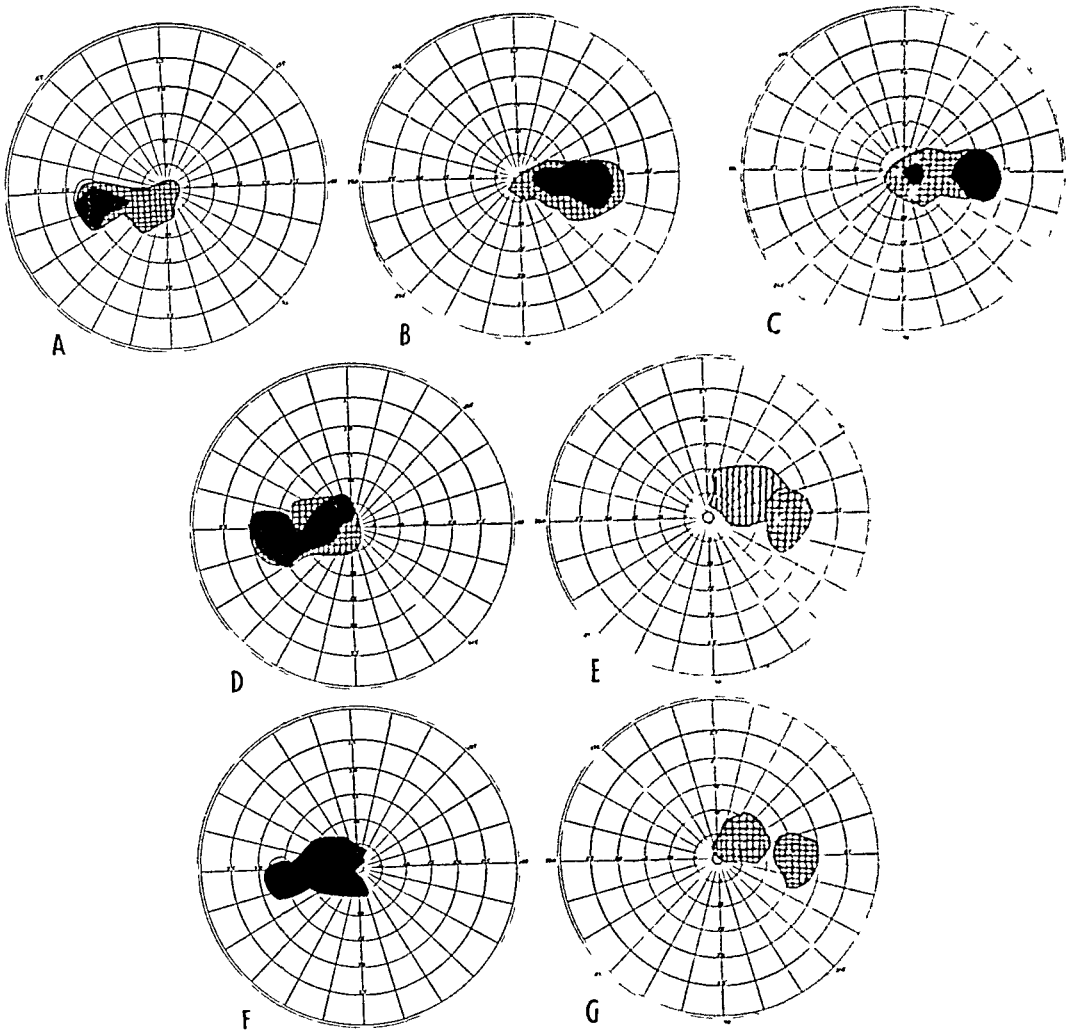


Fig 6—Cecocentral scotomas associated with normal peripheral fields (type 3). The central fields (to 30 degrees only) are shown in the figure. *A* and *B* (case 27) show a scotoma for 1 degree (solid area) and $\frac{1}{2}$ degree (cross-hatched area) white test objects in the left and the right eye, respectively, in a case of moderate atrophy of the papillomacular bundle. Both peripheral fields for a 1 degree white test object were normal. Vision for the left eye was 20/200 and for the right 12/200. *C* (case 28) shows a scotoma in the right visual field for a $\frac{1}{6}$ degree white (solid area) and a 1 degree red (cross-hatched area) test object in a case of moderate primary atrophy of the optic nerve. Vision was 20/70. *D*, *E*, *F* and *G* (case 29) show scotoma in the left and the right visual field on Dec 3, 1934 (*D* and *E*) and May 28, 1936 (*F* and *G*). At the time of the first observation early atrophy of the papillomacular bundle was present and vision for the left eye was 20/20 — 1 and for the right eye 20/15 — 1. At the time of the second observation advanced atrophy, especially of the papillomacular bundle, was present and vision was 20/40 + 3 for the left eye and 20/15 — 3 for the right. The solid area in both charts of the left visual field represents scotoma for a $\frac{1}{2}$ degree white test object. The cross-hatched areas in *D* and in *G* represent scotomas for a 1 degree red object. In *E* the area covered with vertical lines represents relative and the cross-hatched area absolute scotoma for a $\frac{1}{2}$ degree red test object.

eighteen months later showed a slight increase in the density of the scotomas in both eyes. The right eye showed an absolute scotoma for a 1 degree red object extending up to but not covering the fixation point. The vision was 20/15 — 3. In the left eye the scotoma for a $\frac{1}{2}$ degree white object had extended over the fixation point, and the visual acuity had decreased to 20/40 + 3. The ophthalmoscopic examination showed well advanced atrophy, particularly of the papillomacular bundles, and of about the same degree in both eyes.

Type 4. The defect of the fourth type is characterized by central or cecocentral scotoma, as in type 3, combined with a peripheral defect of either type 1 or type 2. There were 22 patients with this type of involvement. The findings are summarized in table 4. In a few cases advanced changes involving the central field were found at the first examination. It is difficult to say definitely whether these should be classified in type 2 or type 4. Such cases are indicated in the table.

In a majority of the patients with changes of this type, both the central and the peripheral defects were present at the first examination. These patients, like those with changes of type 3, showed an early reduction in visual acuity. Repeated studies of the field in 3 cases showed that the central scotoma developed later than the peripheral contraction. The especial involvement of the papillomacular bundle is sometimes apparent at ophthalmoscopic examination, but the most frequent finding is generalized atrophy similar to that seen with types 1 and 2.

Figures 7 to 12 illustrate defects of this type. In case 35 the fields of both eyes for the 1 degree white test object showed rather marked concentric contraction. The left eye showed in addition an absolute cecocentral scotoma for the 1 degree white object. The defects in the field of the right eye were less advanced. Both the 1 and the $\frac{1}{2}$ degree white test object were seen in the central region, but there was a relative central scotoma for the smaller object. The peripheral limits for the 1 degree white object showed less contraction than did those of the left eye, 1 degree red and blue test objects were not recognized anywhere with either eye. The visual acuity was 10/200 for the right and 2/200 for the left eye. Both disks showed primary atrophy, especially marked in the papillomacular area.

In cases 36 and 37 central scotoma was combined with peripheral defects which were more marked in one region of the field. In case 36 the peripheral defect in the right eye was limited almost entirely to the upper temporal quadrant. In the left eye the defect was more advanced. The field for a 1 degree white test object showed a marked defect in the upper temporal region and a slight contraction in the remaining portions. No tests with colors were made of the left eye. The visual acuity was 20/200 in the right and 2/200 in the left eye. The disks

TABLE 4—*Patients with Central Scotomas and Peripheral Defects (Type 4)*

Case No and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
35	O U Concentric contraction and cecocentral scotoma (fig 7)	O U Advanced atrophy, especially of papillo macular bundle	O D 10/200 O S 2/200	Diffuse meningo vascular	Seen only once	
36	O U Cecocentral scotoma and defect in upper tem poral field (fig 8)	O U Advanced atrophy of papillomacular bundle	O D 20/200 O S 2/200	Dementia paralytica	Followed for 8 mo only, slight improvement	
37	O D Large cecocentral scotoma and marked peripheral contraction O S Cecocentral scotoma and peripheral defect, especially in upper field (fig 9)	O D Advanced atrophy, especially of papillo macular bundle O S Moderate atrophy, especially of papillo macular bundle	O D II and motion O S 20/200+1	Diffuse meningo vascular	Seen only once	
38	O D Cecocentral scotoma for 1 colors (fig 10) O S 3 white seen only in nasal field	O U Advanced primary atrophy of optic nerve	O D 20/200 O S Light perception	Tabes	Followed for only 4 mo, no significant change	
39	O D Cecocentral scotoma and marked peripheral contraction, especially in nasal field (fig 10) O S Light perception only in lower field (questionable)	O U Advanced primary atrophy of optic nerve	O D 7/200 O S Hand motion	Tabes	Followed for 2 yr, no sig nificant change	
40 11/2/32	O D Cecocentral scotoma and peripheral defect, especially in temporal field O S 3 white seen in small area in nasal field	O U Advanced primary atrophy of optic nerve	O D 20/200 O S Finger counting at 6 in (15 cm)	Dementia paralytica	Followed for 2 yr, fields became worse	
9/12/34	O D More advanced O S Light perception only in nasal field (fig 11)	O U Advanced primary atrophy of optic nerve	O D S/200 O S Light perception			
41 12/20/35	O U Central scotoma for 1 red, marked peripheral contraction for 1° white O U Fields worse	O U Advanced atrophy, especially of papillo macular bundle	O D 20/100 O S 20/70—1	Tabes	Followed for 2½ yr, fields became gradually worse	
6/19/36	O U Fields worse		O D 20/200 O S 20/200+1			
1/12/37	O U Fields worse		O D 10/200 O S 14/200			
4/28/37	O U Fields worse		O D 4/200 O S 8/200			
11/2/37	O U Fields worse (fig 12)	O U Advanced atrophy, especially of papillo macular bundle O U Moderate atrophy, especially of papillo macular bundle	O D 1/200 O S 5/200	Tabes	Seen only once	Poor cooperation
42	O U Central scotoma and slight peripheral contrac tion for 1 white		O D 15/200 O S 15/200			

	O D Absolute central scotoma for 1° blue, slight concentric contraction for 1° white	O U Advanced primary atrophy of optic nerve	O D 20/30 O S 20/10	Diffuse meningo vascular	I allowed for 6 yr., no signifcant change	Congenital red green color blindness
43	O S Central scotoma, rela- tive for 1° blue, absolute for ½° blue, slight concen- tric contraction for 1° white (red not used because of congenital red green color blindness)	O U Advanced primary atrophy of optic nerve	O D 20/30 O S 20/10	Diffuse meningo vascular	I allowed for 6 yr., no signifcant change	Congenital red green color blindness
44	O D Normal O S Cecocentral scotoma for color and contraction of temporal field	O D Normal O S Moderate primary atrophy of optic nerve	O D 20/15—2 O S 20/70— 20/100	Tubes	Followed for 3½ yr., defect in peripheral field showed questionable im- provement, no change in central scotoma	
45	O D Cecocentral scotoma and peripheral contraction in nasal field O S Marked contraction, mostly in upper field	O U Moderate primary atrophy of optic nerve	O D 20/200— 12/200 O S 6/200	Diffuse meningo vascular	Followed for 3½ yr., no significant change	Perimetric data for O S unre- liable because of unsteady fixation
46	O U Moderate peripheral contraction for 1° white, 1° blue and red not seen, 3° blue seen in midperiphery but not in central field	O U Moderate primary atrophy of optic nerve	O D 20/200+1 O S 20/200+1	Diffuse meningo vascular	Followed for only 3 mo., no change	Poor cooperation, classifi- cation as to type of field is doubtful
47	O D 3° white, 1° blue and 3° red seen only in lower nasal quadrant, 1° red not seen O S Light perception only	O U Advanced primary atrophy of optic nerve	O D 2/200 O S Light perception	Diffuse meningo vascular	Followed for only 1 mo., no change	Advanced defect in field classi- fication as to type uncertain
48	O U Defects in cecocentral and in lower nasal field of nerve fiber bundle type O U More advanced	O U Moderate primary atrophy of optic nerve	O D 20/50—1 O S 20/50—1	Diffuse meningo vascular	Followed for 16 mo., gradual decrease	
10/31/33	O U More advanced	O U Advanced primary atrophy of optic nerve	O D 20/200 O S 20/200 O D 4/200 O S 4/200	Diffuse meningo vascular	Followed for 1½ yr., slight decrease	
2/24/36	O U More advanced (fig 13)	O U Advanced primary atrophy of optic nerve	O D 20/200 O S 20/200 O D 4/200 O S 4/200	Diffuse meningo vascular	Followed for 1½ yr., slight decrease	
49	O D Scotoma extending from blindspot, involving fovea and lower nasal field (nerve fiber bundle type) O S Complete loss of all but upper nasal field	O U Moderate primary atrophy of optic nerve, especially of papillo- macular bundle	O D 10/200 O S 6/200	Diffuse meningo vascular	Followed for 1½ yr., slight decrease	
2/21/35	O U Slightly worse (fig 14)	O U Advanced primary atrophy of optic nerve	O D 16/200 O S 6/200	Diffuse meningo vascular	Followed for only 1 mo., improvement	
9/ 9/36	O U Cecocentral scotoma and defect in lower nasal field (nerve fiber bundle type) Improvement (fig 15)	O U Moderate primary atrophy of optic nerve, more marked in O D O U No change	O D 3/200 O S 5/200 O D 5/200 O S 7/200	Diffuse meningo vascular	Followed for only 1 mo., improvement	
50	O D Light perception in upper nasal field only O S Defects in cecocentral and in lower nasal field (similar to fields in cases 48, 49 and 50)	O U Advanced primary atrophy of optic nerve	O D Light perception O S 6/200	Diffuse meningo vascular	Seen only once	
3/8/37						
4/2/37						
51						

TABLE 4—*Patients with Central Scotomas and Peripheral Defects (Type 4)—Continued*

Case No and Date LX unlined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
32 1/19/35	O D Defect in lower nasal field for 1° white, extending from periphery to blindspot O S Defect in lower nasal field for 1° white O U No change	O D Early primary atrophy of optic nerve O S Normal	O D 20/10— O S 20/10	Clinical of central nervous system, unclassified seronegative cerebrospinal fluid	Followed for 1 yr 7 mo no change during first year, decrease during second year	
1/10/36		O U Early primary atrophy of optic nerve, more marked in O D	O D 20/10— O S 20/10			
9/ 8/37	O U Fields worse defect in left field for 1° red now extends from lower nasal periphery to blindspot (nerve fiber bundle defects similar to those in cases 48 to 51)	O U Early primary atrophy of optic nerve, more marked in O D, and possibly localized some when in papillomacular bundles	O D 20/10— O S 20/20+1			
10 O D Defect in upper temporal field O S Blind (old injury)		O D Moderate primary atrophy of optic nerve O S Advanced primary atrophy of optic nerve	O D 20/10 O S Blind	Tuberc	Followed for 4 yr, no significant change	Congenital red green color blindness
54 2/15/32	O D Peripheral contraction, especially of nasal field O S Not tested (trophic choroiditis)	O D Early primary atrophy of optic nerve	O D 20/20	Diffuse meningovascular	Followed for 6 yr 5 mo defects in field became gradually worse between January and September 1932, no further decrease between September 1932 and April 1937 condition worse in October 1937	Atrophic choroiditis in O S
5/20/32	O D Peripheral contraction, especially of nasal field and eccentric scotoma for 1° blue	O D Moderate primary atrophy of optic nerve	O D 20/70			
9/21/32	O D Peripheral contraction as previously described and eccentric scotoma for 1° white (fig 16)	O D Advanced primary atrophy of optic nerve	O D 20/200			
55 5/31/33	O D Blind (atrophic choroiditis, retinitis proliferans) O S Concentric peripheral contraction for 1° white, 1° blue and red not seen	O S Early primary atrophy of optic nerve	O S 20/10—1	Tuberc	Followed for 1 yr central scotoma developed during first year no further change in scotoma, peripheral field showed gradual improvement	O D Blind (atrophic choroiditis, retinitis proliferans)
4/13/34	O S Relative central scotoma for ½ white peripheral limits show improvement (fig 17)	O S Advanced primary atrophy of optic nerve	O S 20/200, 1			
56 2/13/35	O U Peripheral contraction, particularly in upper and lower meridians O U Increased contraction and involvement of foveal regions (fig 18)	O U Advanced primary atrophy of optic nerve O U Advanced atrophy, especially of papillomacular bundle	O D 20/100 O S 20/70 O D 20/100 O S 20/70—2	Dementia paralytica	Followed for 2 yr, fields became worse	Congenital red green color blindness

showed bilateral atrophy confined to the papillomacular bundle. In case 37 there was a cecocentral scotoma of the left eye combined with a peripheral defect most marked in the upper field. The 1 degree red test object was seen in a small area in the lower field. Because of the advanced field changes in the right eye and the unsteady fixation, the results are somewhat unreliable. The findings indicate, however, that this eye also had a central scotoma combined with a defect in the periph-

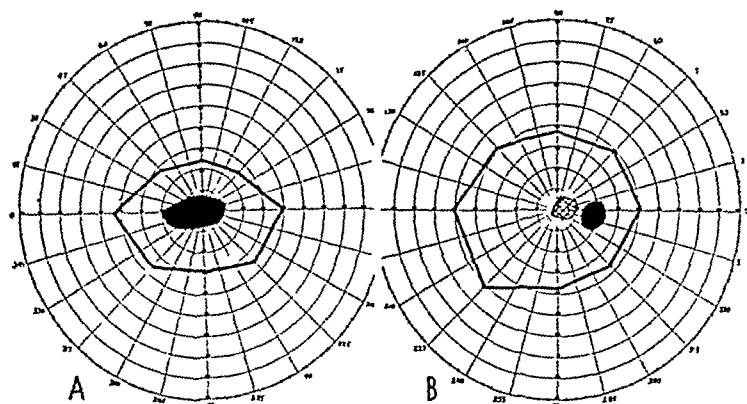


Fig 7 (case 35)—Visual field (A) of left and (B) of right eye in a case of advanced atrophy, especially of the papillomacular bundle, showing cecocentral scotomas and concentric contraction (type 4). Solid areas indicate absolute and cross-hatched areas relative scotoma for a $\frac{1}{2}$ degree white test object. Vision of the left eye was 2/200 and of the right 10/200.

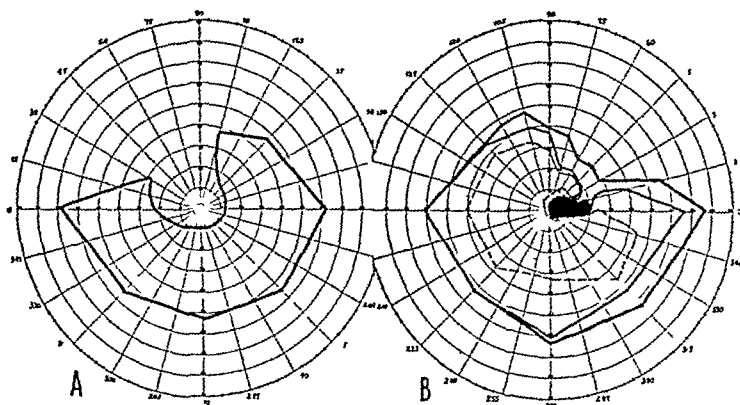


Fig 8 (case 36)—Visual field (A) of left and (B) of right eye in a case of advanced atrophy of the papillomacular bundle. For the left eye, vision was 2/200, and the field outlined is that for the 1 degree white test object. For the right eye, vision was 20/200, and the solid area indicates scotoma for a 1 degree white test object, the broken line bounds the field for the 1 degree red, the inner solid line that for the $\frac{1}{2}$ degree white and the outer solid line that for the 1 degree white test object.

eral field. The visual acuity was 18/200 in the right eye and was limited to perception of hand motions in the left eye. The disks showed primary atrophy, more marked in the papillomacular bundle.

In cases 38, 39 and 40 the fields for the right eyes only are shown, as the vision of the left eye in each case was limited to light perception. In case 38 the central area showed involvement for colors but not for the 1 degree white test object. A cecocentral scotoma was mapped with a 1 degree blue test object. The 1 degree red object was seen only in a small area in the nasal field. The 1 degree white test object showed

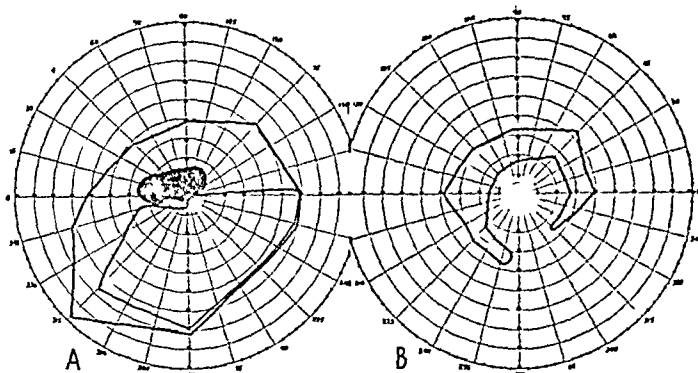


Fig 9 (case 37)—Visual field (A) of left and (B) of right eye in a case of advanced atrophy, especially of the papillomacular bundle. In A, the solid area is a scotoma for a 1 degree white test object, the outer line bounds the field for a 3 degree white and the inner line that for a 1 degree white test object. The field in B is for a 3 degree white test object. Vision of the left eye was 18/200, and that of the right was limited to the perception of hand movements.

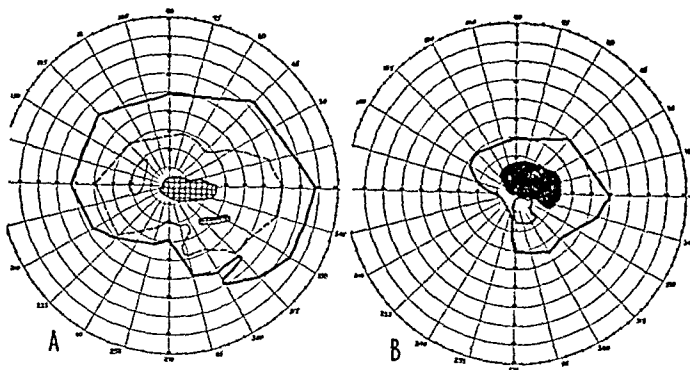


Fig 10—Right visual field in 2 cases of advanced primary atrophy of the optic nerve (A, case 38, B, case 39), showing cecocentral scotomas and peripheral defects (type 4). In A, the field bounded by the solid line is that for a 1 degree white test object, that bounded by long dashes, for a 1 degree blue object, and that bounded by short dashes, for a 1 degree red object. Cross-hatched areas indicate scotomas for the 1 degree blue test object. In B, the outer line bounds the field for a 3 degree white test object, and the inner line, that for a 1 degree white test object. The solid area indicates a scotoma for a 3 degree white test object. Vision in A was 20/200 and in B 7/200.

a defect limited to the lower field. The visual acuity was 20/200 in the right eye and was reduced to light perception in the left. The disks showed generalized pallor, more advanced in the left eye. In case 39

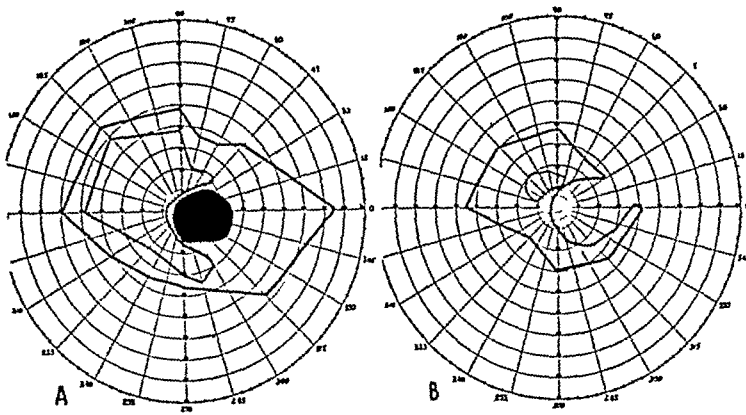


Fig 11 (case 40) —Visual field of right eye in a case of advanced primary atrophy of the optic nerve (*A*) on Nov 2, 1932, and (*B*) on Sept 12, 1934, showing progressive changes in a defect in field of type 4. The outer line bounds the field for a 3 degree white and the inner line that for a 1 degree white test object. The solid area indicates a scotoma for a 3 degree white test object. Visual acuity at the time of the first determination was 20/200 and at the second 8/200.

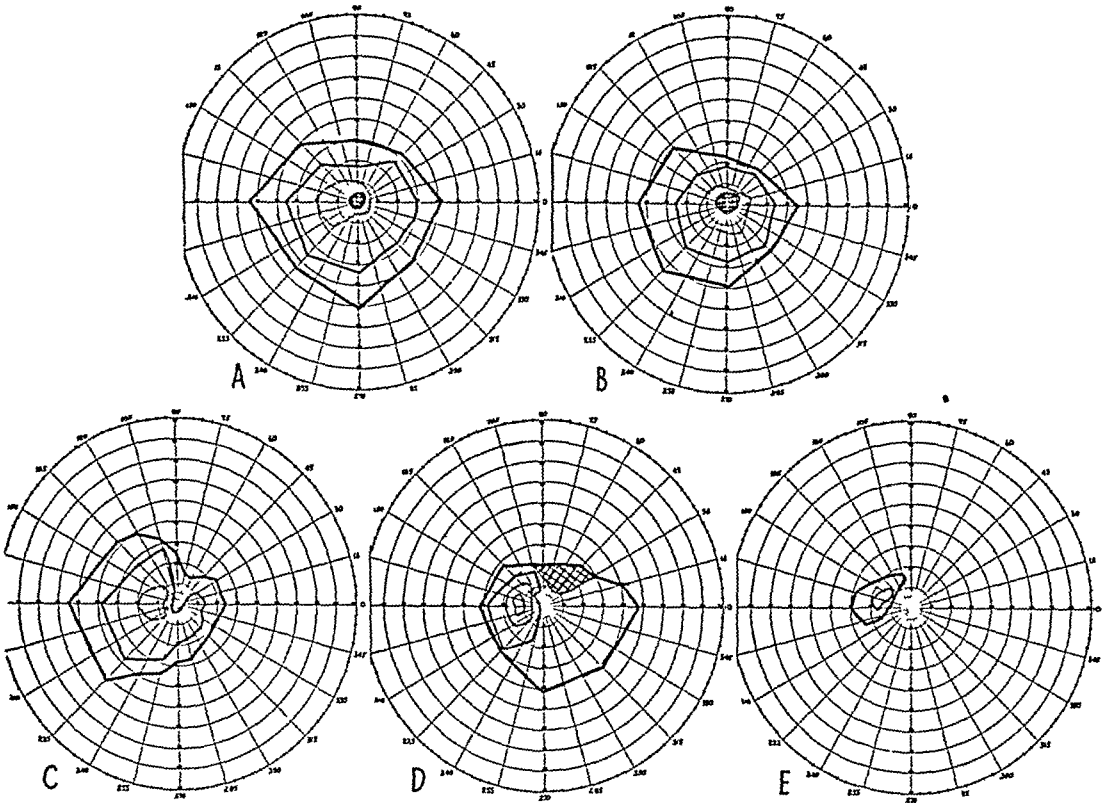


Fig 12 (case 41) —Progressive changes in a type 4 defect in the right visual field in a case of advanced atrophy, especially of the papillomacular bundle. On Dec 20, 1935, and on June 19, 1936 (*A* and *B*), vision was 20/200, on Jan 12, 1937 (*C*), 10/200, and on April 28 and on Nov 2, 1937 (*D* and *E*), 4/200. In *A*, *B* and *C*, the outer solid line bounds the field for a 3 degree white, the inner solid line that for a 1 degree white and the broken line that for a 1 degree red test object. The cross-hatched area in *A* and in *B* represents scotoma for a 1 degree red test object. In *D*, the broken line bounds the field for a 1 degree red test object and the solid lines, progressing from the center toward the circumference of the illustration, bound the fields for $\frac{1}{2}$ degree white, 1 degree white and 3 degree white test objects. Relative scotoma for a 3 degree white test object is represented by the cross-hatched area. In *E* the outer line bounds the field for a 3 degree white and the inner line that for a 1 degree white test object. A $\frac{1}{2}$ degree white and a 1 degree red test object were not seen.

the right eye showed an advanced defect. Perception of color was entirely lost. The 1 degree white object was seen only in a small area in the lower temporal field. Tests with a 3 degree white test object showed a large cecocentral scotoma and marked peripheral contraction. The visual acuity was 7/200 on the right and was reduced to perception of hand motions on the left. In case 40 there was an advanced defect in the field of the right eye, with complete loss of color vision. The 3 degree white test object showed a large central scotoma and moderate peripheral contraction. A retest of the fields about two years later showed a slight increase in the extent of the scotoma and a marked decrease in the peripheral limits. The results on both dates indicated a greater involvement of the temporal than of the nasal field. The visual acuity of the right eye was 20/200 at the time of the first test and 8/200 at the time of the second. Although perimetric studies of the left eye were impossible it is interesting that on the first date a 3 degree white test object was seen doubtfully in the nasal field only, and on the second date perception of light was confined to the nasal field. These findings indicate that the course of development in the left eye was probably similar to that in the right. The patient showed advanced generalized primary atrophy of the optic nerve in both eyes.

The visual fields in case 41, shown for the right eye only, illustrate the progressive changes in a somewhat similar defect. The disks showed marked primary atrophy of the optic nerve especially in the area of the papillomacular bundle. In December 1935 and in June 1936 there was a central scotoma for a 1 degree red but not for a 1 degree white test object. In January 1937 a 1 degree white object was not seen at the fovea and the field for the 1 degree red object was reduced to a small area in the nasal field. In April 1937 the extent of the field for a $\frac{1}{2}$ degree white object was approximately equal to the earlier field for a 1 degree red object. The charts illustrate the way in which defects in the field for color and for small white test objects frequently predict the defects which may later be found for larger white test objects if the progress of the atrophy is not arrested.

In 5 cases defects of type 4 in the earlier stages were similar to the nerve fiber bundle defects seen in cases of glaucoma and in some cases of chorioiditis juxtapapillaris. The progressive changes in the fields in 3 of the 5 cases (48, 49 and 50) are shown in figures 13 to 15. In case 48, at the time of the first examination, a 1 degree white test object showed a scotoma extending nasally and a sector-shaped defect in the lower field giving a large nasal step. The areas blind to the 1 degree white object did not include the fixation point in either eye. Tests with a 1 degree blue object (made only of the right eye) showed a defect extending from the blindspot to the nasal periphery and involving the fixation point. A 1 degree red object was

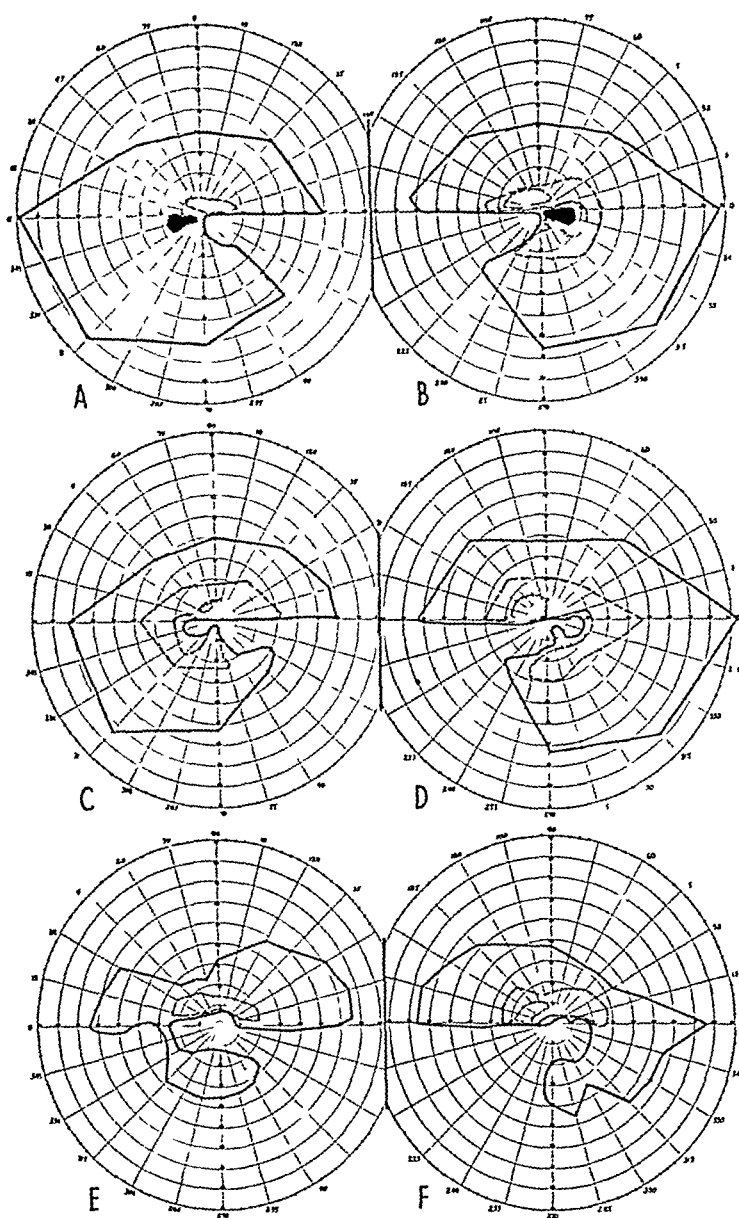


Fig 13 (case 48) —Progressive changes in nerve fiber bundle defects (type 4) On June 28, 1933, moderate primary atrophy of the optic nerves was present and vision in the left (A) and the right (B) eye was 20/50 —1 The solid area in the map of each field at that time represents scotoma for a 1 degree white test object, and the solid line, the boundary of the field for the same object The broken line in A bounds the field for a 1 degree red object, as does the inner broken line in B The outer broken line in B bounds the field for a 1 degree blue test object On Oct 31, 1933, and on Feb 24, 1936, the atrophy was in an advanced stage, the vision of each eye being 20/200 on the earlier date and 4/200 on the later one In C and D, representing the left and the right eye, respectively, on the earlier date, the solid line bounds the field for a 1 degree white test object, the outer broken line, that for 1 degree blue, and the inner broken line, that for 1 degree red In E and F, representing the left and the right eye on the later date, the solid line bounds the field for the 1 degree white and the outer broken line that for the 1 degree blue test object The inner broken line in F bounds the field for a 1 degree red test object (not visible to the left eye at that time)

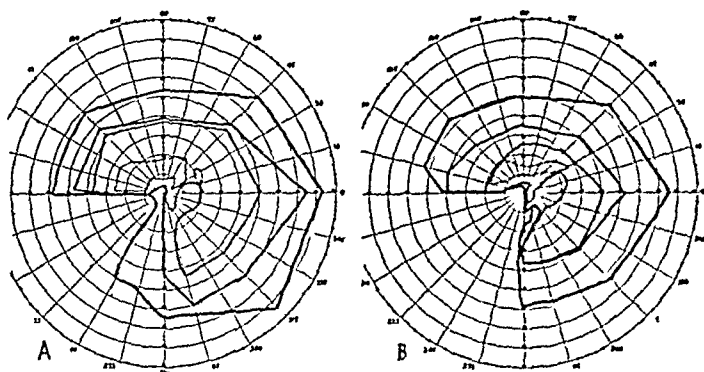


Fig 14 (case 49)—Right visual field (A) on Feb 21, 1935, and (B) on Sept 9, 1936, showing progressive changes in nerve fiber bundle defects (type 4). Moderate atrophy of the optic nerve, especially of the papillomacular bundle, was present at the time of the first observation, and vision was 10/200, at the time of the second observation, primary atrophy of the optic nerve was advanced, and vision was 16/200. A 3 degree red test object was not seen at either time. Progressing centrally from the circumference of the illustration, the solid lines bound the fields for the 3 degree, the 1 degree and the $\frac{1}{2}$ degree white test object, the broken line bounds the field for the 1 degree blue test object.

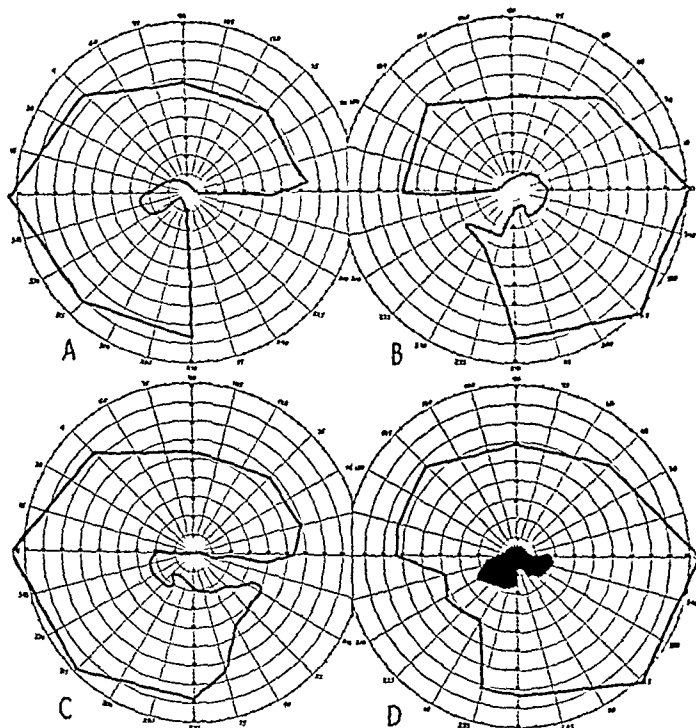


Fig 15 (case 50)—Defects of nerve fiber bundle type (type 4), showing improvement in one month in a case of moderate primary atrophy of the optic nerve. A and B show the left and the right field, respectively, on March 8, 1937, for a 1 degree white test object. Vision was 5/200 for the left and 3/200 for the right eye. C and D show the left and the right field on April 2, 1937, for a 1 degree white test object. The solid area in the right field represents scotoma for a 1 degree white test object. Vision was 7/200 for the left and 5/200 for the right eye.

seen only in a small area in the upper nasal field of each eye. At this time the visual acuity was 20/15—1 in both eyes, and ophthalmoscopic examination showed moderate primary atrophy of the optic nerve. Four months later the regions blind to the 1 degree white object had enlarged and included the fixation point. The visual acuity had decreased to 20/200, and both nerve heads showed advanced atrophy. The last perimetric examination, about two years later, showed, in addition to an increase in the central defect, considerable peripheral loss in all but the upper nasal sector of the field. A 1 degree red object was seen only in a small area in the upper nasal field of the right eye and not at all in the left eye. The visual acuity was 4/200 in both eyes. In these fields, as in those of case 41, the early findings with 1 degree blue and red test objects indicated the future course of the changes for the 1 degree white test object.

In case 49 a defect in the right eye extended from the blindspot into the lower nasal field, producing a nasal step. At the time of the first examination the tests with a 1 degree white object showed no evidence of involvement of the upper and temporal regions. The 1 degree blue test object was seen only in the upper field, however, and the perception of red was lost entirely. In the left eye the 1 degree white object was seen only in the upper nasal field. (Fields for this eye are not shown in the charts.) The vision was 10/200 in the right eye and 6/200 in the left. The disks showed well developed atrophy, especially marked in the area of the papillomacular bundle. Tests made about eighteen months later showed a loss in the upper and temporal portions of the peripheral field of the right eye but little increase in the defect in the lower nasal field. The visual acuity was essentially unchanged. In case 50 the fields showed a similar nerve fiber bundle defect involving the cecocentral area and the lower nasal field. The visual acuity at the time of the first test was 3/200 in the right eye and 5/200 in the left. The disks showed bilateral primary atrophy of the optic nerve, more marked in the right eye. Reexamination of the fields one month later showed some improvement in the defects in the lower nasal quadrants. The visual acuity of the right and of the left eye were 5/200 and 7/200, respectively.

The changes in the fields of the 3 patients in whom central scotoma developed after the peripheral defects are shown in figures 16 to 18. In case 54 the field of the right eye only is shown, atrophic choroiditis was present in the left eye. In the right eye at the time of the first examination there was a contraction of the nasal field for 1 degree white and colored objects. The blindspot for a 1 degree red test object showed a slight enlargement toward the fixation point. The visual acuity at this time was 20/20—. The right nerve head showed early generalized primary atrophy. Three months later a cecocentral scotoma for a 1 degree blue object merged with the defect in the nasal

field The vision had decreased to 20/50, and the disk showed a moderate degree of pallor Four months later there were beginning cecocentral scotoma and slightly greater nasal contraction for a 1 degree white object and complete loss of field for a 1 degree red object The visual acuity had fallen to 20/200, and ophthalmoscopic examination

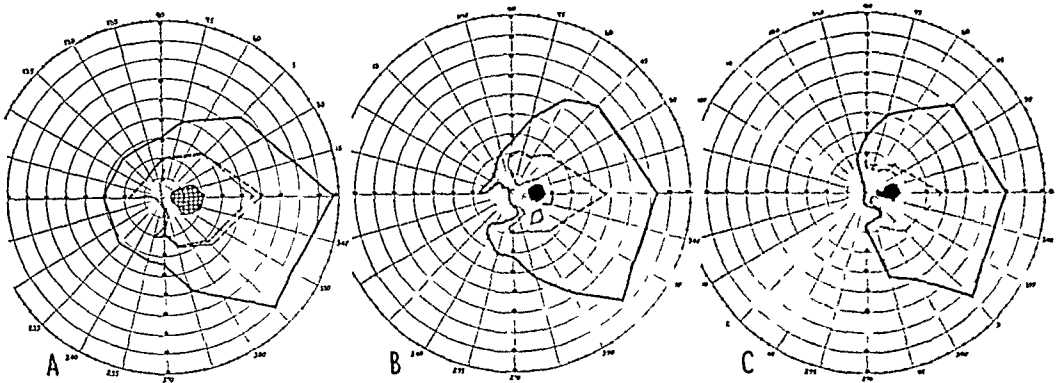


Fig 16 (case 54)—Visual field of the right eye, showing development of cecocentral scotoma in association with (A) early, (B) moderate, and (C) advanced primary atrophy of the optic nerve Observations were made on Feb 15, May 20 and Sept 21, 1932, when vision was 20/20, 20/50 and 20/200 respectively The cross-hatched area represents scotoma for a 1 degree red test object, and the solid areas, scotoma for a 1 degree white test object The solid line bounds the field for the 1 degree white test object, the line of long dashes, that for the 1 degree blue, and the line of short dashes, that for the 1 degree red (not seen at the time of the last observation)

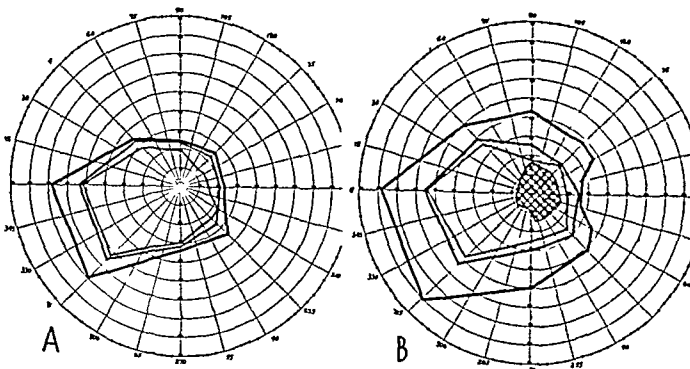


Fig 17 (case 55)—Visual field of the left eye, showing development of central scotoma after peripheral defect in association with (A) early and (B) advanced primary atrophy of the optic nerve Progressing centrally from the circumference of the illustration, the solid lines bound the fields for the 3, the 1 and the $\frac{1}{2}$ degree white test object Observations were made on May 31, 1933, and April 13, 1934, when the vision was 20/40 — 1 and 20/200 + 1, respectively The cross-hatched area indicates relative scotoma for the $\frac{1}{2}$ degree white test object

showed advanced primary atrophy The defects for 1 degree red and blue objects predicted the future loss in the field for the 1 degree white object

In case 55 the field of the left eye only is shown, atrophic chorioiditis was present in the right eye. At the time of the first examination the left eye showed a definite decrease in the extent of the fields for 3, 1 and $\frac{1}{2}$ degree white test objects. One degree blue and red objects were not seen in any part of the field. The corrected visual acuity was 20/40—1. The disk showed incipient generalized primary atrophy. Retests approximately ten months later showed some improvement in the peripheral limits of the field for 3 degree white and essentially unchanged fields for 1 and $\frac{1}{2}$ degree white objects. A large relative scotoma for a $\frac{1}{2}$ degree white test object had developed in the central

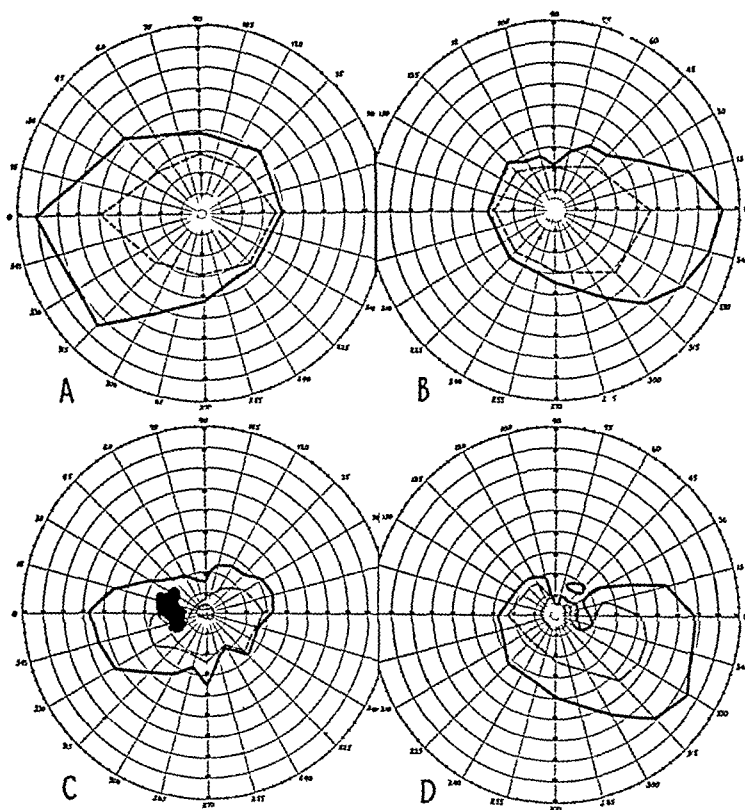


Fig 18 (case 56)—Progression of peripheral contraction and development of central scotomas in a case of advanced primary atrophy of the optic nerve. The fields for a 1 degree white test object are bounded by a solid line and those for a 1 degree blue test object by a broken line, *A* and *B* showing the left and the right field of Feb 13, 1935, and *C* and *D* those of March 19, 1937. In *C* the solid area represents scotoma for a 1 degree white and the cross-hatched area scotoma for a $\frac{1}{2}$ degree blue test object. Vision at both determinations was 20/70 for the left and 20/100 for the right eye. Atrophy was pronounced in the papillomacular bundles at the time of the second determination.

field. The visual acuity had dropped to 20/200 + 1, and the disk showed advanced primary atrophy.

In case 56 at the time of the first examination contracted fields for the 1 degree white object were found in both eyes. The fields for the

1 degree blue object were of normal extent. The 1 degree red object appeared white, but there was a possible congenital red-green color blindness, so that the findings with this test object were of doubtful value. The visual acuity was 20/100 in the right and 20/70 in the left eye. This marked reduction in acuity indicated involvement of the central field and suggested a central defect for red due to the atrophy of the optic nerve, in addition to the probable congenital defect, since the latter does not cause reduction in acuity. The nerve heads at this time showed advanced generalized primary atrophy. Retests of the fields two years later showed greater contraction, particularly in the upper temporal field. In the right eye the defect for blue extended downward to include the foveal region. In the left eye the field for a 1 degree blue test object showed a contraction of the limits of the upper temporal region to within about 6 degrees of the fixation point. There was, in addition, a central scotoma for a $\frac{1}{2}$ degree blue test object. The visual acuity was 20/100 in each eye. The disks on this date showed especially marked pallor in the area of the papillomacular bundles.

Comment—Figures 1 to 18 represent the four general types of defect found in the entire group of 56 patients with syphilitic primary atrophy of the optic nerve. Although the field defects are of different forms, there are certain characteristics common to the entire group. The fields of the two eyes show, as a rule, a marked similarity, although the defects are frequently more advanced in one eye than in the other. For example, some patients have bilateral central scotomas, others have bilateral concentrically contracted fields or localized peripheral defects in both eyes. Moreover, when bilateral localized peripheral defects are present, corresponding regions of the two fields are affected. In case 48 (fig 13), for example, a scotoma extended from the blindspot to the nasal periphery in the lower field of each eye. In other cases the similarity of the changes in the two eyes results in heteronymous hemianopic defects, which may be binasal, bitemporal or altitudinal. With only 1 exception (case 44), all patients with atrophy involving both optic nerves who retained sufficient vision in each eye to permit accurate perimetric study showed this similarity of defects.

The nature of the progressive decrease in the visual fields is illustrated in cases 1, 7, 29, 40, 41, 48, 49, 54, 55 and 56 (figs 1, 3, 6, 11 to 14 and 16 to 18). Defects for red practically always precede those for blue, as is the rule with lesions of the conducting pathways. The results obtained on any one date with a graded series of test objects often give information not only of the extent of involvement but of the future course of the field defect. In case 54, for example, the cecocentral scotoma for the 1 degree red object found at the first examination preceded a similar scotoma for the 1 degree blue and

later for the 1 degree white object. Similarly, in case 41 there was a central scotoma for the 1 degree red object only at the time of the first examination. One year later this central defect was demonstrable with the 1 degree white test object. In case 48, likewise, the fields for the 1 degree white object showed at first only a localized defect, but the results obtained with the 1 degree red object showed widespread involvement.

Forty of the 56 patients were reexamined at later dates. Twelve, or 30 per cent, pursued a steady downhill course, 3 becoming totally blind and 9 showing a steady loss in the visual fields. Seventeen showed unchanged fields during the period of observation. Six showed fluctuations in the fields, while 5 (cases 3, 8, 16, 36 and 50) showed definite improvement, 1 regaining normal fields. Thus, during the period of observation 28, or 70 per cent, showed at least a temporary arrest in the progress of their optic neuropathy. The efficacy of various forms of antisyphilitic therapy in arresting the progress of atrophy of the optic nerve will be the subject of later reports.

The perimetric fields in cases 8 and 16 (figs 4 and 5) illustrate the extent of improvement which may occur. In case 16 the change consisted only in a slight widening of the field for blue, but since this improvement involved the fixation point, it resulted in an increase in visual acuity from 20/70 + to 20/40 — 1. In case 8 a slight defect in the peripheral field for 1 degree white and colored objects disappeared entirely.

From the data for the entire group, summarized in tables 1 to 4, conclusions may be drawn as to the relative frequency of the different forms of field defect and the relation between the type of defect and the type of neurosyphilis, the type of atrophy of the optic nerve and the loss in visual acuity.

Central scotoma (type 3 or 4 defect) occurred in 30 of the 56 patients (53 per cent). No apparent relation exists between type of neurosyphilis and type of field defect. The 56 patients included 30 who had parenchymatous neurosyphilis (tabes, dementia paralytica or the tabetic form of dementia paralytica), 22 who had meningovascular neurosyphilis, 2 who had congenital neurosyphilis and 2 who had latent syphilis of the central nervous system and seronegative cerebrospinal fluid. Forty-seven per cent of the patients who had parenchymatous neurosyphilis and 64 per cent of those who had meningovascular syphilis had central scotoma, i. e., a field defect of type 3 or 4.

There is a definite relation between the type of atrophy of the optic nerve and the type of field defect. Especial involvement of the papillomacular bundle was noted in many of the patients with a defect of type 3 or 4 but was not observed in patients with a defect of type 1 or 2. Only 2 of the 8 patients with central scotomas and normal

peripheral fields (type 3 defect) showed generalized atrophy of the nerves. The remaining 6 patients who had this type of defect showed atrophy limited almost entirely to the papillomacular bundles. Seven of the 22 patients with central scotomas combined with peripheral defects (type 4 involvement) showed a greater degree of atrophy in the areas of the papillomacular bundles than in the remaining portions of the nerves. Thus, central scotoma is invariably present in patients who show distinctly localized atrophy of the papillomacular bundle, and from this clinical finding the presence of such scotoma may be prophesied. In patients with generalized pallor of the nerve however, clinical examination gives no information as to the presence or absence of central scotoma.

A number of the patients with field defects of type 1 or 2 had normal visual acuity, of 20/15, in one or both eyes. For those with defects of type 1 the vision was 20/15 in 7 of the 14 eyes. For those with type 2 defects the vision was 20/15 in 5 of the 36 affected eyes. Only 1 instance of normal visual acuity was observed among the 30 cases of central or cecocentral scotoma. In this case (39) the scotoma in the right eye extended close to but did not involve the fixation point.

A study of the relationship between visual acuity, ophthalmoscopic findings, and visual fields in the 56 cases suggests that the changes in the fields are probably the earliest clinical evidence of atrophy of the optic nerve. Defects in the visual fields were found for all patients who showed any reduction in the corrected visual acuity. However a number of patients, had normal acuity in spite of both defects of type 1 or 2 in the fields and pallor of the nerve heads. Field defects were found in every patient who showed pallor of the disks on ophthalmoscopic examination a number of those showing only early pallor having fairly well advanced defects in the fields. Three patients who had normal nerve heads at the time of the first ophthalmoscopic examination and subsequently had definite atrophy of the optic nerves showed defects in fields at the time of the first examination. It seems evident, therefore, that changes in the visual fields may antedate the ophthalmoscopic appearance of atrophy and that both may precede loss of central vision.

After a diagnosis of primary atrophy of the optic nerve has been made, studies of the visual fields provide a more reliable means of following the course of the atrophy than does either an ophthalmoscopic examination or a determination of the visual acuity. Since it is difficult to differentiate more than three stages of pallor of the disk, it is obvious that moderate progressive changes in the atrophic process may not be detected by ophthalmoscopic examination. The visual acuity may likewise remain unchanged while definite alterations in the visual field are taking place. The impairment of visual acuity depends, as a rule, entirely on the involvement of the fibers of the papillomacular bundle,

which supplies the fovea. When, however, the foveal impairment is sufficient to reduce the central acuity to 20/100 or less, the patient may use slightly eccentric fixation, so that further decrease in visual acuity is dependent on a change in the field in the parafoveal region. In either case the maintenance of visual acuity at a constant level for a considerable period by no means indicates that the progress of atrophy of the optic nerve is arrested. In case 7, for example, between January 1932 and February 1936 successive perimetric tests (fig 3) showed an increasing contraction, but no change was evident in the ophthalmoscopic appearance or in the visual acuity. In case 29 the increase in the scotoma in the right eye (fig 6) was accompanied with an increase in pallor, but because the fovea was not involved normal visual acuity was maintained.

(b) *Patients with Defects in the Fields Associated with Normal Optic Nerve Heads*—Perimetric examination of 200 neurosyphilitic patients whose optic disks were normal or showed only a questionable pallor disclosed in 12 cases definite slight defects in the fields which could not be attributed to poor cooperation on the part of the patient or to other complicating factors. The clinical findings in these 12 cases are summarized in table 5.

Six patients had bilateral field defects, and 3 had unilateral defects. In the remaining 3 only one eye was tested, because of complicating factors in the second eye. Three patients showed a questionable pallor of one or both nerve heads, while the eyes of 9 appeared entirely normal on ophthalmoscopic examination.

Four patients (cases 57 to 60) had slight peripheral field defects of type 1. The left field of 1 of these patients (case 57) is shown in figure 19. Five patients (cases 61 to 65) had slight defects of type 2. Figure 20 shows the fields of 2 of these patients. In 1 a nerve fiber bundle defect produced an alteration resembling that of type 4 but differing in that there was no involvement of the cecocentral region. Seven of the 9 patients with slight peripheral defects of type 1 or 2 had normal visual acuity with correction, no refraction was made for the other 2, and the slightly reduced vision, of 20/30, was presumably due to an error of refraction.

Two patients (cases 66 and 67) had slight defects of type 3. One of these had an absolute central scotoma for $\frac{1}{2}$ degree red and blue test objects and vision of 20/30. The other had only a relative scotoma for $\frac{1}{2}$ degree colored objects and 20/20 vision. One patient (case 68) had a field defect of type 4 in the left eye. The right eye was not tested because of incipient cataract. The field of the left eye on three determinations is shown in figure 21.

Comment—Seven of these 12 patients were followed for from three months to five years. Five were examined only once and then

TABLE 5—*Patients with Defects in Fields and Normal Nerve Heads*

Case No. and Date Examined	Observations in Visual Fields	Appearance of Nerve Heads	Corrected Visual Acuity	Type of Syphilis	Progressive Changes in Visual Fields	Comment
57 3/14/31	O D Slight concentric contraction (results variable) O S Slight but reproducible concentric contraction	O U Normal	O D 20/20+3 O S 20/15-3	Diffuse meningo vascular	Followed for 5 yr 9 mo., gradual decrease in fields	O D Ophthalmoplegia interna, perimetric findings variable because of blurring of near vision
11/10/31	O D Not tested	O U Normal				
2/1/36	O S Further contraction					
12/18/36	O S Further slight decrease (fig 19)	O U Normal	O D 20/15 O S 20/15-1	Dementia paralytica	Followed for 2 yr 8 mo., no change	
58	O U Slight concentric contraction for 1° white	O D Questionable pallor	O D 20/15 O S 20/15-1	Tabs	Followed for 9 mo., no change	
59	O U Slight concentric contraction	O U Normal	O D 20/15-1 O S 20/15	Diffuse meningo vascular	Followed for 2 yr 6 mo., fields improved, became worse and improved again in successive tests	
60	O U Slight concentric contraction	O U Questionable pallor of disks	O U 20/15		Followed for only 3 mo., no change	O D Amblyopia, congenital strabismus
61	O D Not tested O S Slight cut in lower nasal field (fig 20)	O U Normal	O D 20/30-2 (amblyopic eye) O S 20/20	Asymptomatic of central nervous system, late	Seen only once	
62	O D Nerve fiber bundle defect, macular region spared (fig 20) O S Normal	O U Questionable pallor of disks	O D 20/20- O S 20/30 (uncorrected refractive error)	T-tubes		
63	O D Normal O S Slight temporal contraction	O U Normal	O D 20/15 O S 20/15	Asymptomatic of central nervous system, late	Seen only once	
64	O U Slight nasal contraction	O U Normal	O D 20/15 O S 20/15	Asymptomatic of central nervous system, late	Seen only once	
65	O D Normal O S Slight temporal contraction	O U Normal	O D 20/20 O S 20/0 (uncorrected)	Diffuse meningo vascular	Seen only once	
66	O U Relative central scotomas for ½° blue and red, normal peripheral fields	O D Normal O S Normal	O D 20/20-1 O S 20/20+3	T-tubes	Followed for 1 yr., no change	
67	O U Absolute central scotomas for ½° blue and red normal peripheral fields	O D Normal O S Normal	O D 20/20 O S 20/0-1	Diffuse meningo vascular	Seen only once	
68 5/5/31	O D Not tested (lens opaque) O S Relative eccentric scotoma for ½° white, concentric peripheral contraction	O S Normal	O S 20/40 (uncorrected)	Diffuse meningo vascular	Followed for 5 yr 8 mo., complete recovery	O D Opacity of lens
11/17/32 2/15/35 to 1/31/36	O S Improvement O S Normal (fig 21)	O S Normal O S Normal	O S 20/15 corrected 20/40 uncorrected			

were lost from observation. Since none of the patients followed has as yet shown unquestioned pallor of the disks, definite proof is lacking that the field defects were due to incipient atrophy. Four other patients in whom primary atrophy later developed but in whom the

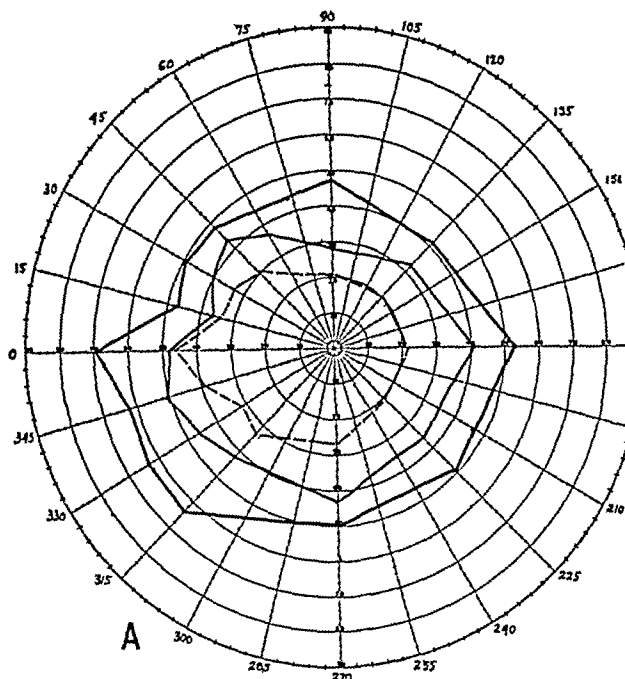


Fig 19 (case 57)—Visual field of the left eye on Dec 18, 1936, showing a defect of type 1. The broken line bounds the field for the 1 degree red test object, the inner solid line, that for the $\frac{1}{2}$ degree white object, and the outer solid line, that for the 1 degree white object.

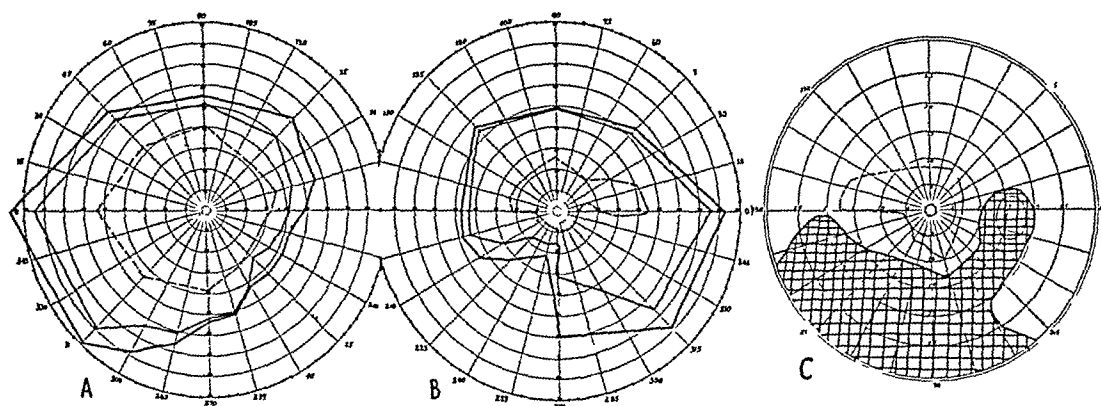


Fig 20—Defects of type 2. The broken line in A (case 61) bounds the visual field of the left eye for a 1 degree red test object, the inner solid line, that for a $\frac{1}{2}$ degree white object, and the outer solid line, that for a 1 degree white object. Visual acuity was 20/20. The lines in B (case 62) bound the right field for the same test objects. Visual acuity was 20/30—(uncorrected). The broken line in C bounds the field for a $\frac{1}{2}$ degree red test object in the same case, and the cross-hatched area represents scotoma for a $\frac{1}{2}$ degree gray test object.

field defects in one or both eyes antedated pallor of the disks have already been reported on under the section dealing with primary atrophy (cases 1, 2, 3 and 52). Since the field defects reported here,

though slight, conform to the types found with established primary atrophy of the optic nerve, it seems justifiable to attribute these early defects to incipient primary atrophy of the optic nerve which is not yet manifest on ophthalmoscopic examination

The outcome in the 7 cases in which prolonged observation was possible was as follows. One patient with a type 1 defect showed a gradual loss in the fields during five years' observation. The vision and the fundi remained normal. Four patients showed no change in fields, fundi or vision during periods ranging from three months to four years. The patient with a type 4 defect in the left eye showed improvement in the field and in vision when retested after six months and complete recovery when retested four and five years after the first examination (fig 21). The nerve head remained normal throughout the period of observation. One patient who had field defects of type

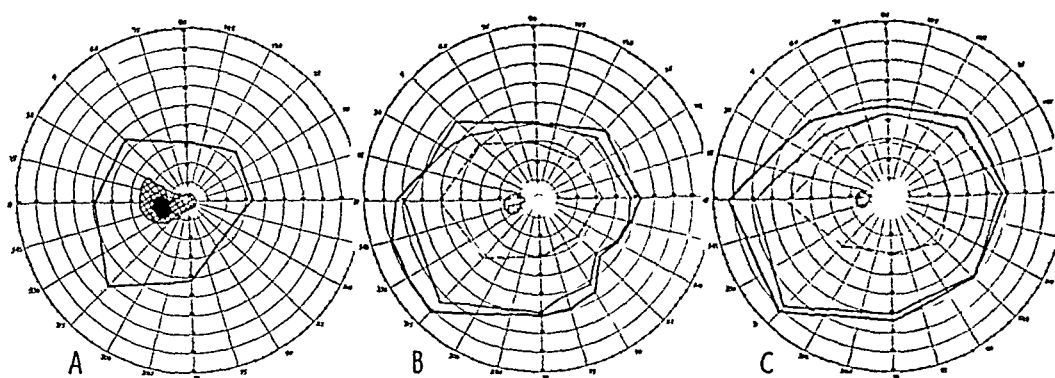


Fig 21 (case 68) —Left visual field on (A) May 5, 1931, (B) Nov 15, 1932, and (C) Feb 15, 1935, showing improvement in a field defect of type 4. At the first examination vision was 20/40. The solid area represents a blindspot for a $\frac{1}{2}$ degree white test object, and the cross-hatched area, relative scotoma for the same object. The field illustrated is that for a 1 degree white test object. At the second examination vision was 20/15. The cross-hatched area represents a blind-spot for a $\frac{1}{2}$ degree red test object, the broken line bounds the field for a 1 degree red test object, the inner solid line that for a $\frac{1}{2}$ degree white test object and the outer solid line that for a 1 degree white test object. At the time of the third examination vision was 20/15. The cross-hatched area represents a blind-spot for a $\frac{1}{2}$ degree red test object. The lines bound fields for the same test objects in C as in B.

1, normal vision and questionable pallor of the disks showed fluctuating changes in the fields, with both increase and decrease in the limits on different examinations. There was no change in the appearance of the nerve heads. The possible effect of antisiphilitic therapy on the progress of the field defects will be considered in a later paper.

Central scotoma was observed in 3 of these 12 patients with defects in the fields and no manifest ophthalmoscopic evidence of atrophy (cases 66, 67 and 68). This is an incidence of only 25 per cent, against one

of over 54 per cent for patients with manifest atrophy. This may indicate that involvement of the papillomacular bundle tends to occur late rather than early in the development of the atrophy.

(c) *Patients with Homonymous Defects in the Fields*—Homonymous defects in the fields were found in 12 of 291 patients with syphilis of the central nervous system. These homonymous defects were partial or complete hemianopia, homonymous quadrantic defects, homonymous scotomas and, in 1 case, double quadrantic defect. Figures 22, 23 and 24 illustrate these changes, and the clinical findings are epitomized in table 6.

Comment—The pertinent questions in these cases of homonymous defects in the fields are: 1. Are such defects to be attributed to lesions involving both optic nerves anterior to the chiasm or to lesions in the tracts or the optic radiations posterior to the chiasm? 2. Are the actual lesions due to syphilitic infection or to some other cause? A study of these 12 cases indicates that the lesions responsible for the visual defects lay posterior to the chiasm and were not definitely related to the syphilitic infection. One patient had a definite medical record of hemiplegia, 2 patients had a record of severe cranial traumatism, and 7 patients had a history indicative of hemiplegia, aphasia or other disease of the central nervous system. These 10 patients showed, variously, normal or questionable pallor of the optic nerves and definite sclerosis of the retinal vessels. In no case could a diagnosis of even early atrophy of the optic nerve be made. The evidence in these 10 cases indicates that the defects in the fields are probably due to a cerebral injury posterior to the chiasm, secondary to either trauma or intracranial hemorrhage.

The 2 remaining patients with homonymous defects in the fields (cases 82 and 83) had no history and showed no evidence of a vascular lesion, a cerebral injury or other complicating factor. Both had Argyll Robertson pupils. One had normal fundi, the other showed advanced primary atrophy of the optic nerve of the right disk and questionable pallor of the left. There was no evidence to indicate the cause of the hemianopia of these 2 patients. However, in view of the fact that there was positive evidence of cerebral damage in 10 patients with similar involvement, it appears probable that intracranial hemorrhage or a localized area of softening affecting the visual pathways was likewise responsible for the hemianopia of these 2 patients. The visible atrophy of the optic nerve of the 1 patient could well have been due to an independent syphilitic process in the optic nerve. That this is the probable explanation is indicated by the visual fields (fig 24). In the right eye, which showed the manifest atrophy, there was evidence of a cecocentral scotoma extending into the right field, in addition to the left hemianopia. Retests six years later showed a slight improvement in the hemianopic

defect in both eyes but an increase in the extent of the cecocentral scotoma in the right eye

(d) *Patients with Active or Inactive Optic Neuritis*—There were 23 patients in this group. The clinical findings are summarized in table

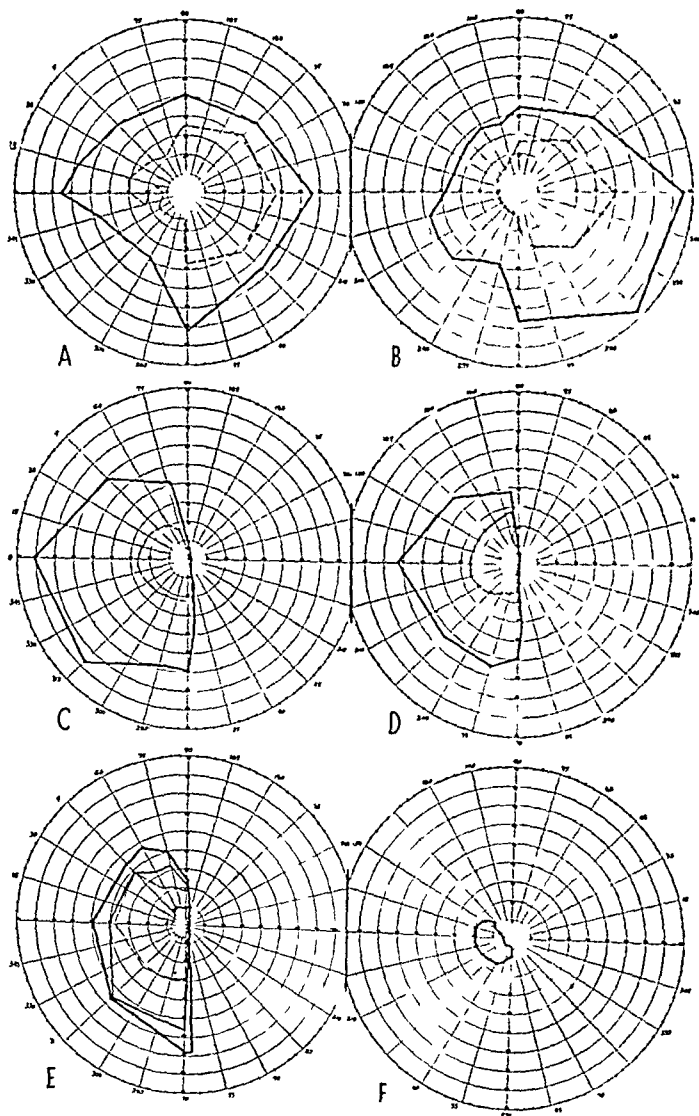


Fig 22—Complete and partial homonymous hemianopia. *A* and *B* (case 69) show the fields for the left and the right eye for 1 degree white (solid line) and 1 degree red (broken line) test objects. Vision for both eyes was 20/15. *C* and *D* (case 70) show the fields for the left and the right eye for 1 degree white (solid line) and 1 degree red (broken line) test objects. Vision for both eyes was 20/30. *E* and *F* (case 71) represent the left and the right visual field, the outer solid line in *E* bounding the field for a 3 degree white, the inner solid line that for a 1 degree white, the outer broken line that for a 1 degree red and the inner broken line that for a $\frac{1}{2}$ degree red test object. The field in *F* is that for a 1 degree white test object. Vision for the left eye was 20/30 + 3 and for the right 10/200.

7 One patient had atrophy of the optic nerve secondary to old optic neuritis and 3 had active optic neuritis. Ophthalmoscopic examination of the other 19 patients showed deposition of glial tissue over the disk, blurred margins or other evidences of old low grade optic neuritis without actual secondary atrophy. The fields of the 3 patients tested during the active stage of optic neuritis are shown in figure 25

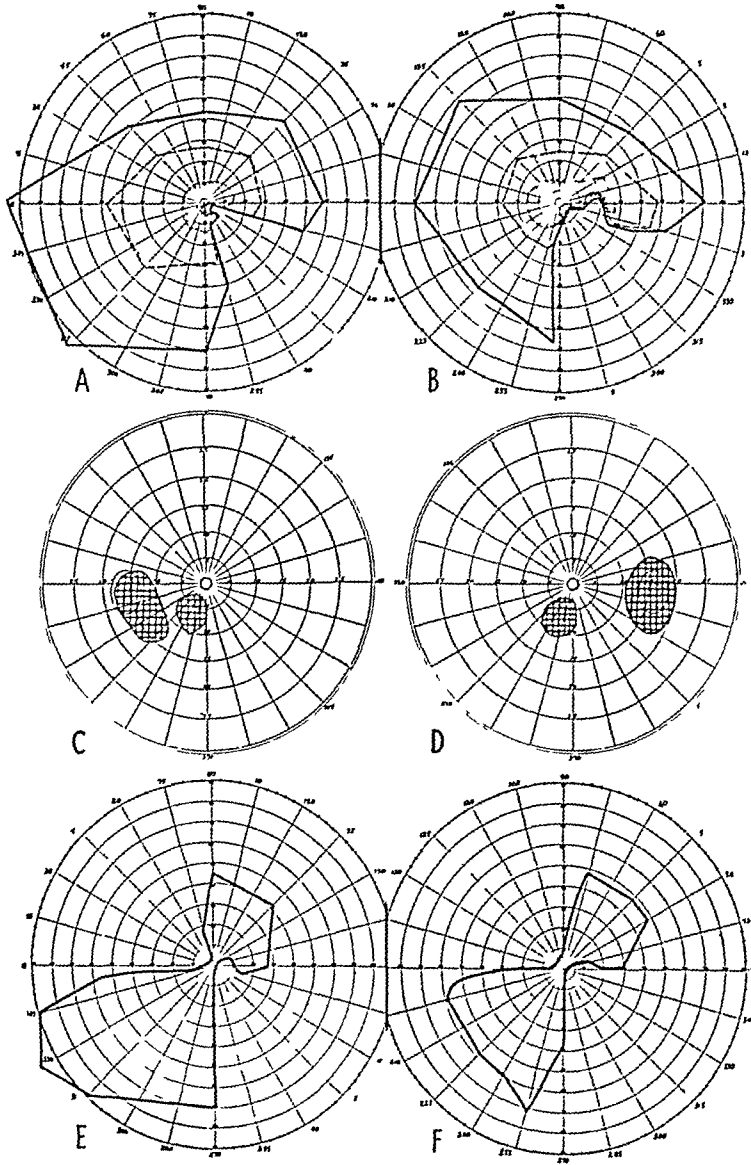


Fig 23—Homonymous quadrantic defects, homonymous scotomas and double quadrantic defects. *A* and *B* (case 72) show the left and the right visual field for 1 degree white (solid line), 1 degree red (outer broken line) and $\frac{1}{2}$ degree red (inner broken line) test objects. Vision for both eyes was 20/15. *C* and *D* (case 73) represent the left and the right eye. Cross-hatched areas represent blindspot and scotoma for a 1 degree blue test object. Vision for both eyes was 20/15. *E* and *F* (case 75) show the left and the right visual field for a 3 degree white test object. Vision for both eyes was 20/40.

In case 81 the field defect consisted solely in an extensive, more or less concentric enlargement of the blindspot, which included the foveal area. The peripheral limits for 1 degree white and red test objects were entirely normal. Retests one year later showed only a slight enlargement of the blindspot and no foveal involvement. In case 82 the peripheral limits for a 1 degree white test object were entirely normal. In both eyes there were enlargement of the blindspot and paracentral scotoma, and in the left eye a large scotoma of the nerve fiber bundle type for a 1 degree blue test object. In case 83 the most marked defect was a nerve fiber bundle scotoma extending from the upper border of

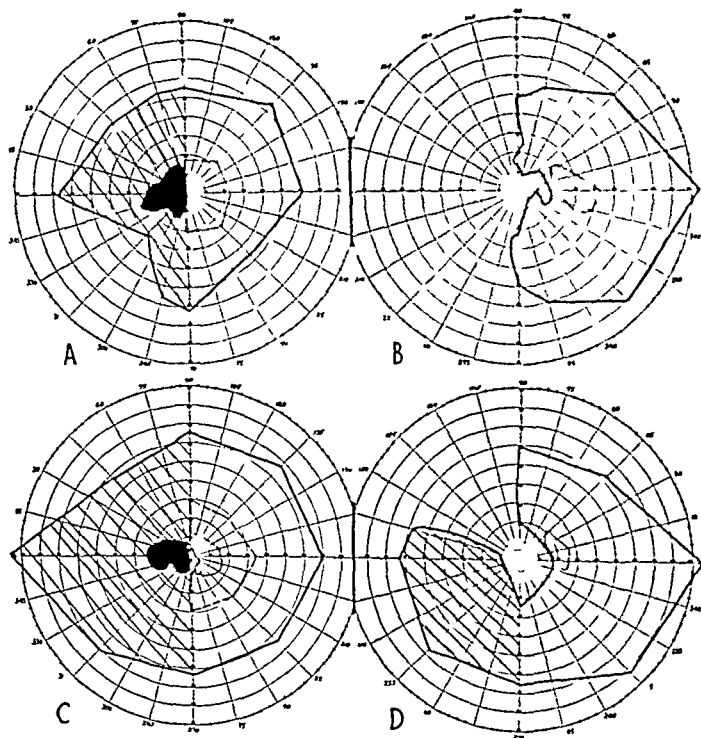


Fig 24 (case 80) —Left homonymous hemianopia, associated with cecentral scotoma of right eye. *A* and *B* show the left and the right visual field on May 12, 1931, *C* and *D* those on Jan 20, 1937. The solid line bounds the field for a 1 degree white test object, the long dashes that for 1 degree red and the short dashes that for a $\frac{1}{2}$ degree red test object. The solid area represents absolute scotoma and the area covered by slanting lines relative scotoma for a 1 degree white test object. Vision in the left eye was 20/20 at the first examination and 20/30 + 3 at the second, that in the right eye was reduced to perception of hand movements at both examinations.

the blindspot. Its location corresponded to the ophthalmoscopic finding of blurring and edema at the lower nasal border of the disk.

The patient whose disks showed definite secondary atrophy (case 84) had advanced field defects consisting of cecentral scotomas and

TABLE 6—Patients with Homonymous Defects in Fields

Case No and Date Examined	Observations in Visual Fields	Results of Ophthalmoscopic Examination	Corrected Visual Acuity	Type of Syphilis	Comment
69 4/11/36	Partial left homonymous hemianopia (fig 22)	O D Normal O S Questionable temporal pallor of nerve head	O D 20/15 O S 20/15	Diffuse meningovascular	Left hemiplegia in 1931
70 1/15/35	Complete right homonymous hemianopia (fig 22)	O U Nerve heads normal, moderate retinal arteriosclerosis	O D 20/30 O S 20/30	Diffuse meningovascular	History of transient paraplegia in March 1931
71	Right homonymous hemianopia with some involvement of left side, especially in field of right eye (fig 22)	O D Questionable temporal pallor of nerve head O S Normal	O D 20/30 O S 20/30	Diffuse meningovascular	Neurologic evidence of bilateral central lesions involving left temporal lobe and right central hemisphere (aphasia, left facial palsy and left hemianesthesia), History of transient blindness, advanced hypertension and cerebral arteriosclerosis
72	Lower right homonymous quadrantic defects (fig 23)	O U Nerve heads normal, retinal arteriosclerosis	O D 10/200 O S 20/30+3	Diffuse meningovascular	Sudden attack of complete blindness and aphasia in 1932, vision returned in 3 days on right side, syphilitic aortitis
73 2/6/33	Homonymous scotoma in lower left and right field (fig 23)	O U Questionable pallor of nerve heads	O D 20/15 O S 20/15	Latent, no evidence of involvement of central nervous system	History of repeated cranial traumatism and cerebral injury, transient attacks of blindness, marked hypertension and arteriosclerosis
74	Right homonymous hemianopia	O D Questionable pallor of nerve head, some connective tissue over cup O S Normal	O D 20/30 O S 20/30 (corneal opacity)	Tertiary cardiovascular	History of complete blindness and aphasia in right leg, central arteriosclerosis, question of multiple sclerosis
75	Upper left and lower right quadrantic defects (fig 23)	O U Nerve heads normal, a little macular degeneration, some arteriosclerosis	O D 20/40 O S 20/40	Latent, no evidence of involvement of central nervous system	History of repeated cranial traumatism and cerebral injury, transient attacks of blindness, marked hypertension and arteriosclerosis
76	Upper left homonymous quadrantic defects	O U Cupping a little blurred, nerve heads otherwise normal	O D 20/30—1 O S 20/50+ refractive error (—8.00 sph and —3.50 cyl)	Dementia praecox	History of preceding cranial injury from a fall, and questionable blood clot
77	Lower right homonymous quadrantic defects	O U Questionable pallor of nerve heads, retinal arteriosclerosis	O D 20/15—2 O S 20/30+3	Asymptomatic late tertiary cardiovascular	Violent injury to head in 1918, roentgenogram shows defects in frontal bone, large irregular sella, chronic aortic insufficiency
78 12/2/11	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of partial paraplegia in 1930, chronic anterior involvement of central nervous system
79	Lower right homonymous quadrantic defects	O U Normal	O D Hand motion O S 20/20	Dementia praecox	No history of cerebral injury, paralysis or other involvement of central nervous system
80 5/12/11	Left homonymous hemianopia, with eccentric scotoma in right eye	O D Advanced primary atrophy of optic nerve mild retinal arteriosclerosis	O D Hand motion O S 20/30+3	Dementia praecox	General arteriosclerosis
81 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
82 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
83 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
84 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
85 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
86 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
87 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
88 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
89 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
90 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
91 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
92 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
93 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
94 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
95 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
96 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
97 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
98 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
99 1/20/17	Upper left homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system
100 1/20/17	Lower right homonymous quadrantic defects	O U Normal	O D 20/15 O S 20/15	Diffuse meningovascular	History of cerebral injury, paralysis or other involvement of central nervous system

TABLE 7—*Patients with Active or Inactive Optic Neuritis*

Case No and Date Examined	Observations in Visual Fields	Corrected Visual Acuity	Type of Syphilis	Results of Ophthalmoscopic Examination
81 6/24/35	O D Marked concentric enlargement of blindspot with relative scotoma extending to fovea O S Not tested (amblyopia ex anopsia)	O D 20/100+1	Of central nervous system, early neuro-recurrence	O D Neuroretinal margins blurred, disks congested and elevated 2 diopters, exudates extending into surrounding retina, a few small flame-like hemorrhages present
6/29/36*	O D Slight enlargement of blindspot, fovea not involved (fig 25)	O D 20/20+2		O D Edges of disk only slightly blurred, disk grayish
82† 2/9/34	O D Scotoma below fovea, not connected with blindspot O S Scotoma in lower nasal quadrant, and nerve fiber bundle scotoma extending from upper border of blindspot	O D 20/40— O S 20/30	Early secondary, cutaneous, with alopecia	
2/16/34	O U Slight improvement (fig 25)	O D 20/40 O S 20/20—2		O U Blurred margins and obliterated cups, no measurable elevation in O D, about 1 diopter in O S
83	O D Not tested (amblyopic) O S Nerve fiber bundle defect extending from upper temporal border of blindspot, general peripheral contraction (fig 25)	O S 20/10	Of central nervous system, early neuro-recurrence	O S Slight pallor of disk, definite edema in lower nasal quadrant, with some edema of adjacent retinal fibers
84	O U Cecentral scotomas and marked peripheral contraction for 1° white (fig 26)	O D 3/200 O S 3/200	Dementia paralytica	O U Secondary atrophy of optic nerves, disks flat and slightly pale, glial tissue in cups and along vessels a short way out from disks, peripheral fundi normal
85	O U Definite peripheral contraction, especially marked in upper field for 1° white, slight defects in upper field of each eye for 1° red, connected with blindspot in left (fig 27)	O D 20/15 O S 20/15	Dementia paralytica	Disks show blurred neuroretinal margins and blurred cups, no activity
86	O U Marked peripheral contraction for 1° white, slight contraction for 1° red, slight enlargement of blindspot (fig 27)	O D 20/15—2 O S 20/15—2	Tabs	Disks show a little blurring of neuroretinal outlines and definite increase in capillarity
87	O U Concentric peripheral contraction for 1° white and color, enlargement of blindspots (fig 27)	O D 20/15—1 O S 20/15	Dementia paralytica	Disks flat and slightly irregular, some perivascular connective tissue on vessels over disk, no activity
88	O U Concentric contraction, marked for 1° white, slight for 1° blue and red, slight enlargement of blindspot in right eye	O D 20/15 O S 20/20—3	Latent, no evidence of involvement of central nervous system	O U Slight hyperemia of disks and blurred cups
89	O U Peripheral contraction, definite for 1° white, questionable for 1° color, blindspots normal	O D 20/15 O S 20/15—1	Tabs	A little fibrosis over cups possibly slight pallor
90	O D Slight contraction for 1° white, questionable contraction for 1° red, blindspot for ½° white enlarged O S Slight contraction for 1° white and red, normal blindspot for ½° white (fig 27)	O D 20/15 O S 20/15	Dementia paralytica	O D Nerve head a little blurred, cup lost O S Nerve head almost normal, cup lost

* Metamorphopsia in region 5° to right of fixation point

† Patient notices constant "flickering" before eyes

TABLE 7—*Patients with Active or Inactive Optic Neuritis*—Continued

Case No and Date Examined	Observations in Visual Fields	Corrected Visual Acuity	Type of Syphilis	Results of Ophthalmoscopic Examination
91† 6/8/32	O U Concentric contraction for 1° white, normal fields for 1° blue and red O S Slight enlargement of blindspot	O D 20/15—3 O S 20/15	Tabes	O U Disks good color, cups obliterated, neuroretinal margins a little blurred
10/26/32	O U Fields normal	O D 20/15 O S 20/15		O U Disks good color, right cup obliterated and left just seen, a little connective tissue along vessels
92	O U Fields for 1° white normal and for 1° colors show questionable contraction O D Blindspot enlarged O S Normal blind spot (fig 28)	O D 20/15 O S 20/15	Early secondary, cutaneous	O D Neuroretinal margins a little blurred, connective tissue in cup, no atrophy O S Slight blurring of cup
93	O D Blindspot for ½° blue and ½° red shows scotomatous extension, fields otherwise normal (fig 28) O S Normal	O D 20/15 O S 20/15	Diffuse meningo vascular	O D Nerve head shows blurred margins, blurred cup, a little connective tissue, is possibly slightly paler than left O S Blurred cup
94	O D Normal peripheral fields for 1° white and color, questionable enlargement of blindspot O S Questionable contraction of the fields for 1° white and color, questionable enlargement of blindspot	O D 20/15—1 O S 20/15—1	Early secondary, recurrent, cutaneous and arsenamine resistant	O U Nerve heads show blurred margins, cups obliterated, connective tissue formation along vessels, no activity
95	O D Normal O S Field for 1° white shows slight contraction, field for 1° blue and red, normal, blindspot normal	O D 20/15—1 O S 20/15—2	Diffuse meningovascular and tertiary osseous, with osteitis of skull	O U Disks flat, of good color, a little fibrosis over cups
96	O D Normal O S Normal except for questionable defect in field for 1° red	O D 20/15 O S 20/15	Tabes	O U Disks flat, of good color, a little fibrosis over cups
97	O D Normal O S Questionable contraction of fields for 1° white, blue and red, blindspot normal	O D 20/15—3 O S 20/15—2	Dementia paralytica	O U Fundi a little hyperemic and cups indistinct otherwise normal
98	O U Field for 1° white normal, slight contraction for 1° red, questionable enlargement of blindspot	O D 20/15—2 O S 20/15—2	Dementia paralytica, at autopsy small hypophyseal adenoma	O U Glial tissue formation over disks
99	O D Normal O S Scotomatous enlargement of blindspot for ½° color, otherwise normal	O D 20/15—3 O S 20/15—3	Dementia paralytica	O U Disks flat, of good color, cupping a little indistinct
100	O U Normal	O D 20/15—2 O S 20/15—2	Asymptomatic of central nervous system	O U Disks flat, of good color, cupping blurred
101	O U Normal	O D 20/15 O S 20/15—1	Dementia paralytica	O U Disks flat, of good color, cupping slightly blurred
102	O U Normal	O D 20/20 O S 20/20	Diffuse meningo vascular	O U Disks flat, of good color, cupping slightly blurred
103	O U Normal	O D 20/20 O S 20/15	Dementia paralytica	O U Disks a little vascular cups blurred but of good color

† Subarachnoid hemorrhages and choked disk in January 1932

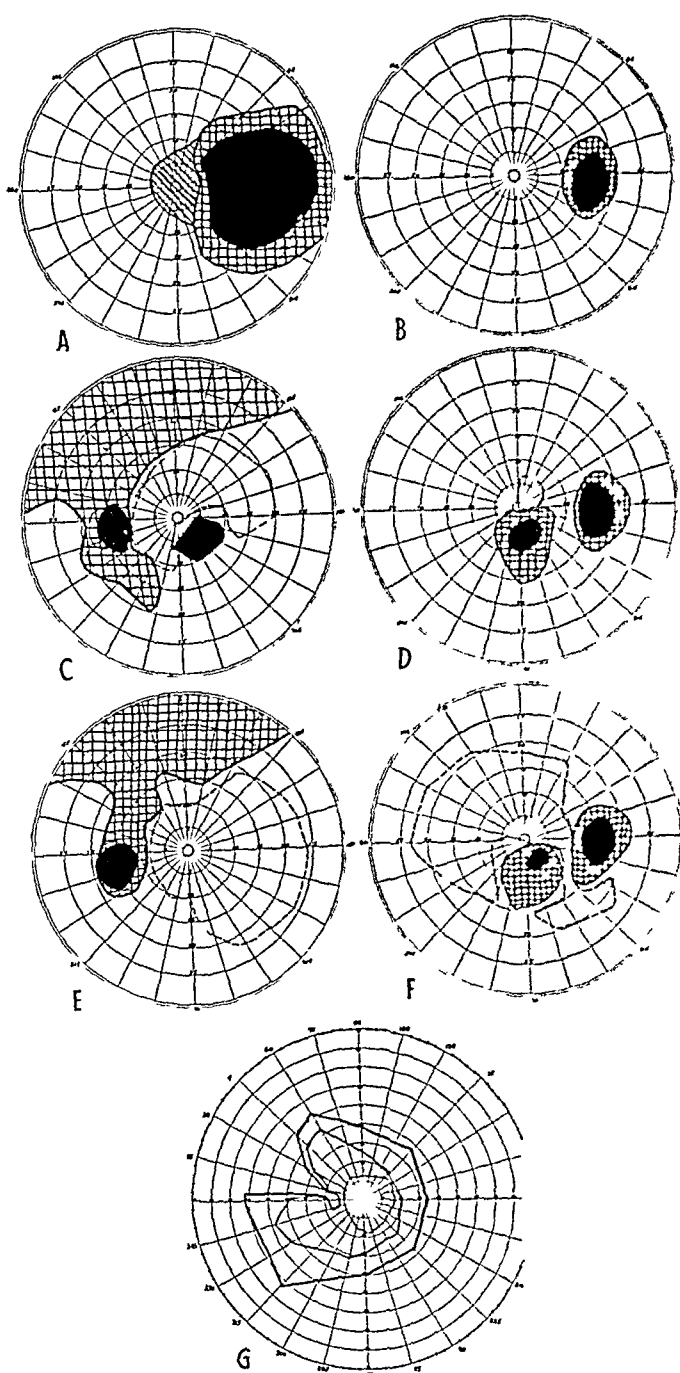


Fig 25—Visual fields in cases of active optic neuritis *A* and *B* (case 81) represent the right eye on June 24, 1935, and June 29, 1936, respectively. In *A*, the solid area represents the blindspot for a 1 degree white test object, the cross-hatched area, that for a 1 degree red object and the area covered by slanting lines, relative scotoma for 1 degree white and red objects. Vision was 20/50. In *B* the solid area represents the blindspot for a $\frac{1}{2}$ degree white test object and the cross-hatched area that for a $\frac{1}{2}$ degree red test object. Vision was 20/20 + 2. The peripheral field for white and colored test objects was normal at both determinations. *C*, *D*, *E* and *F* (case 82) represent the fields of the left and the right eye on Feb 9 and on Feb 16, 1934. A solid area represents blindspot or scotoma for a $\frac{1}{2}$ degree white test object and a cross-hatched area blindspot or scotoma for a 1 degree blue test object. The broken line represents the boundary of the field for a 1 degree blue test object. At the first determination vision for the left eye was 20/30 and for the right 20/40— At the second determination vision for the left eye was 20/20 — 2 and for the right 20/40. The peripheral field for a 1 degree white test object was normal in both case 81 and case 82. *G* (case 83) shows the left visual field for 3 degree white (outer solid line), 1 degree white (inner solid line) and 1 degree blue (broken line) test objects. Vision was 20/40.

marked peripheral contraction for a 1 degree white test object. The visual acuity of each eye was reduced to 3/200 (fig 26). The 19 patients who had changes in the fundi indicative of previous low grade optic neuritis which was inactive at the time of examination

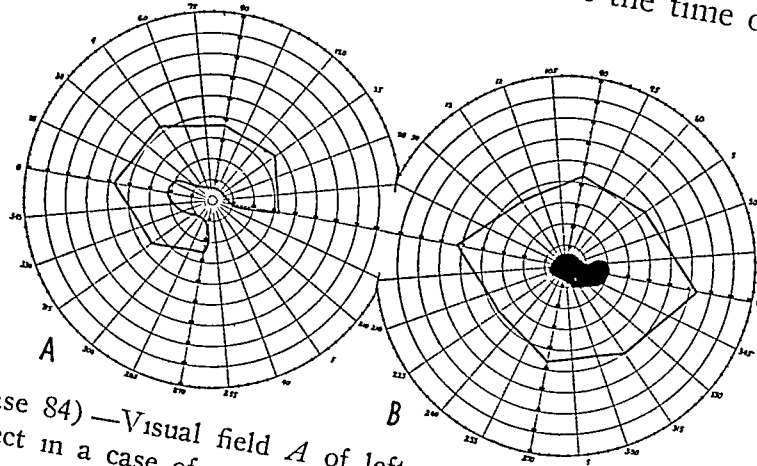


Fig 26 (case 84)—Visual field *A* of left and *B* of right eye for a 1 degree white test object in a case of secondary atrophy of the optic nerve. Vision was 3/200 for both eyes. The solid area represents scotoma for a 1 degree white test object.

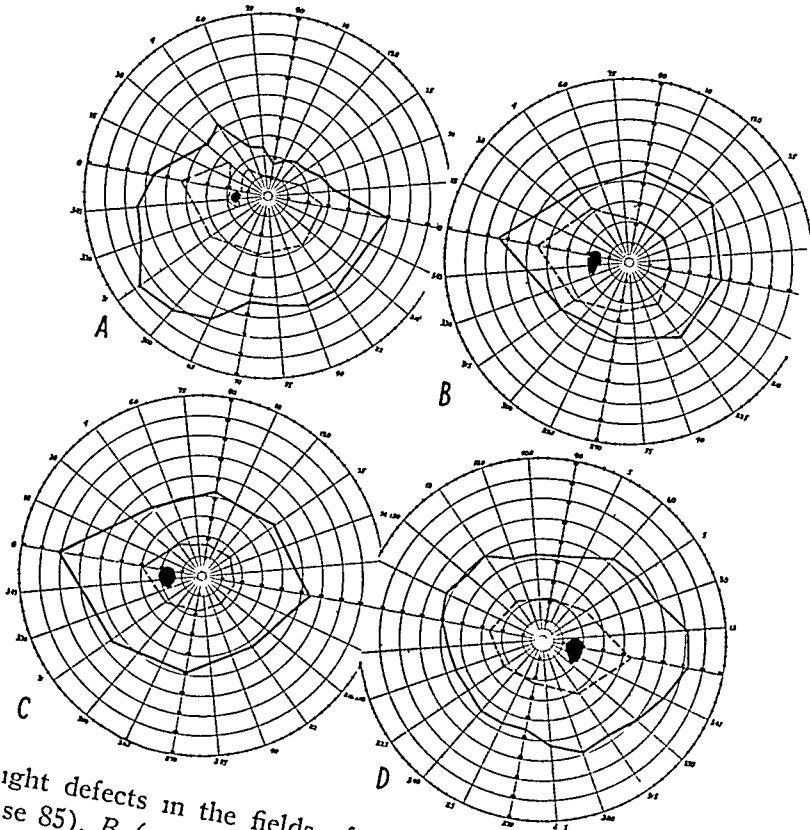


Fig 27—Slight defects in the fields of patients with inactive low grade optic neuritis. *A* (case 85), *B* (case 86) and *C* (case 87) show the left visual field and *D* (case 90) the right visual field for 1 degree white (solid line) and 1 degree red (broken line) test objects. The solid area in each illustration represents the blindspot for a $\frac{1}{2}$ degree white test object. In *A*, *C* and *D*, vision was 20/15, in *B*, 20/15 — 2.

had visual acuity of 20/20 or better. The perimetric findings included slight but definite field defects, questionable defects and definitely normal fields. Seven patients had definite defects, such as are illustrated in figure 27. These consisted in localized or concentric contraction of the peripheral limits for white, for colored or for both white and colored test objects and slight defects connected with the blindspot, giving either concentric enlargement or nerve fiber defects extending from the upper or the lower border. No other types of scotoma were found. The fields of 8 patients showed only questionable deviations from normal, either in the peripheral limits for white or colored test objects or in the size of the blindspot. (Such borderline defects are illustrated in figure 28.) Four patients had definitely normal fields for 1 degree white, blue and red test objects and normal blindspots for $\frac{1}{2}$ degree white, blue and red objects.

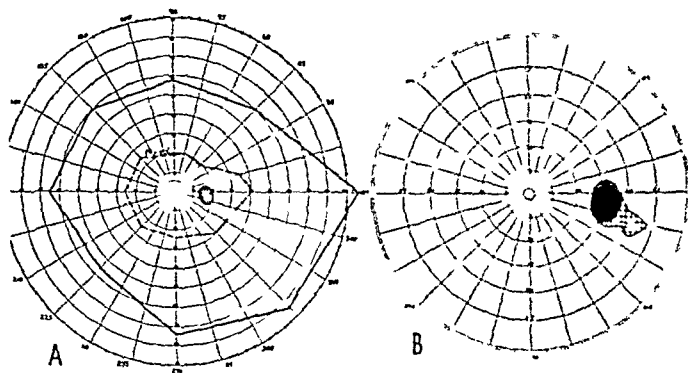


Fig 28—Questionable defects in the fields of patients with inactive low grade optic neuritis. *A* (case 92) shows the right visual field for 1 degree red (broken line) and 1 degree white (solid line) test objects. The solid area represents a blindspot for a $\frac{1}{2}$ degree white test object. Vision was 20/15. In *B* (case 93) the solid area represents a blindspot for a $\frac{1}{2}$ degree white test object and the cross-hatched area one for a $\frac{1}{2}$ degree blue object in the right field of a patient whose peripheral fields for white and colored test objects were normal. Vision was 20/15.

Comment—The fields of the 3 patients with active optic neuritis showed, variously, enlargement of the blindspot, central and paracentral scotomas and nerve bundle defects. The patient who had definite secondary atrophy after the neuritis subsided showed concentric contraction and nerve bundle defects with scotomas, while the 19 patients who recovered without secondary atrophy had normal vision and either normal fields or only slight defects.

On the basis of this knowledge it appears that the initial field change in cases of optic neuritis may be only an enlargement of the blindspot. If the inflammation of the optic nerve is sufficiently severe there may be nerve bundle defects and central or paracentral scotoma,

and the changes may resemble those in cases of primary atrophy of the optic nerve. In patients with severe neuritis, secondary atrophy of the nerve may ensue and these changes in the field become permanent. However, in the majority of the patients the inflammation in the nerve subsides and leaves no serious sequelae, vision is normal and the visual fields either are normal or have defects limited to enlargement of the blindspot and slight changes for white or color. Enlargement of the blindspot and a normal field for white associated with a concentrically contracted field for color were not found in cases of primary atrophy of the optic nerve and appear characteristic of old optic neuritis.

GENERAL COMMENT

In the light of these studies on syphilitic patients with primary atrophy of the optic nerve, with normal fundi and field defects, with homonymous field defects and with evidences of active or inactive optic neuritis, there are certain obvious general conclusions.

It appears that the conclusions of Uhthoff, which have dominated the thought of ophthalmologists on field changes incident to primary syphilitic atrophy of the optic nerve for over thirty years, are incorrect. Central or cecocentral scotoma, far from being uncommon, occurs in over 50 per cent of cases. The view that such scotoma is the result of an involvement of the exposed optic tracts at the base of the brain by a basilar syphilitic meningitis also appears to be in error. Such central scotoma has been observed with equal frequency in patients with parenchymatous neurosyphilis and in patients with diffuse meningo-vascular syphilis.

It is obviously preferable to demonstrate the position of a given lesion from histologic evidence rather than to postulate its position from clinical evidence. Nevertheless, in the absence of histologic material, the clinical evidence here presented appears to give definite information relative to the location in the optic neuro-pathways of the lesion responsible for the field defects.

The absence of homonymous defects in the fields in any of the 56 patients with frank primary syphilitic atrophy of the optic nerve makes untenable the view that the primary lesion responsible for the atrophy is posterior to the chiasm. Likewise, the finding of cerebral trauma or of arteriosclerosis with vascular accident in 84 per cent of the syphilitic patients with homonymous defects in the fields is evidence that when homonymous defects are present in syphilitic patients they are probably due to those causes rather than to degenerative syphilitic lesions involving the tracts, the geniculate bodies or the optic radiations. Therefore, the primary lesion responsible for such a defect must lie in the chiasm or in the optic nerve.

When first considered, the tendency toward similar or heteronymous defects in the two eyes would appear to be evidence in favor of a chiasmal lesion. Otherwise it would be necessary to assume lesions occupying similar positions in the two nerves to explain this peculiar and characteristic finding. The assumption of a lesion in the periphery of the chiasm might account for such symmetric changes as altitudinal hemianopia and bitemporal defects. A widespread involvement of the entire periphery of the chiasm might account for concentrically contracted fields in both eyes. However, the heteronymous defects in the fields in cases of syphilitic primary atrophy of the optic nerve do not in general show any resemblance to changes in the visual fields produced by other known chiasmal lesions. Binasal defects associated with normal temporal fields, as in case 9 (fig 4), could be produced only by two separate chiasmal lesions and type 3 defects (bilateral cecocentral scotomas and normal peripheral fields) are likewise difficult to explain on the basis of a chiasmal lesion. Fields such as those in case 48 (fig 13), with definite nasal steps, could not be produced by a lesion at the chiasm. Consequently, aside from the fact that such a location does not explain bilateral symmetry in the fields, the findings argue for lesions located in the optic nerves rather than in the chiasm.

If the view that the fibers supplying the outer regions of the visual field have a peripheral location in the nerve is accepted, the various forms of field defect can be explained on the basis of a lesion starting somewhere in the periphery of the optic nerve. A diffuse lesion affecting the entire circumference of the nerve would produce concentric peripheral contraction of the visual field. If the lesion were in the foraminal or the intracranial portion of the nerve, the cecocentral area of the field would be involved late, owing to the axial location of the papillomacular bundle. A defect of type 1 would consequently be expected as a result of a lesion in the posterior portion of the nerve. If, however, the lesion were located near the papilla, where the papillomacular bundle has a peripheral location, a diffuse perineural lesion would produce a cecocentral scotoma in addition to the peripheral contraction. Isolated cecocentral scotoma, nasal steps and combinations of these two defects would result from a lesion in the anterior portion of the nerve involving the papillomacular bundle, the fibers contiguous to it or both. Other localized defects in the visual fields would result from a more or less circumscribed lesion in either the anterior or the posterior part of the optic nerve.

It is difficult to explain the peculiar and almost characteristic similarity of the defects in the fields of the two eyes in cases of primary atrophy. As already pointed out, altitudinal and bitemporal defects may possibly be due to a lesion starting in the chiasm, but to explain

the great majority of these defects it appears necessary to assume that similar lesions are present in the two optic nerves

The observation that field defects for colored and for small white objects may either regress or completely disappear argues in favor of Stargardt's conclusion that the primary lesion in cases of syphilitic atrophy of the optic nerve is a peripheral and interstitial neuritis and that the actual degeneration of the nerve fibers is secondary to this inflammatory change. If antisyphilitic therapy can be instituted before the nerve fibers are damaged beyond repair, there is reasonable hope that the peripheral and interstitial neuritis may be controlled and vision and field defects clear without permanent damage. This is in line with Moore's contention that antisyphilitic therapy in cases of atrophy of the optic nerve offers hope only when instituted before vision has fallen to any great degree.

SUMMARY

The field defects in a group of 56 patients with primary syphilitic atrophy of the optic nerve were of four separate types: (1) concentric contraction of the peripheral field associated with late loss of vision, (2) sector-shaped, or nerve bundle, defects, with which loss of vision might be early or late, depending on the involvement of the papillomacular bundle, (3) central or cecocentral scotoma with normal peripheral fields, associated with early loss of visual acuity and (4) central or cecocentral scotoma with defects in the peripheral fields, also associated with early loss of vision.

Central or cecocentral scotoma was found in 53 per cent of cases of primary atrophy of the optic nerve.

A separate group of neurosyphilitic patients showed defects in the visual fields and normal optic nerves. A study of these patients and of patients with frank syphilitic atrophy of the optic nerve indicates that perimetric changes may antedate either visual failure or pallor of the disk in the development of syphilitic atrophy of the optic nerve.

Homonymous defects in the visual fields when present in patients with neurosyphilis appear to be dependent on such factors as cerebral trauma and arteriosclerosis with vascular accident, rather than on syphilitic involvement of the optic tracts or radiations.

The defects in the visual fields in cases of syphilitic optic neuritis may simulate the changes in cases of primary syphilitic atrophy. There is usually a tendency to almost complete recovery, with no residuum other than enlargement of the blindspot or a minor peripheral defect.

The clinical evidence from these studies indicates that the site of the lesion responsible for syphilitic atrophy of the optic nerve and visual field defects lies in the optic nerve rather than in the chiasm or posterior to it and is probably a peripheral and interstitial neuritis associated with secondary degeneration of the nerve fibers.

CANCER OF THE EYELIDS, CONJUNCTIVA AND CORNEA

II SQUAMOUS CELL EPITHELIOMA

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The purpose of this report is to emphasize the importance of squamous cell epithelioma of the eyelids, conjunctiva and cornea. The material is taken from a series of 464 cases¹ of carcinoma of all types which involved the eyelids and the conjunctiva. A few cases of squamous cell epithelioma of the cornea are included.

Histologic examination was performed in all cases except those in which treatment consisted entirely of radium therapy and in which, consequently, biopsy was not performed. As the basis for the histologic portion of the study, 230 cases were chosen. In 59, or approximately 25 per cent of the histologic material, the growths were squamous cell epitheliomas. Basal cell epithelioma and mixed basal cell and squamous cell epithelioma composed 75 per cent of the 230 tumors and have been considered elsewhere.²

OCCURRENCE

Squamous cell epithelioma may involve any portion of the eyelids, conjunctiva or cornea. The area most frequently involved is the lower eyelid (fig 1). All types of epithelioma occur more often on the lower lid than elsewhere about the eye, but squamous cell lesions occur less frequently on the lower eyelid than do any other types of epithelioma (fig 1).

The distribution of squamous cell lesions about the eye differs from that of other types of epithelioma in that the conjunctival surfaces are

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Abridgment of a thesis submitted to the faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Ophthalmology.

1 Birge, H. L. Epithelioma of the Eyelids, Conjunctiva and Cornea, thesis submitted to the faculty of the Graduate School of the University of Minnesota, 1937.

2 Birge, H. L. Cancer of the Eyelids. I. Basal Cell and Mixed Basal Cell and Squamous Cell Epithelioma, Arch. Ophth. **19** 700-708 (May) 1938.

so frequently involved (fig 1) Approximately 37 per cent of the squamous cell lesions in this series were removed from the conjunctiva, the cornea or the junction of the conjunctiva and the cornea, the limbus

In approximately 20 per cent of the cases of squamous cell epithelioma in which the histologic grade of malignancy was determined the lesion occurred on the conjunctival surface of a lid or the globe The corneal limbus was involved in slightly more than 10 per cent of the cases The cornea alone was involved by squamous cell lesions in approximately 5 per cent of this group Primary corneal malignant growths are rare, and cases of this sort will be dealt with in detail subsequently

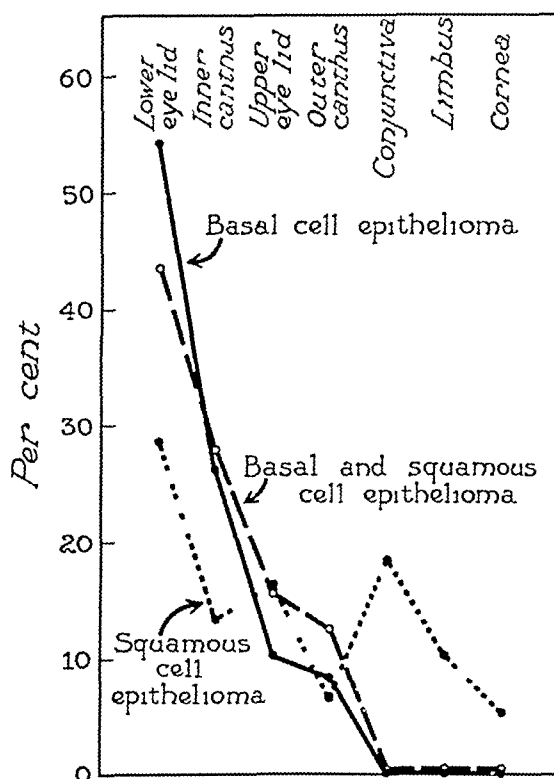


Fig 1—Chart showing the relative frequency of the situation of the various types of epithelioma of the eyelids and adnexa in a series of 230 cases

HISTOLOGIC GRADE OF MALIGNANCY

The pathologic material was graded according to Broders' method (table 1) In this series squamous cell lesions of grades 2 and 3 occurred most frequently The most highly malignant lesions, those of grade 4 were least common

ETIOLOGY

Squamous cell epithelioma is known to occur frequently at the junction of two different types of epithelium³ There are two places about

³ Benedict, W L., and Knight-Asbury, M The Treatment of Malignant Lesions of the Eyelids, New York State J Med 29 675-677 (June 1) 1929

the eye where the epithelium undergoes a change in type. One is at the junction of the conjunctiva and the skin of the eyelid, the other, at the junction of the conjunctiva and the cornea.

A factor of considerable importance in the causation of epitheliomas in general is chronic irritation. In this group the squamous cell lesions were associated with a history of chronic irritation and trauma in nearly the same percentage of cases as were other types of epithelioma reviewed in the entire series.¹ Of the patients with squamous cell epithelioma, 32.2 per cent gave a history which supported the belief that the lesion arose as a result of chronic irritation or trauma. Among the factors listed as being associated with the development of a squamous cell lesion were warts, moles and chalazions. Some patients stated that squeezing a pimple on a lid resulted in the growth. Others stated

TABLE 1—*Grade and Situation About the Eye of Fifty-Nine Squamous Cell Epitheliomas*

Grade	Con junc tiva	Cornea	Limbus	Outer Can thus	Inner Can thus	Upper Lid	Lower Lid	Total
1	1		2		1	1	3	8
2	6	1	4		3	3	4	21
3	1	2		4	2	3	6	18
4	2					2	2	6
Not graded	1				2	1	2	6
Total in various situations	11	3	6	4	8	10	17	59

that being hit in the eye with a cow's tail or getting the lid scratched by a thorn or a finger nail led to the occurrence of the growth. One man was struck by lightning, and shortly thereafter a tumor of the eyelids developed. A few felt that pressure of long standing exerted by glasses on the side of the nose caused the lesion. Chronic blepharitis or meibomitis of long standing was associated with squamous cell epithelioma of the eyelids in 1 case.

Correlation did not occur between the type of chronic irritation and the type or grade of epithelioma. Most of the men were farmers and were exposed only to the elements. Lane⁴ and Kennaway⁵ have dealt with the occurrence of malignant lesions of the lids in workers exposed to carcinogenic agents, such as pitch and tar. Apparently cancer of the eyelids develops less frequently in workers who come in contact with carcinogenic agents than in persons exposed to the ordinary types of

⁴ Lane, L. A. An Occupational Study of Cancer of the Eye and Adnexa, Surg, Gynec & Obst **64** 458-464 (Feb 15) 1937

⁵ Kennaway, E. L. The Anatomical Distribution of the Occupational Cancers, J Indust Hyg **7** 69-93 (Feb.) 1925

chronic irritation and trauma of everyday life. This should not minimize the importance of tar and allied chemicals as carcinogenic agents but merely shows that in a group of patients in whom malignant growths of the eye and eyelids developed many other types of irritation were present.

Inadequate treatment of a benign lesion of the eyelids may have a bearing on the development of a malignant growth. Many of the patients in this series had been treated with various salves, roentgen irradiation and the electric needle before the growths were excised. Treatment that does not completely destroy a lesion produces a reaction of inflammation in the lesion which may be a carcinogenic factor if continued long enough. Chalazions which have become malignant under treatment have been reported.⁶ It does no harm to take specimens for biopsy from recurrent chalazions. Sometimes the first sign of recurrence of such tumors is metastasis to the parotid or submaxillary glands.⁷

Age is an etiologic factor in squamous cell epithelioma, as it is in all types of malignant growths. Most of the squamous cell lesions in this series occurred in the fifth decade of life, one decade earlier than that in which the basal cell lesions occurred.² The youngest person in this series to have a squamous cell epithelioma was a girl 3½ years of age. None of the other types of epithelioma occurred at as young an age.

Sex also may be an etiologic factor in squamous cell lesions. Of the patients who had such lesions, 76 per cent were men.

TREATMENT

Treatment in the 53 cases of squamous cell epithelioma reviewed here in which the malignancy of the growths was graded was about equally divided between surgical operation alone and operation in combination with irradiation (table 2). None of the cases in which treatment consisted of irradiation alone is considered. Treatment of lesions of the eyelids by irradiation alone usually means that treatment is given without knowledge of the pathologic diagnosis.

The surgical measures employed were excision, cauterization, fulguration and plastic repair, as indicated. In 1 case only was simple

6 DeJean, C., Harant, and Vernières. Pseudo-tumeur récidivant de la paupière à cellules géantes sarcoplasmiques, *Arch Soc d sc méd et biol de Montpellier* **15** 240-244 (May) 1934, abstracted, *Am J Cancer* **24** 167 (May) 1935. Kiewe, P. Ueber eine rezidivierende, unter dem Bilde eines Chalazions auftretende Geschwulst der Lider (Zur Frage eines entzündlichen Pseudotumors der Lider), *Acta ophth* **13** 139-149, 1935.

7 Hagedoorn, A. Adenocarcinoma of a Meibomian Gland, *Arch Ophth* **12** 850-867 (Dec) 1934.

enucleation required, that was a case of a malignant lesion of the limbus, grade 2. Exenteration of the contents of the orbit was required with increasing frequency as the histologic grade of malignancy increased (table 2). Exenteration was performed in 25 per cent more cases in which the lesions were of grade 3 than in cases in which they were of grade 2, and exenteration was performed in 60 per cent more cases in which the lesions were of grade 4 than in cases in which they were of grade 3.

RESULTS

The malignancy of squamous cell epitheliomas as a group, judged by the percentage of mortality, is greater than that of any or of all other

TABLE 2—*Behavior of Squamous Cell Epitheliomas on the Basis of Histologic Grading*

Grade	No of Patients		Died of Ocular Cancer		Recur- rences		Invasion of Orbit		Treatment, No of Patients		Blind in Affected Eye		Exenter- ation	
	Total	Traced	No of Patients	Percentage	No of Patients	Percentage	No of Patients	Percentage	Surgical Treat- ment Alone	Surgical Treat- ment and Irradiation	No of Patients	Percentage	No of Patients	Percentage
1	8	6	0	0	0	0	0	0	5	3	0	0	0	0
2	21	20	3	15.0	10	50.0	6	30.0	8	13	5	25.0	3	15.0
3	18	15	7	46.7	14	93.3	9	60.0	9	9	8	53.3	6	40.0
4	6	5	4	80.0	3	60.0	5	100.0	4	2	5	100.0	5	100.0
Ungraded growths	6													

types of epithelioma (except melanotic epithelioma) found on the eye or the adnexa. The correlation between the clinical behavior and the grade of histologic malignancy was close in the series reviewed here. All squamous cell lesions are not of equal malignancy. A follow-up study of patients, in some instances for more than fifteen years, demonstrated that the histologic grading of the malignancy bears a close relation to the clinical course over a period of years.

Squamous cell lesions of grade 1 of the eye or eyelids did not cause any deaths in this series, or the loss of any eyes. There were no recurrences after treatment, nor was there invasion into the orbit. Cure was effected in all cases by surgical excision (table 2).

Squamous cell lesions of grade 2 were the most frequent type of lesion in this series (table 1). They caused loss of the affected eye in 25 per cent of the cases and were responsible for the death of 15 per cent of the persons affected. Recurrences were frequent, and invasion of the orbit occurred in 30 per cent of the cases (table 2).

Squamous cell lesions of grade 3 were next in frequency of occurrence (table 2). They caused loss of the affected eye in 53 per cent of the cases and death in 46 per cent. There were recurrences in nearly all cases following all types of treatment. This includes cases in which treatment consisted of unacceptable remedies as well as those in which treatment was surgical. Apparently, satisfactory initial treatment of lesions of grade 3 must be more vigorous. In 60 per cent of these cases the growth invaded the orbit. In 40 per cent, exenteration of the contents of the orbit was required.

Squamous cell lesions of grade 4 of the eye or adnexa occurred with least frequency of all grades of epithelioma (table 1), yet this type of lesion accounted for more deaths than did lesions of grade 2. There was invasion of the orbit in every case, followed by exenteration, and in 80 per cent of the cases the lesion proved fatal (table 2).

The behavior of lesions of grade 4 in no way resembled that of lesions of grade 1 (fig. 2). None of the persons with lesions of grade 4 survived five years, although 75 per cent were living three years after treatment at the clinic (table 3). Persons with lesions of grade 3 demonstrated the same percentage of survival after three years as did those who had lesions of grade 4. After the first ten years, 100 per cent of persons who had lesions of grade 3 were free from the effects of carcinoma. This fact may be due to the small number of cases, as it probably is, but it supports the idea that if a person who has a malignant lesion survives ten years his chances of dying of cancer are materially reduced.

When the period of survival of persons who have lesions of grade 2 is considered, it is seen that a relatively high percentage survived five years or more (table 3), but that during the next five years many persons succumbed to the effects of the epithelioma. The majority of patients in this group died in the second five years. None of the patients who had squamous cell growths of grade 2 lived fifteen years, those with malignant growths of grade 3 proved an exception (fig. 2).

While the survival periods of persons with lesions of various grades of malignancy yield some interesting figures, a more graphic picture of the relative malignancy of the various grades is obtained from figure 3. This graph is based on the data in table 2 and, in addition, shows the malignancy of the other types of epithelioma, namely, basal cell epithelioma and mixed basal cell and squamous cell epithelioma of the eye or eyelids. It is easy to see that (fig. 3) although squamous cell epithelioma may be more malignant than all other types of epithelioma about the eye (except melanotic epithelioma) this evidence of greater malignancy is due to the behavior of growths of grades 3 and 4.

TABLE 3—*Survival Period of Patients with Squamous Cell Epithelioma on the Basis of Histologic Grading*

Grade	Total Cases	Lived 3 or More Years After Operation		Lived 5 or More Years After Operation		Lived 10 or More Years After Operation		Lived 15 or More Years After Operation	
		No of Patients Operated On*	No of Patients Traced	No of Patients Operated On*	No of Patients Traced	No of Patients Operated On*	No of Patients Traced	No of Patients Operated On*	No of Patients Traced
1	8	7	6	6	100	5	4	4	100
2	21	19	16	14	87.5	14	11	9	81.8
3	18	16	12	9	75	15	12	7	58.3
4	6	4	4	3	75	3	3	0	0
Not graded	6	6	4	3	75	6	4	1	25
Entire series	59	52	42	35	83.3	43	34	21	61.8

* The three year group comprises the patients operated on three or more years prior to the time of inquiry, the five year group, those operated on five or more years prior to the time of inquiry, the ten year group, those operated on ten or more years prior to the time of inquiry and the fifteen year group, those operated on fifteen or more years prior to the time of inquiry

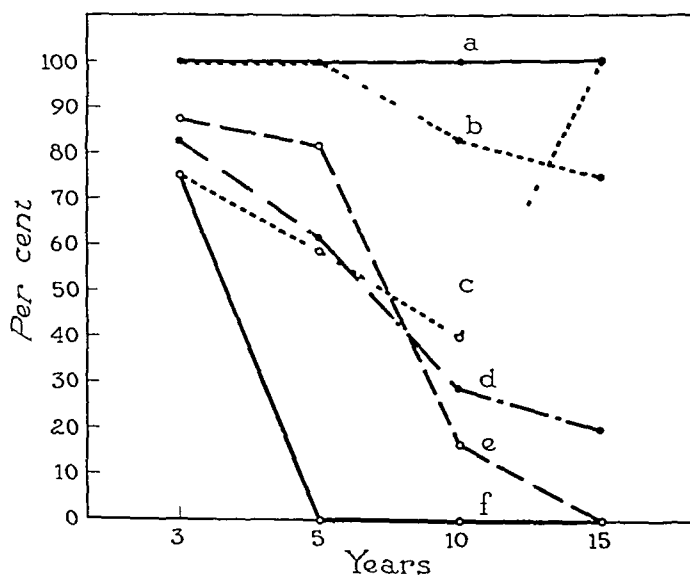


Fig 2—Chart showing the percentage of three, five, ten and fifteen year cures of epitheliomas of the eyelids, conjunctiva and cornea of various grades and types in a series of 230 cases a, percentage of cures in cases of squamous cell epithelioma of grade 1, b, in cases of basal and squamous cell epithelioma, c, in cases of squamous cell epithelioma of grade 3, d, in cases of basal cell epithelioma, e, in cases of squamous cell epithelioma of grade 2, and f, in cases of squamous cell epithelioma of grade 4 Birge¹²

COMMENT

Epitheliomas of the eyelids compose probably from 3 to 5 per cent⁸ of the epitheliomas which occur on the body in general. Of these, roughly 25 per cent are of the squamous cell variety. The squamous cell epitheliomas that account for the greatest mortality are those graded 3 and 4, and these make up slightly less than 50 per cent of all the squamous cell epitheliomas of the eye and the eyelid. In order to reduce the mortality from squamous cell epithelioma, attention must be centered on the lesions of grades 3 and 4, which are not often seen. Even with the most vigorous treatment, namely, exenteration, few of the highly malignant lesions (table 3) allow a person to survive long

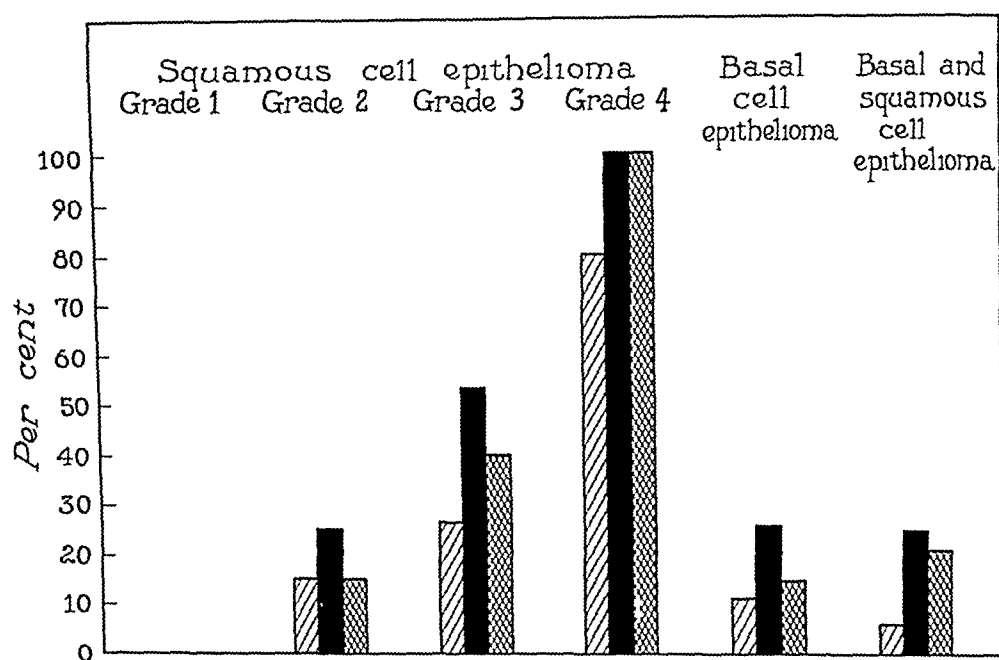


Fig 3—Chart showing the relative malignancy of graded squamous cell epitheliomas and other epitheliomas found on the eyelids and external coverings of the eyeball in a series of 230 pathologically classified tumors. The diagonal lines indicate the number of patients who died of cancer of the eye, the solid area, those who became blind as a result of the lesion of the eye, and the crossed lines, those who underwent exenteration.

It would seem that earlier exenteration of growths graded 3 and 4 might offer a means of prolonging the life of persons affected.

Primarily, in all cases in which a tumor of the eye or eyelids is suspected, histologic examination must be made. Secondly, attention

⁸ Geschickter, C. F., and Koehler, H. P. Ectodermal Tumors of the Skin, *Am J Cancer* **23**: 804-836 (April) 1935. Broders, A. C. Squamous-Cell Epithelioma of the Skin, *Ann Surg* **73**: 141-160 (Feb) 1921. Nicolini, R. C. Cancer del parpado: consideraciones sobre diagnostico, pronóstico y tratamiento, *Semana méd* **1**: 1630-1632 (June 6) 1935, abstracted, *Am J Cancer* **28**: 634 (Nov) 1936.

must be paid to the situation of the tumor on the eye or the eyelids. Lesions in certain places are attended by greater mortality and cause more blindness than lesions in other places. This is evident from glancing at table 4, regardless of histologic grade of malignancy, more deaths and more blindness occurred from lesions of the upper lid. Lesions situated at the inner canthus were nearly as dangerous as those on the upper lid. More exenterations were done on lesions at the inner canthus than elsewhere, perhaps causing a slightly better gross result, in keeping with the more vigorous treatment.

TABLE 4—*Behavior of Squamous Cell Epitheliomas (Graded) According to the Situation on the Eyelids*

Situation of Growth	Number of Patients		Exenteration		Blind in Affected Eye		Died of Ocular Cancer	
	Total	Traced	No of Patients	Per centage	No of Patients	Per centage	No of Patients	Per centage
Lower lid	17	13	4	30.7	4	30.7	2	15.4
Upper lid	10	10	4	40.0	6	60.0	4	40.0
Inner canthus	8	6	3	50.0	3	50.0	2	33.3
Outer canthus	4	3	0	0	0	0	0	0

TABLE 5—*Behavior of Conjunctival, Corneal and Limbic Squamous Cell Epithelioma on the Basis of Histologic Grading*

Situation of Growth	Number of Patients		Exenteration		Blind in Affected Eye		Died of Ocular Cancer	
	Total	Traced	No of Patients	Per centage	No of Patients	Per centage	No of Patients	Per centage
Conjunctiva	11	11	3	27.3	3	27.3	2	18.1
Limbus	6	5	1	20.0	2	40.0	0	0
Cornea	3	3	0	0	0	0	0	0

Any tumor about the eyelids is likely to prevent perfect closure of the lids and to subject the cornea to exposure and ulceration. A lesion of the upper lip is more likely to cause keratitis from exposure than a lesion of the lower lid. Sometimes this factor has a bearing on the type of treatment chosen, for instance, if plastic repair is contemplated after excision of part of the upper lid, a repair must be done immediately, and this entails the risk of covering some malignant cells, should any remain, with healthy tissue.

Lesions of the outer canthus were rendered exceptionally harmless by excision (table 4). A lesion in this location is more easily completely removed than a lesion at the inner canthus, which, perhaps, tends to invade the orbit more easily.

Lesions situated on the conjunctiva and cornea were not as malignant as were lesions of the upper lid or inner canthus (tables 4 and 5). Corneal lesions, in fact, were as benign as were lesions of the outer canthus.

Primary Corneal Epithelioma—Primary corneal epithelioma is of sufficient rarity to justify digression here a moment for more full discussion of the 3 cases included in table 5. These growths all involved the cornea primarily. Each of them was associated with a pterygium. Furthermore, in all 3 of the cases some sort of trauma had been inflicted in an attempt to remove the pterygium before the lesion occurred. In 2 of the cases the growth originated at the junction of the pterygium and the normal cornea, in the third, it appeared separated from the advancing edge of the pterygium by approximately 1 mm, so that it may have been connected with some small portion of the pterygium.

Malignant lesions of the cornea, when they occur, are usually of the squamous cell variety, because of the single layer of cuboidal epithelial cells that compose the outer epithelium of the cornea. Sarcoma of the cornea has been reported⁹ and probably arises from the corneal stroma, which is mesodermal in origin. Melanosis of the cornea also may occur, secondary to melanotic tumor of the eye, a case has been reported by Schmidt¹⁰ in which the growth occurred eight years after excision of a melanosarcoma of the limbus.

The relation of these 3 cases of corneal squamous cell epithelioma to the trauma inflicted in an attempted removal of a pterygium is in keeping with the generally accepted idea that epitheliomas arise in many cases from chronic irritation.

Corneal squamous cell epitheliomas are generally relatively benign, in spite of the high grade of malignancy seen under the microscope (table 1). In 1 of these cases the growth was graded 2, and in the other 2 the growths were graded 3, yet in none of them was blindness or death the result (table 5).

Limbic Epithelioma—Squamous cell epitheliomas that arise at the corneal limbus appear to be less frequent than those on the bulbar or palpebral conjunctiva (table 1, fig. 1), but they are more frequent than primary corneal epitheliomas. The growths were all graded either 1 or 2, and while none of the 5 patients who were traced died, an eye was lost in 40 per cent (table 5). Typical limbic epitheliomas are shown in figure 4*a* and *b*. The growth in figure 4*a* probably represents a secondary involvement of the limbus, while that in figure 4*b* represents a growth which may have been a primary limbic epithelioma.

9 Gastew, A. A., and Werpoukhowsky, M. M. Sarcome de la cornée, *Ann d'ocul* **172** 587-595 (July) 1935.

10 Schmidt, R. Isolierte Melanosis corneae, 8 Jahre nach Exzision eines oberflächlichen Melanosarkoms am Hornhautlimbus, *Klin Monatsbl f Augenh* **93** 164-170, 1934.

Epitheliomas in this region usually are fed by rather large vessels¹¹ and tend to grow rapidly. Often they are thought to be caused by injury. The discomfort of the sensation of the foreign body makes the patient seek early consultation, and consequently malignant lesions in this region are likely to be seen early.

Conjunctival Epithelioma—The conjunctiva of the globe or lids is frequently involved by squamous cell epithelioma. In this series less blindness was caused by lesions of the conjunctiva than by lesions at the limbus. Conjunctival lesions, however, caused a higher percentage of deaths than did lesions of the lower lid, the outer canthus or the cornea.

In this series none of the conjunctival lesions was of the basal cell type. Mazzi,¹² however, has reported a rare case of basal cell epithelioma on the conjunctiva of each eye. Lopes de Andrade¹³ reported bilateral squamous cell epithelioma of the conjunctiva, but in none of the cases in this series did the growth occur bilaterally.

Conjunctival squamous cell epitheliomas are shown in figure 4 *c* and *d*. Lesions of the conjunctiva may be highly malignant, and it is important to have a histologic estimation of the malignancy before making a final decision as to the method of treatment.

Recurrence and Metastasis—Recurrences after treatment are common with all types of carcinoma of the eye and eyelids. Several factors probably contribute to this fact. Among them is the desire to save the eye and at the same time to cure the lesion. This tends to influence the clinician to choose the minimal treatment rather than a safer method of removal of cancer, which may entail removal of the eye. Another factor influencing the frequency of recurrence is the thought that many of the lesions are essentially benign and consequently do not need histologic examination. Many of the patients in this series came in with recurrences after treatment by unacceptable methods, such as the use of salves, escharotics and out and out quack cancer "cures."

In this group of cases more than 50 per cent of the patients had recurrences after one or another form of treatment. This percentage is based on all patients who received any sort of treatment after the cancer appeared. Many of them were not seen at the clinic until comparatively late. Lesions of grade 3 had the greatest tendency to recur (table 2).

11 Benedict, W. L. Epithelioma of the Limbus, *S. Clin. North America* **9** 813-822 (Aug.) 1929.

12 Mazzi, L. Epitelioma epibulbare bilaterale simmetrico, *Arch. di ottal.* **41** 260-273 (June-Nov.) 1934, abstracted, *Am. J. Cancer* **28** 815 (Dec.) 1936.

13 Lopes de Andrade, A. Epitheliome bilatéral du limbe scléro-corneen, *Ann. d'ocul.* **172** 897-907 (Nov.) 1935.

The greatest danger from lesions about the eyes is that of extension to the orbit. The majority of squamous cell lesions about the eyes drain into the orbit before the lymph nodes of the face or the neck are affected. Lesions of the cornea or conjunctiva almost always extend into the orbit rather than down to the facial nodes.

Metastasis to the lymph nodes of lesions of the lower lids usually extends first to the submaxillary glands, and the metastatic growths

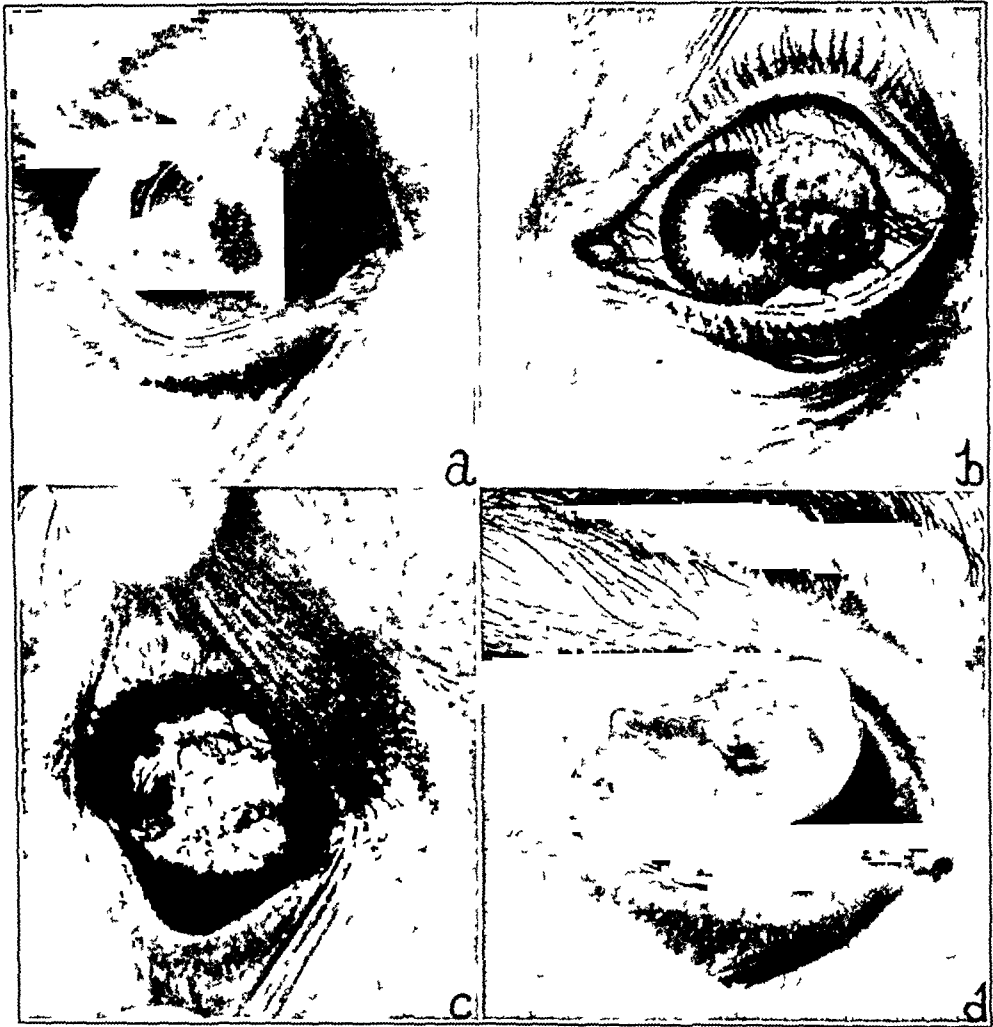


Fig 4—Squamous cell epitheliomas. *a*, *b* and *c*, squamous cell epithelioma of the conjunctiva and the corneal limbus, and *d*, a squamous cell epithelioma of the conjunctival surface of the tarsus and the upper lid.

are found under the angle of the jaw. The cervical nodes will not be affected early. Lesions of the upper lid usually drain into the preauricular nodes or into small nodes in the temporal region. In this series treatment of such metastatic growths consisted of irradiation.

The greater the malignancy, the greater is the chance of involvement of either the lymph nodes or the orbit, regardless of treatment (table 2).

Of lesions of the squamous cell type about the eye, 30 per cent of those of grade 2, 60 per cent of those of grade 3 and 100 per cent of those of grade 4 invaded the orbit

Blindness from squamous cell lesions of the eyelids, conjunctiva and cornea is directly proportional to the grade of malignancy (table 2) There is more blindness from squamous cell lesions as a whole and from individual grades of such lesions, except those of grade 1, than there is from any other type of epithelioma of the eye or eyelids

The chance of saving the eye in the presence of lesions of the eye and adnexa is about 50 per cent if the lesions are of grade 3 and zero if they are of grade 4 With basal cell lesions of this type, not considering the situation of the lesion on the lids, which is of considerable importance, there is generally about a 75 per cent chance of saving the eye

The mortality rate of epidermoid carcinoma of the eyelids and conjunctiva in general is about 12.2 per cent,¹ but this does not hold true of the individual grades of squamous cell epithelioma The correlation between the various grades of malignancy and the span of individual life is shown in figure 2

While squamous cell epithelioma of grade 4 caused the death of 80 per cent of the patients in this small series and that of grade 3 caused the death of 46.7 per cent, there must have been many cases of epithelioma of a low grade of malignancy to give a general mortality of 12.2 per cent in 464 cases In the majority of cases growths of a low grade of malignancy do not prove fatal unless treatment is neglected, but the growths that persistently cause death are those of a high grade of malignancy (fig 3) These are fatal regardless of treatment, and apparently the best way of reducing the mortality from them is by earlier and more extensive treatment

Most carcinomas develop over a long period,¹⁴ during which there is plenty of opportunity for treatment Simmons¹⁵ expressed the belief that the frequent recurrence of cutaneous cancer is due to failure to excise enough tissue around the lesion and failure otherwise to follow recognized principles of surgical technique Belgeri¹⁶ stated that inadequate local treatment causes loss of valuable time and plays havoc with the prognosis Although these statements are true in some instances, there is another more important fact Most of the malignant growths

14 (a) Figs. F A Epithelioma of the Lower Lip Results of Treatment, Surg., Gynec. & Obst. **59** 810-819 (Nov.) 1934 (b) Birge¹ (c) Benedict¹¹

15 Simmons, H T Surgery in the Treatment of Primary Skin Carcinoma, Lancet **2** 938-940 (Oct. 26) 1935

16 Belgeri, F Tumores malignos del aparato visual su diagnostico y su tratamiento, Semana med **1** 1048-1055 (April 11) 1935, abstracted, Am J Cancer **28** 814 (Dec.) 1936

around the eyelids that cause either blindness or death are those of a high grade of malignancy or those of which treatment has been greatly neglected

Carcinoma about the eyelids can be cured in most cases if removal is effected early enough. The eyelids are constantly in view. Yet the average duration of most of the lesions when the patients seek treatment is well over a year, Figi^{14a} reported that the average duration of lesions of the lips before the patient comes for treatment is twenty-eight and three tenths months. MacCarty¹⁷ has shown that the average size of surgically excised malignant growths has not diminished in the past fourteen years. It is entirely possible that removal of all tumors of the eyelids, benign or malignant, when first seen, would reduce the amount of blindness and the mortality from carcinoma of the eyelids.

REPORT OF CASES

Photomicrographs will not be reproduced here because the grading of squamous cell epitheliomas has been amply covered in the literature and the lesions on which this paper is based had the same cellular structure as have other squamous cell epitheliomas.

CASE 1—A man aged 57 had a growth on the right eye near the limbus measuring 6 by 3 by 2 mm, which involved the cornea (fig 4a). It was excised and cauterized. The histologic diagnosis was squamous cell epithelioma of grade 1. Vision in the right eye before excision was 6/6 and in the left eye 6/5. Shortly afterward, reexamination showed the vision to be the same.

CASE 2—A man aged 59 came to the clinic because of a growth on the conjunctiva of the left eye, near the limbus (fig 4b). This had been noted six weeks before admission, and the condition had grown steadily worse. Vision in the right eye was 6/20 and in the left eye 6/12. The mass was excised, and a histologic diagnosis of squamous cell epithelioma of grade 2 was made. Over the course of a year the epithelioma was treated with 3,050 milligram hours of radium, the eye being screened by 2 mm of lead. At the end of this time vision in the right eye was 6/12, and in the left eye 6/10.

CASE 3—A man aged 50 came to the clinic because of a small clear tumor on the inner side of the right cornea, which he had noticed ten years before his admission. Six weeks before admission it had begun to grow (fig 4c). A histologic diagnosis of squamous cell epithelioma of grade 2 was made. The epithelioma was excised from the conjunctiva and cornea, fulguration was done, and the wound was left open. One year later vision in the right eye was 6/10 and in the left eye 6/7.

CASE 4—A mass suddenly developed in the right upper eyelid of a man aged 25 (fig 4d). The mass had appeared one year before he came to the clinic. It had been opened as a cyst three times but had become progressively worse. Radium was applied. At the clinic, vision in the right eye was 6/6 and in the

17 MacCarty, W. C. The Size of Operable Cancers (a Study of 7,179 Specimens), *Am J Cancer* 17 25-33 (Jan.) 1933

left eye 6/7 Histologic examination disclosed inflammatory tissue The mass recurred six months after the radium therapy and was removed, this time the histologic diagnosis was squamous cell epithelioma of grade 2 The growth was treated with 1,400 milligram hours of radium, a 15 mm lead filter being used Two months later a small tumor grew from the temporal side of the right upper eyelid It was excised, and 25 milligram hours of radium was given The histologic diagnosis was inflammatory tissue

Three months after the foregoing excision, the tumor recurred and measured 12 by 18 mm Excision was done, and plasma cell infiltration of inflammatory tissue, with amyloid degeneration, was found Radium was again applied, 1,400 milligram hours, and the eye was screened with a shield composed of 2 mm of lead and 15 mm of monel metal A 25 mg plaque, kept in motion thirty minutes, was applied

Four months later another recurrence was noted, and 1,400 milligram hours of radium was applied, a shield of 2 mm of lead and 15 mm of monel metal being placed 25 cm from the right orbit A plaque of 25 mg was kept in motion for thirty minutes

Six months elapsed, and then another ulcer, or a recurrence, developed, which was cauterized Histologic examination revealed that glands deep in the lid had a tendency to form squamous cell epithelioma

Four months subsequent to this examination, or three years after the growth was first noticed, vision in the right eye was 6/6 and in the left eye 6/5 There was no sign of a new growth, and there was only moderate contraction of the right upper lid, with no adhesions or symblepharon The patient is still being followed

CASE 5—A woman aged 53 came to the clinic because of a growth on the right upper eyelid (fig 5a) She had first noticed the growth five years before, it had varied in size from time to time For five months she had noticed a stinging sensation A pathologic diagnosis of squamous cell epithelioma of grade 2 was made The lesion was excised, and the skin flaps were drawn together

CASE 6—A woman 51 years of age came to the clinic because she had had a tumor on the right upper eyelid for one year (fig 5b) It had broken down occasionally and had never healed There was no pain Vision in the right eye was 6/6 and in the left eye 6/6 The mass measured 1 by 1 by 0.5 cm and was ulcerated on the conjunctival surface It hung down over the pupillary area and obstructed vision It was excised under local anesthesia and was found to be squamous cell epithelioma of grade 3 Treatment consisted of 1,112 milligram hours of radium and the placement of three needles in the skin, equaling 1,040 milligram hours of radium

Six years later the growth recurred and was excised so widely that the lids had to be sutured in an attempt to save the eye This time the growth was a mixed squamous cell epithelioma and an adenocarcinoma of grade 4 Radium, 2,400 milligram hours, was applied, a shield composed of 2 mm of lead and 15 mm of monel metal being placed 25 cm from the right upper lid

CASE 7—A man 44 years of age had had a growth on the left upper lid for three years (fig 5c) It had gradually increased in size He had been given treatment with radium after two years When he was seen at the clinic, vision in his right eye was 6/15 and in his left eye 3/60 There was osseous involvement, and the preauricular and submaxillary glands were enlarged Exenteration was done, and the malignancy of the squamous cell epithelioma was graded 3 Radium

was applied, 2,400 milligram hours, the eye being screened with a shield composed of 2 mm of lead and 15 mm of monel metal

CASE 8—A man aged 56 nine months before seeking treatment noticed a growth on the side of his nose (fig 5 *d*) Pastes had been applied for six months Radium had been used for three months Vision in the right eye was 6/20 and in the left eye 6/30 The submaxillary glands were enlarged The man had diabetes The histologic diagnosis was squamous cell epithelioma of grade 3 Exenteration was done, and the patient lived for five months

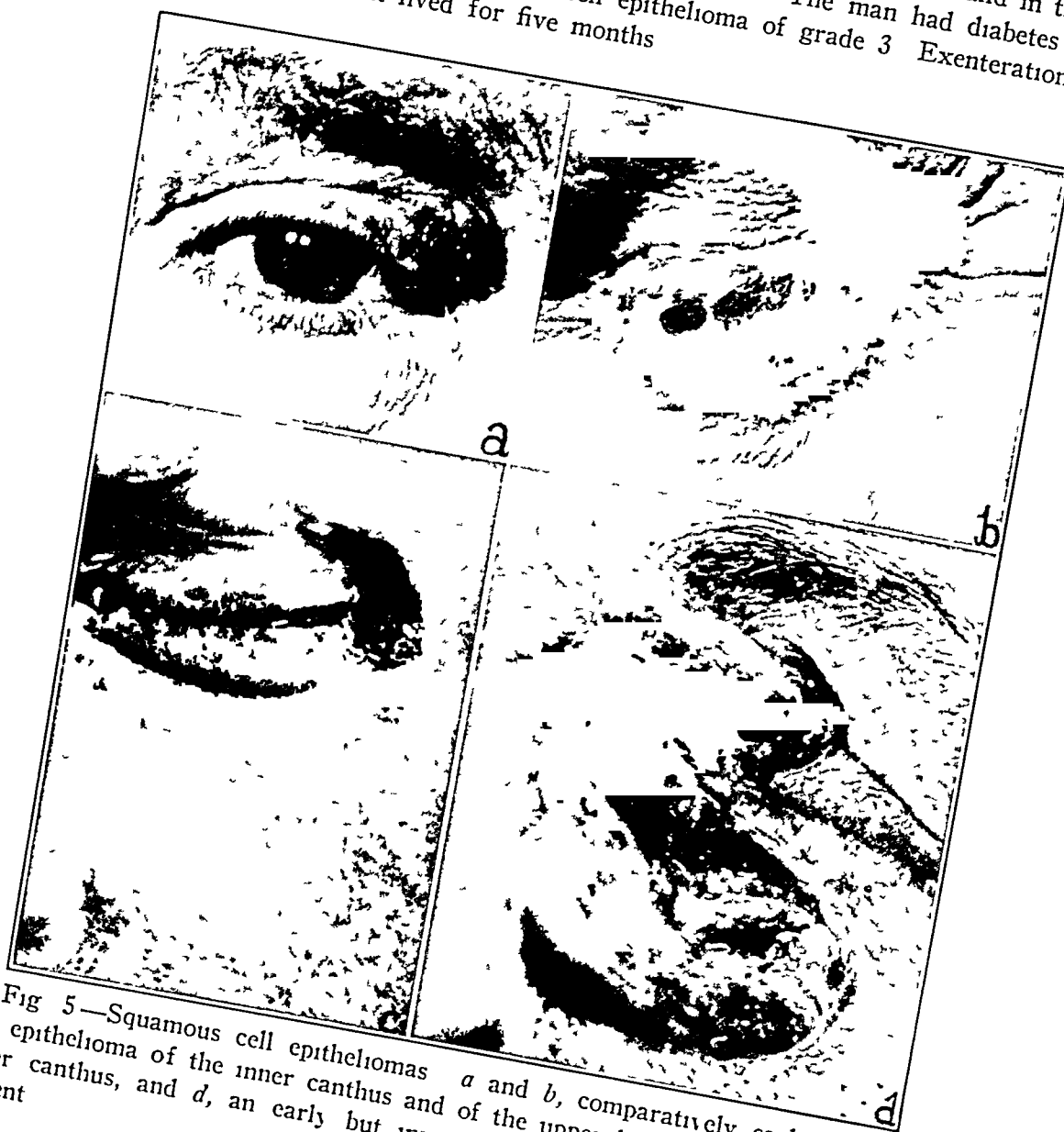


Fig 5—Squamous cell epitheliomas *a* and *b*, comparatively early squamous cell epithelioma of the inner canthus and of the upper lid, *c*, a late lesion of the outer canthus, and *d*, an early but invasive squamous cell lesion of a diabetic patient

SUMMARY AND CONCLUSIONS

Squamous cell epithelioma of the eyelids, conjunctiva and cornea occurred in 25 per cent of the cases of epithelioma of the eye and its adnexa that were studied Thirty-seven per cent of the squamous cell epitheliomas arose from the conjunctival surfaces, including the cornea Primary corneal squamous cell epithelioma occurred in 5 per cent of the

cases in this series. The malignancy of most of the lesions in the entire series was either of grade 2 or grade 3 on the basis of histologic examination. Chronic irritation or trauma was an etiologic factor in 32.2 per cent of the cases.

There was close correlation between the clinical behavior over a period of fifteen years and the grade of malignancy. Lesions of grade 1 did not cause death or loss of an affected eye. Lesions of grade 2 caused loss of the affected eye in 25 per cent of cases and death in 15 per cent. Lesions of grade 3 caused loss of the affected eye in 53 per cent of cases and death in 46 per cent. Lesions of grade 4 caused loss of the affected eye in all cases and death in 80 per cent.

The situation of the lesion about the eye or eyelids is of considerable importance, judged by the related percentage of mortality and blindness. Recurrences were frequent in this series. The average mortality of all types of epithelioma of the eye and eyelids was about 12 per cent. Mortality and blindness were directly proportional to the histologic grade of malignancy. Lesions of grades 3 and 4 were responsible for the largest part of the mortality and blindness.

Given (1) early recognition of the grade of malignancy and (2) treatment proportional with the grade of malignancy, carcinomas about the eye should carry low mortality.

TREATMENT OF STAPHYLOCOCCIC CONJUNCTIVITIS WITH STAPHYLOCOCCUS TOXOID

PRELIMINARY NOTE

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NEW YORK

In a previous communication¹ toxin-producing staphylococci were brought forward as an important cause of chronic catarrhal conjunctivitis. Characteristic of conjunctivitis believed to be staphylococcic is the failure of locally applied antiseptics to heal it or to rid the eye of the offending bacteria. This failure is believed to be related to the predilection of staphylococci for the skin and glands of the margins of the lids, here the bacteria are protected from the action of antiseptics and are able to reinfect the conjunctiva.

In view of the efficacy of immunization with staphylococcus toxin in protecting rabbits against instillations of toxin which would produce severe conjunctivitis in normal rabbits,¹ an attempt has been made to obtain healing in human beings with conjunctivitis by immunization. Although good results with crude toxin have been reported by Burky,² toxoid was employed in the present study because of its greater safety and availability. The Lederle Laboratories made available a supply which, according to the report of Blair and Hallman,³ could be expected to produce a notable rise in the antitoxin titer after a single course.

The present report records the results obtained in the treatment of 57 patients with conjunctivitis harboring staphylococci the toxin-producing ability of which was either tested on the rabbit conjunctiva or presumed from associated presumptive tests. No other possible etiologic factor could be found in 46 of the patients, of the remaining 11, 3 had a conjunctival eosinophilia suggesting an associated allergy, 5 had mild acne rosacea and 3 had cicatricial trachoma of questionable activity. In order to minimize as far as possible the factor of spontaneous healing, only persons were chosen whose conjunctivitis had resisted local

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1 Thygeson, P. Bacterial Factors in Chronic Catarrhal Conjunctivitis. Role of Toxin-Forming Staphylococci, *Arch Ophth* **18** 373 (Sept.) 1937. *Allen, J. Am. J. Ophth.* **20**:1025, 1937.

2 Burky, E. L. *Am. J. Ophth.* **19** 841, 1936.

3 Blair, J. E., and Hallman, F. A. *Proc. Soc. Exper. Biol. & Med.* **34** 637, 1936.

Results of Toxoid Therapy in the Treatment of Staphylococcic Conjunctivitis

No	Patient	Type of Conjunctivitis	Type of Hemolytic Staphylococcus	Results of Toxoid Therapy	
				Clinical	Bacteriologic (Cultures at Last Examination)
37	W U	Chronic conjunctivitis (acne rosacea)	Aureus	Healing	—
33	R H	Chronic keratoconjunctivitis	Aureus	Healing	—
78	J P	Chronic keratoconjunctivitis	Aureus	Healing	—
1a	A C	Chronic blepharconjunctivitis with ulcers	Aureus	Healing	—
2a	A M	Chronic blepharconjunctivitis with ulcers (10 yr)	Aureus	Healing	—
3a	J L	Chronic blepharconjunctivitis	Aureus	Healing	—
1	H E S	Chronic keratoconjunctivitis	Albus	Healing	+
28	G A	Trachoma III IV	Aureus	Healing	+
372	J S	Chronic catarrhal conjunctivitis	Aureus	Healing	+
420	S R	Chronic blepharconjunctivitis	Aureus	Healing	+
506	R K	Chronic blepharconjunctivitis	Aureus	Healing	+
9	E H J	Chronic conjunctivitis with meibomitis	Albus	Healing	No record
43	E G	Chronic conjunctivitis with recurrent styes	Aureus	Healing	No record
25	P T W	Chronic blepharconjunctivitis	Aureus	Healing, recurrence, healing	+
216	M R	Chronic blepharconjunctivitis	Aureus	Healing, recurrence, healing	—
6	B K	Chronic blepharconjunctivitis	Aureus	Healing, recurrence, healing	+
18	J P	Chronic blepharconjunctivitis	Aureus	Healing, recurrence, healing	+
40	F W	Chronic keratoconjunctivitis	Aureus	Healing, recurrence, healing	+
55	H R	Chronic keratoconjunctivitis with styes	Aureus	Healing, recurrence, healing	+
538	H M	Chronic keratoconjunctivitis	Aureus	Healing, recurrence, healing	+
622	R E	Chronic keratoconjunctivitis	Aureus	Healing, recurrence, healing	+
37	E R	Chronic catarrhal conjunctivitis	Aureus	Improvement	—
39	M H	Chronic catarrhal conjunctivitis	Albus	Improvement	—
56	J H	Chronic blepharconjunctivitis	Aureus	Improvement	—
59	G B	Chronic catarrhal conjunctivitis	Albus	Improvement	—
61	R B	Chronic catarrhal conjunctivitis	Albus	Improvement	—
70	J B	Chronic catarrhal conjunctivitis	Aureus	Improvement	—
94	J K	Chronic catarrhal conjunctivitis	Albus	Improvement	—
105	D R	Chronic blepharconjunctivitis	Aureus	Improvement	—
107	L A W	Chronic blepharconjunctivitis	Albus	Improvement	—
244	C D	Chronic blepharconjunctivitis	Aureus	Improvement	—
250	R G	Chronic blepharconjunctivitis	Aureus	Improvement	—
286	J D	Chronic keratoconjunctivitis (acne rosacea and seborrheic dermatitis)	Aureus	Improvement	—
4	S N	Chronic catarrhal conjunctivitis	Aureus	Improvement	+
23	G M A	Chronic catarrhal conjunctivitis (eosinophilia)	Aureus	Improvement	+
54	H K	Chronic catarrhal conjunctivitis	Aureus	Improvement	+
83	W L	Chronic keratoconjunctivitis	Albus	Improvement	+
103	J B	Trachoma III and IV	Aureus	Improvement	+
239	N M	Chronic blepharconjunctivitis (sycoosis vulgaris)	Aureus	Improvement	+
405	E T	Chronic catarrhal conjunctivitis with recurrent styes	Aureus	Improvement	+
48	M J	Chronic catarrhal conjunctivitis (chlamydia)	Albus	No improvement	—
52	H C	Chronic catarrhal conjunctivitis	Albus	No improvement	—
72	I K	Chronic keratoconjunctivitis	Aureus	No improvement	—
87	B S	Chronic catarrhal conjunctivitis	Aureus	No improvement	—
135	F M	Chronic keratoconjunctivitis	Albus	No improvement	—
11	I G	Chronic catarrhal conjunctivitis (acne rosacea)	Albus	No improvement	+
19	M S	Chronic blepharconjunctivitis (sycoosis vulgaris)	Aureus	No improvement	+
21	R S	Chronic conjunctivitis (acne rosacea)	Aureus	No improvement	+
112	H W	Chronic catarrhal conjunctivitis	Aureus	No improvement	+
119	D H	Chronic catarrhal conjunctivitis (sensitivity to drug)	Aureus	No improvement	+
149	E W	Chronic blepharconjunctivitis (eosinophilia)	Aureus	No improvement	+
271	V S	Chronic keratoconjunctivitis	Aureus	No improvement	+
404	M W	Chronic blepharconjunctivitis	Albus	No improvement	+
486	T P	Trachoma III and IV	Aureus	No improvement	+
494	A C H	Chronic blepharconjunctivitis	Aureus	No improvement	+
557	M G	Chronic blepharconjunctivitis	Albus	No improvement	+
152	D G	Chronic catarrhal conjunctivitis	Albus	No improvement	No record

treatment not less than two months. The majority of the patients had had conjunctivitis for over a year.

The toxoid was given biweekly, starting with an initial injection of 0.01 cc of dilution no. 2. The dose was increased to a maximum of 1 cc in steps of 0.02, 0.04, 0.06, 0.08, 0.1, 0.2, 0.4, 0.6 and 0.8 cc. In the few cases in which general reactions occurred, this schedule was modified. Injections of less than 0.5 cc were given intradermally, and the remainder were given subcutaneously. In those cases in which noticeable improvement did not occur on completion of the series, injections of 0.5 cc were continued at weekly intervals for at least six weeks. In all cases the same local treatment which had previously proved ineffective was continued.

The results of treatment are recorded in the table. Healing occurred in 21 of the 57 patients, 19 showed clinical improvement, and the condition of 17 remained unchanged. The cultures of 24 of the 57 patients became negative. There was a recurrence of symptoms after periods varying from four to six months in 8 of the 21 persons in whom clinical healing occurred. In all of these the symptoms disappeared after a second course of toxoid therapy. This recurrence was probably related to the fact that in 7 of the 8 patients some toxigenic staphylococci were still present after healing. The cultures of the 13 patients in whom healing occurred, and who have as yet had no recurrence, were negative after healing.

Before the favorable results are credited to the production of a specific resistance to staphylococcus exotoxin through the formation of antitoxin, the following factors must be considered: (1) nonspecific protein effect, (2) spontaneous healing and (3) effect of local treatment. There seems to be no way of evaluating the first factor, but the second and third would appear to be of minor importance, since the series included only persons with conjunctivitis of long standing which had long resisted local treatment.

SUMMARY AND CONCLUSIONS

A series of 57 patients with conjunctivitis presumably due to toxigenic staphylococci were treated by immunization with staphylococcus toxoid. The conjunctivitis was of long standing and had resisted local treatment for periods of not less than two months. Twenty-one of the 57 patients showed healing, 19 showed clinical improvement and 17 showed no improvement. The cultures of only 24 of the 57 patients became negative after treatment. Eight of the 21 patients in whom healing occurred had a recurrence of symptoms, these disappeared after a second course of toxoid therapy. The recurrences appeared to be related to the failure of treatment to eliminate the offending bacteria. It is believed that the results obtained warrant further clinical trial of staphylococcus toxoid.

MANNITOL FERMENTATION AS AN INDICATOR OF CONJUNCTIVAL PATHOGENICITY OF STAPHYLOCOCCI

PHILLIPS THYGESON, M D

NEW YORK

The recent reports of Burky,¹ Thygeson,² Allen³ and O'Brien and Allen⁴ indicate an increasing recognition of the important role of toxin-producing staphylococci in conjunctival and corneal inflammations. The common occurrence of saprophytic varieties of staphylococci on the margins of the lids and on the conjunctiva necessitates some test by which toxigenic strains may be recognized readily. Any strain can be tested for its ability to elaborate a conjunctivitis-producing toxin by a method which has been previously described,² but this is a time-consuming and expensive procedure and not adapted to routine use.

Of the simple tests which have been shown to have some correlation with the production of toxin, the mannitol fermentation test appears to permit of widest application. Overnight readings can be obtained on phenol red-mannitol agar plates inoculated directly from the conjunctiva, a distinct advantage over the coagulase and crystal violet agar tests which require secondary inoculations. In a former study² a correlation between the ability of staphylococci to ferment mannitol and their ability to produce a conjunctival toxin was found in only 52 of 57 strains, or 91.2 per cent, and of 57 strains producing no conjunctival toxin, 9, or 15.7 per cent, fermented mannitol. These tests, however, were made by the tube method, which subsequent work has shown to be less reliable than the plate method.

In the present study 196 strains of staphylococci from eyes with conjunctivitis were tested for ability to ferment mannitol and for ability to form a conjunctivitis-producing toxin. The toxins were prepared according to the method of Leonard and Holm⁵ and tested on the

From the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, and the Institute of Ophthalmology, Presbyterian Hospital

1 Burky, E. L. *Am J Ophth* **19** 841, 1936

2 Thygeson, P. *Bacterial Factors in Chronic Catarrhal Conjunctivitis*
Role of the Toxin-Forming Staphylococci, *Arch Ophth* **18** 373 (Sept) 1937

3 Allen, J. H. *Am J Ophth* **20** 1025, 1937

4 O'Brien, C. S., and Allen, J. H. *Am J Ophth* **21** 641, 1938

5 Leonard, G. F., and Holm, A. *J Immunol* **29** 209, 1935

conjunctiva of rabbits, as described in a previous report² The mannitol fermentation test was carried out by streaking the culture on phenol red-mannitol agar (Difco) plates,⁶ and the reaction was considered positive if after incubation overnight the medium around the colonies had changed from pink to yellow Of the 92 strains which showed definite production of toxin, all but 1 fermented mannitol, and of the 104 nontoxicogenic strains, only 4 showed fermentation These results (a correlation of 97.4 per cent) compared favorably with those obtained with the coagulase test (considered by most workers to be the most reliable indicator of pathogenicity) for this series, all but 1 of 86 strains tested giving positive reactions and all but 2 of 82 nontoxicogenic strains giving negative reactions (a correlation of 98.2 per cent)

The mannitol fermentation test, as performed on phenol red-mannitol agar plates, would seem therefore to be a reliable indicator of probable conjunctival pathogenicity of staphylococci It is employed routinely in the studies on conjunctivitis now being conducted at the Vanderbilt Clinic

⁶ This medium was called to my attention by Mr George H Chapman, New York

RECURRENT RETINAL AND VITREOUS HEMORRHAGES IN THE YOUNG — EALES' DISEASE

REPORT OF TWO CASES

R TOWNLEY PATON, MD
NEW YORK

The first adequate description of recurring retinal hemorrhages in young adults was given by Henry Eales in 1880¹ In 1882² he further summarized the characteristics of his earlier reported cases in a paper entitled "Primary Retinal Haemorrhage in Young Men" His first impression was that all patients with this condition suffer from severe constipation and epistaxis He later elaborated on this theory in the following manner

From the absence of any evidence of any of the various blood conditions known to cause haemorrhage, from the absence of albuminuria, diabetes, gout, syphilis, and of any high arterial tension, from the character of the haemorrhage, and from the evidence of local variations of circulation, and from the slow pulse, constipation, flushing of the face, headache, and puffiness and discolouration of the eyes, I am inclined to attribute this combination of conditions to a neurosis affecting both the circulatory organs and the digestive system, leading on the one hand to partial inhibition of the muscular movements of the bowels, and to a vaso-motor contraction of the vessels of the alimentary canal, with inhibition of its secretory functions, thereby causing dyspepsia, constipation, malnutrition, and on the other hand to a compensatory dilatation of the systemic capillaries, especially those of the head, and, in these cases, of the retina, causing over-distention of the venous system and systemic capillaries, with liability to rupture on the occurrence of any intensifying cause Hence the headache, the epistaxis, the retinal haemorrhages, the tortuosity and fullness of the retinal vessels, and temporal artery

It is interesting to note that in Eales' series of cases the left eye was most frequently affected He attributed this to the anatomic condition of the vessels, that is, the vessels going to the right side take a more circuitous course and do not have a direct extension from the aortic arch Both factors favor a higher vascular tension on that side

From the ophthalmologic service of the Manhattan Eye, Ear and Throat Hospital

Read at the Seventh Congress of the Pan American Medical Association, Jan 20, 1938

1 Eales, H Cases of Retinal Haemorrhage with Epistaxis and Constipation, Birmingham M Rev 60 262-273, 1880

2 Eales, H Primary Retinal Haemorrhage in Young Men, Ophth Rev 1 41, 1882

INCIDENCE

This interesting observation concerning the incidence of the condition in the left eye is borne out by the numerous case reports appearing in the literature³ and by 5 cases which I observed personally, in which the hemorrhages usually began in the left eye. The right eye when involved does not as a rule show the same severity of the disease. That this condition is rare may be deduced from a study of the literature and from my personal observation in the clinics of the Wilmer Ophthalmological Institute and at the Manhattan Eye, Ear and Throat Hospital. At the Manhattan Eye, Ear and Throat Hospital, where about 29,000 persons with ophthalmic conditions are treated a year, only 6 cases of typical Eales' disease have been brought to my attention. In all I have observed 12 cases of this rare condition. Eales stated that he saw only 1 patient with the condition among 12,000 patients treated for ocular diseases.

It is generally agreed that the condition has a predilection for the male sex,⁴ although many typical cases occurring in young females have been reported.

ONSET

The hemorrhages occur spontaneously and usually have an acute onset, not associated with exertion. Some patients complain of a slight blurring of their vision for a few days before the attack, which they usually attribute to autointoxication or eyestrain. The hemorrhages are not related to hemorrhages elsewhere in the body, though epistaxis has been reported. All the patients are in apparent good health, though some complain of a slightly depressed feeling, with sluggishness of the bowels or severe constipation. About the only ophthalmoscopic sign concerning which there seems to be a general agreement is that there is a noticeable distention of the veins at the time the hemorrhages occur.⁵

3 Davis, A. E. Recurrent Retinal Hemorrhage of Adolescence, *Am J Ophth* **3** 657, 1920. Kennedy, W. A., and Wagener, H. P. Recurrent Idiopathic Retinal Hemorrhages, *Am J Ophth* **17** 532, 1934. Kokott, W. Klinische Untersuchungen über die Ätiologie der juvenilen rezidivierenden Glaskörperhamorrhagien, *Klin Monatsbl f Augenh* **94** 327, 1935. Lowenstein, A. Zur Klinik der Augentuberkulose. Zur Frage des Ursprungs der rezidivierenden Glaskörper- und Netzhautblutungen jugender Individuen, *ibid* **95** 458, 1935.

4 Moore, R. F. *Medical Ophthalmology*, London, J. & A. Churchill, 1925, p. 243. von Hippel, E. Zur Frage der Perivasculitis retinae (rezidivierende Glaskörperblutungen bei Jugendlichen), *Arch f Ophth* **134** 121, 1935. Adams, P. H. Discussion on the Significance of Retinal Haemorrhages, *Tr Ophth Soc U Kingdom* **42** 216, 1922.

5 Lyle, T. R. Vascular Diseases of the Retina, *Proc Roy Soc Med* **29** 391, 1936. Pavia, I. L., and Dusseldorp, M. Flebitis y hemorragias de retina en tuberculosos, *Rev oto-neuro-oftal* **8** 7, 1933.

In one of my own cases this distention was noticeable for two years, and only after the last attack subsided did the veins begin to have a normal appearance. During this two year interval there had occurred at least eight attacks of hemorrhage in the vitreous, the vision being reduced to perception of hand movements at times. The distention of the veins always appeared greatest just previous to an attack, though of course the fundus could not be studied at all times, owing to the severity of the hemorrhages.

TYPES OF HEMORRHAGES

It may be roughly stated that hemorrhages in the retina and vitreous are the result of two chief causes: an altered condition of the blood itself or a diseased condition of the walls of the blood vessels. The hemorrhages seen in Eales' disease are usually confined to the periphery of the retina, being found only occasionally near the disk, and then only around the larger venous trunks. In some cases retinal exudates have been present in addition. Flame-shaped hemorrhages are rarely seen although they may be present. The fovea and macula are nearly always spared. The hemorrhages usually extend anteriorly into the vitreous and in 1 case that I am reporting they were entirely confined to the vitreous. Though hemorrhages in the vitreous are usually profuse, often blotting out the entire vision, it is surprising how completely they will absorb, with a return of normal vision.

CAUSES OF HEMORRHAGES

Hemorrhages in the retina or vitreous are always of traumatic, toxic or infective origin. Probably the most difficult factor to be ruled out is a focal infection, such as an infection of the teeth, tonsils or sinuses. Syphilis, diabetes and cardiovascular renal disease must also be completely ruled out.⁶ Hypothyroidism and hyperthyroidism, often associated with a calcium deficiency brought about by a diminished parathyroid secretion, have been fully discussed by Zentmayer,⁷ Young⁸ and Jeandelize.⁹ That this condition often occurs at the time of puberty

6 Ellett, E. C. Sequels of Vitreous Hemorrhages, *Am J Ophth* **6** 496, 1923. Redding, L. C. Recurrent Hemorrhages into the Retina and Vitreous in Young Persons, *Atlantic M J* **27** 640, 1924. Radcliffe, McC., and Young, C. A. Recurrent Hemorrhages in the Vitreous, *Am J Ophth* **6** 496, 1923.

7 Zentmayer, W. Recurrent Retinal Hemorrhages, *Am J Ophth* **3** 652, 1920.

8 Young, C. A. Recurrent Hemorrhages into the Retina and Vitreous Calcium Deficiency as Possible Cause, *Tr Am Acad Ophth* **34** 191, 1929.

9 Jeandelize, P., Bretagne, P., and Richard, G. Hemorragies recidivantes du vitre et hyperthyroïdie, *Ann d'ocul* **159** 655, 1922.

certainly suggests some endocrine imbalance. No one, however, has offered an explanation of why recurrent hemorrhages of this type occur most frequently in male patients.

Careful analysis of the blood invariably rules out the various blood dyscrasias, such as polycythemia, pernicious anemia, leukemias and primary anemias. The lack of evidence of circulatory or trophic disturbances elsewhere in the body can usually rule out Raynaud's and Buerger's disease¹⁰ in the absence of ophthalmoscopic changes such as have been described by Ballantyne and Michaelson¹¹ and Cunningham¹². It must be remembered that, as von Hippel pointed out, "cold feelings in the extremities, paresthesia, etc., are quite commonly found in healthy people and that the pulse of the dorsal artery of the foot is very often not easily palpable. The most definite sign of thrombo-angitis is gangrene."

Local phlebitis of the walls of the vessels, as was made clear by Coats many years ago and more recently by Finnoff and Reynolds,¹³ Friedenwald¹⁴ and Verhoeff,¹⁵ might cause hemorrhages. The tubercle bacillus has been found occasionally in the peripheral blood vessels of the eye by Otori,¹⁶ Gilbert,¹⁷ Stock¹⁸ and others, so that there can be no doubt as to what causes this phlebitis. In many of the observed cases the hemorrhages were brought about by a diapedesis and were not the result of gross rupture of the walls of the vessels, for when the hemorrhages have absorbed no lesions can be seen. Pathologic examination of such eyes has not been reported to my knowledge.

10 Marchesani, O. Eine neue Auffassung des Krankheitsbildes der sogenannten juvenilen rezidivierenden Glaskörperblutungen, *Klin Wchnschr* **13**:993, 1934.

11 Ballantyne, A. J., and Michaelson, I. C. A Case of Perivasculitis Retinae Associated with Symptoms of Cerebral Disease, *Brit J Ophth* **21**:22, 1937.

12 Cunningham, J. P. Retinal Hemorrhages and Exudations in a Young Subject, *Tr Ophth Soc U Kingdom* **32**:177, 1912.

13 Finnoff, W. C., and Reynolds, E. M. Clinical and Experimental Studies of the Spread of Tuberculosis in the Eye, *Tr Am Acad Ophth* **38**:152, 1933.

14 Friedenwald, J. S. Pathology of the Eye, New York, The Macmillan Company, 1929, p. 70.

15 Verhoeff, F. H. Histologic Observations in a Case of Localized Tuberculosis Chorioretinitis, *Arch Ophth* **1**:63 (Jan.) 1929.

16 Otori, K. Pathologisch-anatomische mit experimentelle Untersuchungen zur Kenntnis der primären Netzhauttuberkulose, *Arch f Augenh* **79**:44, 1915.

17 Gilbert, W. Ueber intra-okulare Tuberkulose, *Munchen med Wchnschr* **6**:306, 1914.

18 Stock, W. Weitere Untersuchungen über hämatogene Tuberkulose der Augen des Kaninchens, *Ber u d Versamml d ophth Gesellsch* **32**:297, 1905, Tuberkulose als Aetiologie der chronischen Entzündungen des Auges, *Arch f Ophth* **66**:1, 1907.

COMPLICATIONS

The commonest dangers associated with this condition are acute rises in tension, with ultimate loss of the eye, permanent opacities in the vitreous, partially destroying the sight, cataract, detachment of the retina, thrombosis, newly formed blood vessels,¹⁹ vascular veils, and various degrees of proliferating retinitis. Perhaps one of the best descriptions of vascular growths in the vitreous accompanied by hemorrhages is given by Marple.²⁰ A case similar to one that I am about to present was first described, according to this article, by Coccus in 1859. The patient was a woman aged 36. Marple also cited a similar case in a man aged 27, which was reported by Mauthner and Jaeger (1867). The most interesting thing to me, and something that I have noticed in my last case, is that there is a tendency for these vessels to disappear once the hemorrhages cease, no matter how far forward the vessels may project into the vitreous.

PROGNOSIS

Where vascularization of the vitreous occurs, the prognosis is not necessarily poor. Often the vessels will recede and even disappear if there are no hemorrhages over a period of years. Vascularization of the vitreous has been reported even in older persons, with retention of normal acuity of vision (Flint and Harrington^{19a} and Giri^{19b}). If the hemorrhages occur solely in the vitreous and there is no accompanying retinitis, the prognosis, though guarded, seems to be fairly good with proper treatment. Whenever the hemorrhages are limited to the retina and there has been an accompanying thrombosis, the prognosis is bad.

TREATMENT

While no one can definitely say that recurrent hemorrhages of non-specific origin may not occur in the vitreous of young adults, it is the consensus that the hemorrhages are a direct result of a tuberculous process in the blood vessels. Treatment should be more or less empiric and guided by common sense. The importance of fresh air, rest, freedom from overstrain and a diet rich in vitamins has long been recognized as helpful. That tuberculosis of the eye in one form or another may exist without any systemic infection is now generally admitted, however, this conclusion must be derived only after most careful and painstaking clinical and roentgenographic examinations of the patient have been

19 (a) Flint, G, and Harrington, D. New Vessel Formation in the Vitreous, *Brit J Ophth* **18** 27, 1934. (b) Giri, D. V. Vascularisation of the Whole Vitreous in a Case of Hemorrhagic Retinitis with Retention of Normal Acuity of Vision, *ibid* **18** 24, 1934.

20 Marple, W. B. Contribution to the Pathology of Vascular Growth into the Vitreous, *Tr Am Ophth Soc* **9** 254, 1901.

made, for the treatment will depend somewhat on the results of these examinations. For example, it is not necessary to send a patient with this or any other form of ocular tuberculosis to a sanatorium. The patient should be hospitalized when the hemorrhages are most severe, for it is only in the hospital that adequate local treatment can be given. Local treatment consists of injections of saline solution, atropinization, the use of ethylmorphine hydrochloride and hot applications after clotting of the blood has taken place. Special procedures, such as roentgen irradiation, treatment with ultraviolet rays and autohemotherapy, can also be carried out best while the patient is under careful supervision.

There are many forms of treatment which have been suggested and tried with various measures of success. Perhaps it would be well to mention a few of these—the administration of calcium lactate or calcium gluconate, the intravenous injection of vitamin C (cevitamic acid), the injection of snake venom, the use of the galvanic electric current, the administration of ergot, glyceryl trinitrate, or gold sodium thio-sulfate and the injection of foreign proteins and horse serum. One by one these measures may be eliminated for lack of clinical evidence. That tuberculin is helpful in treating other forms of ocular tuberculosis, there can be little doubt. Woods and Randolph,²¹ Wilmer,²² Friedenwald,²³ Gay²⁴ and Eggston²⁵ have agreed to this.

REPORT OF CASES

CASE 1²⁶—W. R., a white man aged 28, had his first severe hemorrhage on June 24, 1931. The vision of the right eye with correction was 20/40 and of the left eye 3/200. A thorough survey was made, including a dental examination, roentgenographic examinations of the chest and sinuses, a basal metabolic test, a study of the blood chemistry, a determination of the blood pressure, a complete examination of the blood and urine, a Wassermann test and a general physical examination. The results of all tests and examinations were negative, except that the patient gave a positive reaction to 0.1 cc. of a 1:100,000 dilution of tuberculin and had a slight chronic infection of the right ethmoid sinus and antrum. He was immediately given tuberculin therapy, the injections being con-

21 Woods, A. C., and Randolph, E. Treatment of Ocular Tuberculosis, *Arch Ophth* **18** 510 (Oct.) 1937. Woods, A. C. Tuberculosis of the Eye, *Internat Clin* **1** 96, 1933.

22 Wilmer, W. H. Clinical Aspects of Ocular Tuberculosis, *Arch Ophth* **57** 1, 1928.

23 Friedenwald, J. Allergy and Immunity in Ocular Tuberculosis, *Arch Ophth* **9** 165 (Feb.) 1933.

24 Gay, L. N. Treatment of Ocular Tuberculosis with Tuberculin, *Arch Ophth* **3** 259 (March) 1930.

25 Eggston, A. A. The Use of Tuberculin in Diagnosis and Treatment in Ophthalmology, *Arch Ophth* **8** 671 (Nov.) 1932.

26 This case was reported at the New York Academy of Medicine, Section of Ophthalmology, in April 1935 and a report was published in the *ARCHIVES OF OPHTHALMOLOGY* (**14** 860 [Nov.] 1935).

tinued biweekly for two years. In the meantime the patient had seven severe attacks of hemorrhage in the right eye and four in the left eye. During the height of one attack the vision in the right eye was reduced to perception of hand movements at 1 foot (30 cm) and in the left eye to 2/200. Between attacks, when the fundus could be seen, the veins appeared markedly engorged, and only once was a small retinal hemorrhage seen. The diagnosis of tuberculous perivasculitis was made by numerous observers, though there appeared to be an absence of the usual perivascular inflammatory changes. The patient has had no attacks since July 1932. The vision with correction is 20/15—in each eye. He is still receiving weekly injections of tuberculin for the purpose of maintaining his acquired immunity, although he has had no hemorrhages for at least five years. No lesion was ever noted in the fundi.

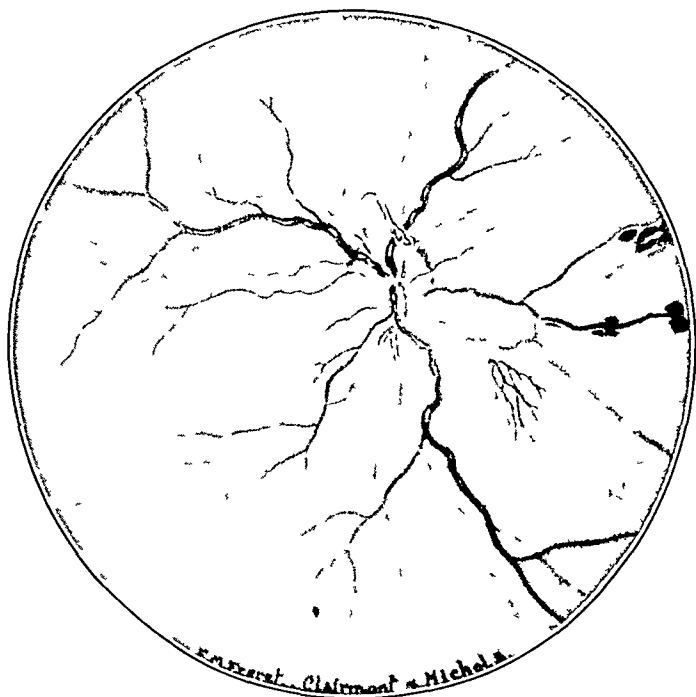


Fig 1 (case 2)—Right eye, showing newly formed vessels sprouting from the optic disk and retina. The drawing was made three years after the first attack of hemorrhage. The clumps of pigment seen in the nasal quadrant were thought to be of congenital rather than of inflammatory origin. One year after the drawing was made the vessels had completely atrophied and disappeared. The veins are still somewhat dilated and tortuous.

CASE 2—M. S., a white man aged 21, had his first hemorrhage in the left eye in April 1933. The vision has steadily remained blurred in the left eye, whereas the right eye has retained vision of 20/15, in spite of repeated retinal hemorrhages and vascularization of the vitreous. A most exhaustive laboratory study of the patient's blood was made, including determinations of the platelet count, the differential count, the bleeding and clotting times and the calcium, the phosphorus and the cholesterol content and the rest of the usual chemical analyses. All the usual known tests to eliminate tuberculosis and Raynaud's or Buerger's disease were made. Roentgenograms of the sinuses and chest were normal. The



Fig 2 (case 2) —Left eye, showing preretinal fibrinous tissue and new vessel formation. There is a small streaklike retinal hemorrhage on the nasal side of the disk.



Fig 3 (case 2) —Drawing of the same eye as shown in figure 2, pictured more anteriorly. The branching of the vessels appears dichotomous, and there are few free terminals. There have been a marked regression of the vessels and a thinning of the fibrinous tissue during the past year.

patient was found to give a slightly positive reaction to 0.01 mg of tuberculin and treatments were begun several months before he came under my observation. The injections of tuberculin were continued throughout the course of the disease, and the patient is still receiving weekly injections. Because of a low basal metabolic rate, -20 , the patient has been taking small doses of thyroid. When the basal metabolic test was repeated, the rate was found to be -5 , and the administration of thyroid was discontinued. At one time it was thought the patient had a low platelet count. On repeated checks, it was found to be normal. In order to build up the patient's resistance, numerous therapeutic measures were used. He was given a large number of intravenous injections of cevitamic acid and five injections of gold sodium thiosulfate. He was told to lead an outdoor life, with a moderate amount of exercise, and to stay in bed after any severe hemorrhage, until the vision showed a definite return to normal. The most interesting feature of the case appears to be the atrophy or recession of the blood vessels, especially noted in the right eye. The small vessels projecting from the disk and the whorl arrangement of blood vessels shown in figure 1 have entirely disappeared. The only sign that there has been any active hemorrhage is some scar tissue with slight clumping of the retinal pigment. There seems also to be atrophy of the fibrinous tissue of the left eye, with great improvement in the visual field, which at one time was completely blotted out and could be taken only with the ophthalmoscope light. The patient's vision on his last visit, March 1938, was 20/15 in the right eye with a -0.75 D sph and 20/50 in the left eye with a -1.50 D sph.

COMMENT AND SUMMARY

The striking difference in the picture of the fundi in these two cases of recurrent hemorrhages in the vitreous deserves some comment. Both patients, young men in apparent good health, were observed for several years, during which time there was marked disturbance of vision and at times blindness was threatened because of the severity of the hemorrhages. That both patients should recover, one with normal vision in each eye and the other (case 2) with restoration of two thirds of his sight in spite of a partially vascularized vitreous, should be of more than passing interest. A diagnosis of tuberculous perivasculitis could not be made with certainty in case 1, as at no time were there visible changes in the vessels. Most observers agreed that the veins appeared markedly engorged, especially at the time or just previous to an attack of hemorrhage. At the end of six years, long after the hemorrhages had ceased, both fundi appeared normal. The veins still looked somewhat dilated, and there were no scars or other signs of an old inflammatory process in the retina. The vitreous, except for a few filamentous opacities, had also cleared, and the patient experienced no ocular discomfort. The faint opacities in the posterior cortex of the lens have not increased in density during the past five years and have caused no measurable loss of vision.

The hemorrhages in the vitreous in case 2 illustrate another type and undoubtedly are not the same as those first described by Eales, though the etiologic basis may be the same. The condition in this case resembles in many ways that in the 3 cases recently reported by Knapp²⁷ under the heading "Formation of Preretinal Connective Tissue in the Vitreous in Acute Choroiditis."

²⁷ Knapp, A. Formation of Preretinal Connective Tissue in the Vitreous in Acute Choroiditis, *Arch Ophth* **18** 558 (Oct) 1937

AN OPHTHALMIC CARRIAGE

JOHN N EVANS, M D

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Though ophthalmology as a medical science has ever marched in the forefront, there are certain portions of clinical work wherein it seems to lag behind. A review of these deficiencies and of possible methods for their correction demonstrated an urgent need for improvement in bedside facilities for the study of patients with ophthalmic conditions.

A portable instrument carriage seemed to me essential to facilitate study in cases in which ophthalmic consultation is required and for the transportation of equipment during rounds in the hospital.

If one reviews the bedside notes made during such rounds, it becomes evident that in many instances there has been an omission of study procedures owing to a lack of satisfactory facilities. The omission of a postoperative test of the visual acuity of the sound eye as well as of the affected eye is not uncommon, and when the visual acuity is studied, the conditions under which the test is made are ordinarily far inferior to those under which it was made on the same patient previous to admission to the hospital. This is but one example of the need for improved bedside facilities for ophthalmologic study, and it was determined to endeavor to provide the means to supply this need.

A description of the portable carriage devised follows.

An oak filing cabinet of standard type was altered so that its final exterior dimensions were height, 26 inches (66 cm), depth, $27\frac{3}{4}$ inches (70 cm), and width, $16\frac{1}{2}$ inches (42 cm). This alteration provided for two large drawers, which open from the front end of the carriage. They were shortened to provide a space within the cabinet for the storage of eight dry cells (ignition type, used ordinarily for the operation of doorbells and buzzers), for the accommodation of a rheostat (for the reduction of house current for the hammer lamp) and an automatic reel for the lamp cord and for the housing of charts for testing visual acuity. Further space was allowed for a small rheostat for the reduction of the current of the aforementioned batteries and an incorporated jack to receive a plug (so that electric ophthalmoscopes, retinoscopes, an O'Brien slit lamp, a transilluminator and other devices can be operated from the battery supply).

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On the top of this cabinet was mounted a brass tray having an upturned flange of $1\frac{1}{2}$ inches (37 cm) . At one end of this tray a slot with a surrounding flange was provided, so proportioned that two standard charts for testing visual acuity can be raised and lowered with their appropriate frames and locking mechanism . At the opposite ends of the cabinet, in each corner, were provided two tubular sockets proportioned for the reception of a rod and adapted with lock nuts, so that the rod can be elevated or depressed and fixed in position as required . On the upper end of this rod a hammer lamp was mounted in a lockable universal joint . (This permits the hammer lamp to be

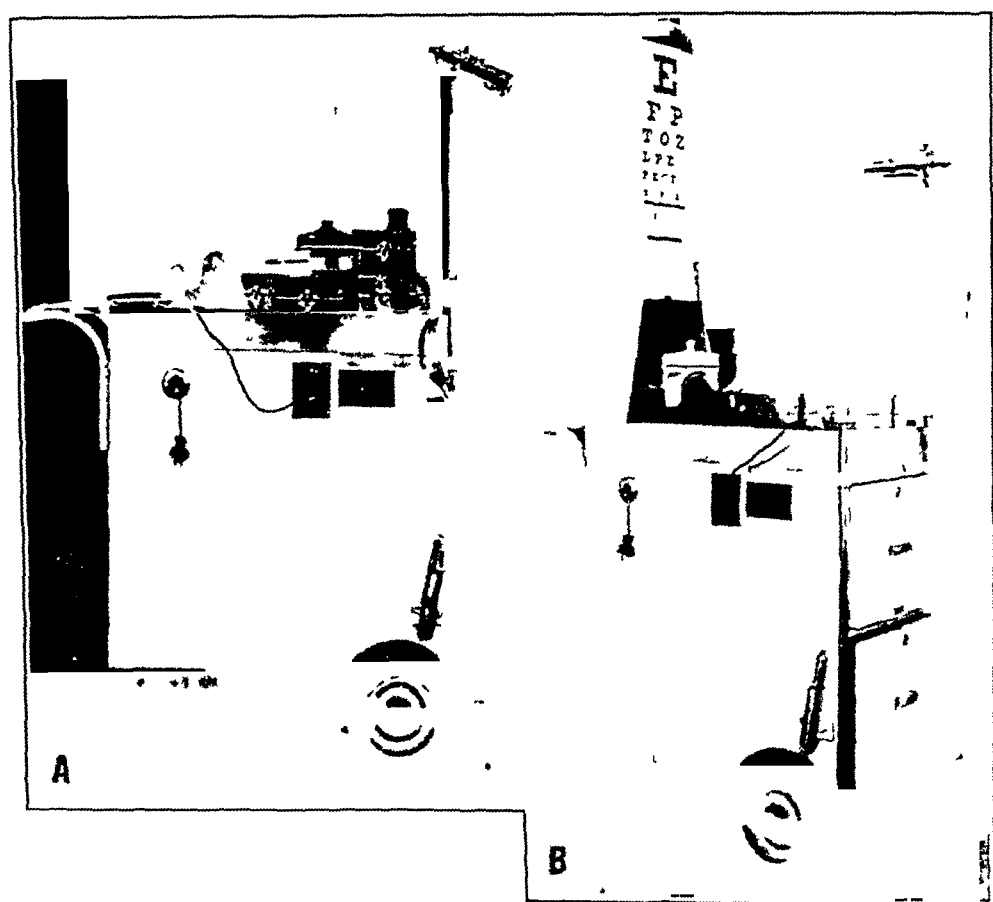


Fig 1—*A*, ophthalmic carriage arranged for the physician when doing bedside dressings . The solution bottle, dressing jar, treatment boxes and adhesive tape carrier are in evidence . For purposes of illustration, the O'Brien hand slit lamp and binocular magnifier are in view . *B*, ophthalmic carriage showing the arrangement of the light and the visual acuity test chart . It will be noted that the chart is so elevated that it can be seen by the patient without material elevation of the head of the bed .

directed for the illumination of a visual acuity test chart, the dressing tray or the examination field of a patient . It also permits its use for indirect ophthalmoscopic and retinoscopic examinations and for oblique illumination . A ground glass disk was devised for the hammer lamp, to soften and diffuse the light and an adjustable diaphragm was also

provided, so that the hammer lamp can be used as a muscle light or for other purposes) The hammer lamp is thus readily demountable, so that it can be used as a hand instrument and can be stored with its accessories in a rack provided for that purpose in the upper drawer of the cabinet The remaining space in the upper and lower drawers of the cabinet was equipped with the aforementioned instruments and accessories as well as with a hand perimeter, a folding campimeter, a small trial case with frames and accessories, a visual acuity test chart for testing near vision, a Placido disk, an ophthalmoscope handle with adaptors to receive various makes of heads, Stilling's pseudoisochromatic

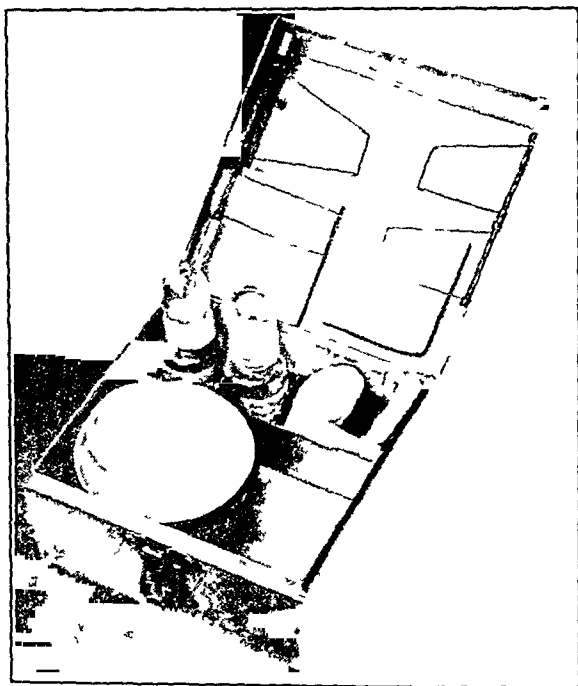


Fig 2—Individual dressing box This box is equipped and assigned to each patient as he enters the hospital The body of the box is so divided that there is room for four medication bottles These bottles have glass stoppers which are ground to the outside of the neck of the bottle The graniteware cup is merely used as a container for the sterile paper cup, the latter being put into the box just before the dressing is commenced The clips in the lid of the box retain dressing pads, hard sponges, soft sponges, loosely and tightly wound cotton applicators, all sterile-wrapped The loose-wound applicators are used for instilling the medication

plates for testing color sense, a combination millimeter-inch measuring tape, Berens' combination accommodation rule and one set of muscle prisms

The upper surface of the cabinet was covered with a heavy brown linoleum, which was so sealed in place as to be effective in waterproofing the top of the cabinet The top, with its brass rim, thus provides sufficient space for six individual dressing boxes (Each patient

is allotted a special cadmium-plated dressing box when he is admitted to the hospital. It contains special bottles of such freshly prepared medication as is ordered for him, together with applicators, minor dressings and a cup for saline solution, an adhesive tape dispenser [the new transparent tissue is being used] and flasks of sterile saline solution, sponges and dressings.)

The carriage was mounted on pneumatic tires and a swivel wheel was provided with a brake mechanism. There were added a convenient handle for propelling the vehicle and a fender bar to protect door frames.

A study of figure I will make the description sufficiently complete.

The following equipment is kept on or in the carriage:

- 1 Zeiss hammer lamp, with attached rheostat, wiring and wire spool assembled
- 2 Diffusing lens attachment for the hammer lamp
- 3 Selective aperture diaphragm attachment for the hammer lamp
- 4 Hand condensing lens, for use with the hammer lamp
- 5 Six cadmium-plated dressing boxes
- 6 Three glass-capped medication bottles in each box
- 7 Six packages of toothpick applicators (sterile-wrapped) in each dressing box
- 8 Six packages of dressings for the eye (sterile-wrapped) in each dressing box
- 9 Three packages of cotton balls (sterile-wrapped) in each dressing box
- 10 One enamelware container for a sterile paper cup
- 11 One roll of cellulose adhesive tape and dispenser (table type)
- 12 Two American Medical Association visual acuity test charts in adjustable holders
- 13 Visual acuity test charts for near vision (two forms)
- 14 One O'Brien hand slit lamp
- 15 One Berger loupe
- 16 One small trial case, with lenses and accessories
- 17 One adjustable trial frame
- 18 One red glass in a holder (for the study of muscles)
- 19 One transilluminator (Zeiss)
- 20 One electric retinoscope (L I C H)¹
- 21 One accommodation rule (Berens' combination)
- 22 One vest pocket dark room
- 23 One ophthalmoscope handle, flexible cord and jack plug (assembled)
- 24 One hand perimeter (Schweigger's)
- 25 One hand campimeter (Peter's)
- 26 One Placido disk
- 27 Plates for testing color sense (Ishihara's test)
- 28 One millimeter-inch tape (meter long)
- 29 Instrument for Maddox wing test
- 30 Eight dry cell batteries, one spring reel for the lamp cord, with a receptacle plug (assembled for the current in use)
- 31 One hand pump for the inflation of tires
- 32 One extra bulb for the hammer lamp

The firm of J. H. Penny cooperated in the construction of this carriage.

¹ This instrument was devised by the department of ophthalmology, Long Island College Hospital.

TOPOGRAPHY AND FREQUENCY OF COMPLICATIONS OF UVEAL SARCOMA

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From 1928 to 1935 inclusive, 995 eyeballs were examined at the Eno Laboratory of the New York Eye and Ear Infirmary. Of this number, 126 (12.8 per cent) were found to have sarcoma of the uvea. In addition to these tumors, 62 other uveal tumors the histologic sections of which were available were studied. This material constitutes the basis of this report.

The chief purpose of this paper is to present the results of a study of this series of uveal tumors from the standpoint of topography and to discuss the frequency of the complications arising from such growths. Other interesting information which was correlated in studying the sections is also reported.

TABLE 1—*Classification of Uveal Tumors*

Site	Present Series	Fuchs	Lawford and Collins
Sarcoma of choroid alone	164 (88%)	221 (85%)	94 (91%)
Sarcoma of choroid and ciliary body	2 (1%)		2 (2%)
Sarcoma of ciliary body	4 (2%)	22 (9%)	6 (6%)
Sarcoma of ciliary body and iris	9 (4.5%)		
Sarcoma of iris	9 (4.5%)	16 (6%)	1 (1%)

Table 1 shows how the tumors in this series were grouped and how this classification compared with that of other authors. Fuchs¹ collated all previous papers on sarcoma of the uvea and added 22 cases of his own to those already reported, making a total of 259. Lawford and Collins² reviewed the reports of 103 cases.

In connection with the data in table 1, it must be stated that the point of origin of the tumor which extended over more than one part of the uvea was extremely difficult to determine.

SARCOMA OF CHOROID

For purposes of study, the tumors of the choroid alone and of the choroid with extension to the ciliary body (166) were divided on the following bases:

Site—The choroid is commonly divided into an anterior, an equatorial and a posterior zone. I have subdivided these zones, in turn, into

1 Fuchs, E. Das Sarkom des Uvealtractus, Vienna, Wilhelm Braumüller, 1882.

2 Lawford, J. B., and Collins, E. T. Ophth. Hosp. Rep. London **13** 12, 1891.

a nasal and a temporal region and these again into a superior and an inferior region. Although tumors of the circumpapillary and macular regions are located posteriorly, they were classified separately, as is shown in table 2.

As can be seen from table 2, of 166 sarcomas of the choroid, 86 (51.7 per cent) were located posteriorly in contrast to 33 (19.8 per cent) located anteriorly and 27 (16.2 per cent) located equatorially. The largest individual sector was the posterior temporal region, where 41 (24.7 per cent) of the choroidal sarcomas were found, whereas only 8 (4.8 per cent) were located posteriorly and nasally. The total number of tumors in the temporal zone, that is, in all sections of this zone amounted to 62 (37.3 per cent), while the total number of tumors

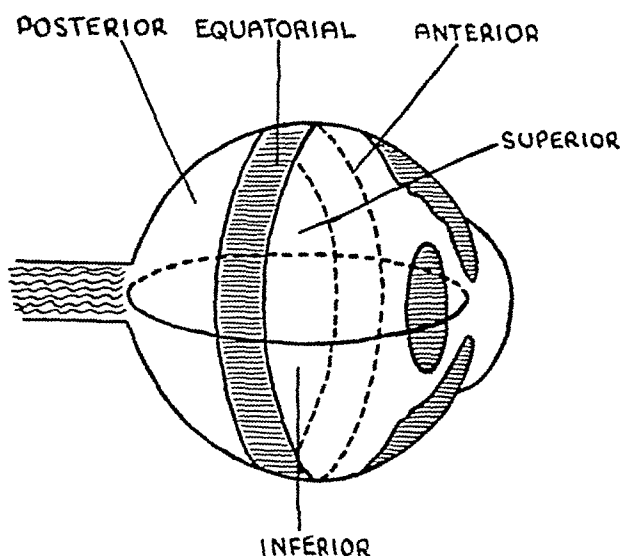


Diagram illustrating the division of the eyeball into zones

in the nasal region amounted to 23 (13.8 per cent). It is of interest to note that 14 tumors were located in the circumpapillary region and were beginning to encroach on the disk. Forty-seven tumors (28.2 per cent) could not be localized temporally or nasally. The foregoing material, however, is sufficient to justify the conclusion that most sarcomas of the choroid arise posteriorly and especially temporally. This is of course, fortunate since central vision is affected early and attention is drawn to the eye.

In table 3 is presented a comparison of my findings in regard to location with those of Fuchs¹ and of Lagrange,³ as given by Lagrange's pupil Baudouin. The data for my series are more conclusive because of the greater number of localizations ascertained.

3 Lagrange, F. *Traité des tumeurs de l'oeil*, Paris, G. Steinheil, 1901.

TABLE 2—*Topographic Distribution of Sarcoma of the Choroid*

Site	T	TS	TI	Mae	Total		N	NS	NI	Total		O	Per centage	Und No	Und Per centage	Future Series	
					Total No	Per centage				Total No	Per centage					Total No	Total Per centage
Anterior segment	9	1	1		11	6.6	8	1	1	10	6.0			12	7.2	33	19.8
Equatorial segment	7	0	3		10	6.0	5	0	0	5	3.0			12	7.2	27	16.2
Posterior segment	20	7	7	7	41	24.7	7	0	1	8	4.8	11	8.1	23	13.8	86	51.7
Whole globe																8	4.9
Half globe																8	4.9
Location unknown																1	2.5
Total					62	37.3				23	13.8	11	8.4	47	28.2	166	100

* T indicates the temporal region, TS, the temporal superior region, TI, the temporal inferior region, Mae, the macular region, N, the nasal region NS, the nasal superior region, NI, the nasal inferior region, and C, the circumpapillary region Und indicates sarcomas, the location of which was undetermined

Age—The average age of the patients with sarcoma of the choroid in the present series was found to be 52.6 years, the youngest patient being 14 and the oldest, 84. Fuchs found the average age of 259 patients with uveal sarcoma to be 42.2 years. Lawford and Collins ascertained it to be 48.42 years, and Pawel⁴ gave the average age as 48.7 years.

Sex—The distribution of the tumors according to sex is presented in table 4. A comparison of my findings with those of other authors is made. As can be seen the tumors occurred equally in the two sexes.

TABLE 3—*Comparison of the Distribution of Tumors in the Present Series with That Reported by Other Authors*

Site	Present Series	Fuchs	Lagrange
Anterior segment	33 (19.8%)	14	38
Equatorial segment	27 (16.2%)	12	7
Posterior segment	86 (51.7%)	74	24
Whole globe	8 (4.9%)		
Half globe	8 (4.9%)		
Unknown	4 (2.5%)	121	61

TABLE 4—*Distribution of Tumors on the Basis of Sex*

Sex	Present Series	Fuchs	Lawford and Collins	Pawel
Male	54 (36.4%)	137 (53%)	59 (56%)	47 (47%)
Female	44 (25%)	168 (44.7%)	44 (44%)	33 (53%)
Unknown	68 (38.6%)	6 (2.3%)		

TABLE 5—*Distribution of Tumors on the Basis of the Eye Affected*

Site	Present Series	Fuchs	Lawford and Collins	Pawel
Right eye	39 (23.4%)	108	41	43
Left eye	36 (21.6%)	101	60	48
Bilateral		5		
Unknown	91 (55%)	45	2	9

Eye Affected—The data found by other authors with regard to the eye affected are present in table 5 for comparison with similar data for the present series.

Pigmentation—At one time sarcomas of the choroid were divided into pigmented and nonpigmented growths, the latter being the leukosarcomas. The opinion that all tumors possess a prepigment substance which is converted into pigment by oxidation, as expressed by Samuels,⁵ has gained ground. This conversion varies according to the circulatory supply. Thus some tumors are pigmented in some areas and

4 Pawel Arch f Ophth 49 1, 1900

5 Samuels, B. Pennsylvania M J 36 895, 1933

not in others. If one examines sufficient sections, it will be found that practically all tumors are pigmented. The findings in the present series of tumors in regard to pigmentation are presented in table 6 and are compared with those of Fuchs¹ and Pawel.⁴ All available sections of the 33 nonpigmented sarcomas were examined, but the evidence is not conclusive, since the entire tumor was never sectioned. If other sections had been taken, pigment might have been found. However, the predominance of pigmented tumors is apparent from the figures given.

Types of Cells—The types of cells found in the sarcomas were either round, spindle or mixed. I found however that a sharp demarcation between the types of cells is not possible. The same cell, when sectioned at a different angle, may have a different appearance. Round cells were found in 47 of the 166 tumors in this series, spindle cell in 54 and mixed round and spindle cell in 41. The type of cells was not determined in the remainder of the growths. Fuchs reported that 65 of 147 sarcomas of the choroid were of the spindle cell type, 40 were of the round cell type and 28 were of the mixed round and spindle cell type, the remainder were of various types.

TABLE 6—*Distribution of Tumors on the Basis of Pigmentation*

Type	Present Series	Fuchs	Pawel
Pigmented	127 (76.5%)	229 (88.4%)	46 (78%)
Nonpigmented	33 (19.9%)	30 (11.6%)	13 (22%)
Unknown	6 (3.6%)		

Shape—No definite figures were given by any of the foregoing authors as to the shape of the tumors. Yet the determination of the shape is valuable for prognosis, since an early tumor is inclined to be flat because it is compressed by the lamina vitrea on one side and by the sclera on the other. The tumor then grows in the perichoroidal space without meeting much resistance until it reaches the attachment of the ciliary body anteriorly or the zone in which the choroid is bound to the sclera posteriorly. At this stage the tumor has the shape of a loaf of bread. The tumor continues to increase in size and since the lamina vitrea offers the least resistance, it breaks through at some point, forming a hole, and the subsequent growth results in a mushroom-shaped tumor. Further growth results in a spherical mass, which eventually occupies a lesser or a greater portion of the eyeball. The distribution of the 166 tumors in regard to shape is presented in table 7.

COMPLICATIONS

The following complications may be encountered in cases of uveal tumor.

Detachment of the Retina—The retina immediately surrounding the growth is not necessarily detached from the underlying choroid. In

some cases it remains attached to the head of the tumor. In most cases, however, albuminous fluid collects underneath the retina which, according to Parsons,⁶ is a result of the irritation set up in the choroid by the tumor and, according to Samuels,⁷ is a result of transudation from the choroidal vessels due to a disturbance of the circulation by the growing mass. Frequently, however, a retinal detachment also arises in other parts of the retina because of the sinking of the fluid. Sometimes a large detachment develops and hides the tumor behind it, making diagnosis somewhat difficult.

In the present series, detachment occurred in 127 cases, it did not take place in 37 cases, and in 2 the status of detachment was not determined.

It is of interest to note that sarcomas of the circumpapillary region almost always cause detachment of the retina. Of 14 such tumors, 13 (92.8 per cent) were complicated by detachment. The frequency of the occurrence of detachment was about the same for all the other tumors.

TABLE 7—*Distribution of Tumors on the Basis of Shape*

Shape	Number	Percentage
Flat	18	7.8
Loaf of bread	27	10.3
Spherical	27	10.3
Mushroom shaped	61	36.7
Irregular	10	9.0
Whole globe	8	4.8
Half globe	5	3.0
Unknown	9	5.5

Glaucoma.—Glaucoma is a typical outcome of uveal tumor and is considered to mark the second stage in the development of the growth. Eighty-two of the 166 eyes were glaucomatous, 89 were nonglaucomatous and the condition of 4 with reference to glaucoma was not determined. The glaucoma is not directly due to the new growth but is caused by the pressure of the subretinal fluid, which presses forward on the lens and the iris. Since frequently in these cases no preoperative histories were available, the criteria of glaucoma in the microscopic sections were an occluded anterior iris angle, patches of degeneration in the iris and abnormal excavation of the disk with atrophic changes of the nerve fibers.

In regard to the relation of the topographic localization of the tumors and the frequency of glaucoma as a complication, it was noted that they were about equal (table 8).

⁶ Parsons, J. H. Pathology of the Eye, London, Hodder & Stoughton, 1905, vol. 2.

Extraocular Extensions—Extraocular extensions of the growth take place along the perforating vessels. From here they continue to grow into the orbit, and at the same time they may also metastasize to other parts of the body. In the present series of tumors the presence of tumor cells in the perforating vessels was considered as evidence of extraocular extensions. Thus, I found that 60 (36.1 per cent) of the tumors showed extraocular extensions, while 102 (61.4 per cent) did not. The status of 4 (2.5 per cent) of the tumors in regard to these extensions was not determined.

The site of the tumor did not seem to have any influence on the frequency of extraocular extensions (table 9).

Necrosis—Another possible outcome of choroidal sarcoma is necrosis of the tumor tissue. In some cases of sarcoma a severe rubeocyclitis breaks out. Anatomically, this has been found to be due to

TABLE 8—*Relation of Topographic Localization and the Presence of Glaucoma*

Segment	Glaucoma	No Glaucoma
Anterior	16 (53.3%)	14 (46.7%)
Equatorial	13 (48.1%)	14 (51.9%)
Posterior	39 (45.4%)	47 (54.6%)

TABLE 9—*Data on Extraocular Extensions in Relation to Distribution of Tumors*

Segment	Extraocular Extensions	No Extraocular Extensions
Anterior	12 (40%)	18 (60%)
Equatorial	8 (29.7%)	19 (70.3%)
Posterior	27 (32.4%)	59 (68.6%)

toxins resulting from the tumor. Deficient nutrition due to poor circulation of the blood is the cause of the necrosis. Samuels,⁷ in his paper on "Anatomic and Clinical Manifestations of Necrosis in Eighty-Four Cases of Choroidal Sarcoma," amply and fully described this result. He stated that of 106 sarcomatous eyes, 31 were highly necrotic. In only a small percentage of these was necrosis suspected preoperatively. Twenty of 31 eyes were enucleated under other diagnoses. No studies of necrosis were made in this series, and nothing further was added to Samuels' conclusions.

In studying the present series of tumors it was found to include 10 metastatic carcinomas of the choroid the primary origin of which could not be ascertained. Histologically 6 of them were adenocarcinomas and 4 were medullary carcinomas. Four were in the circum-papillary region, and 4 occupied the posterior part of the eye. In 2

7 Samuels, B. Anatomic and Clinical Manifestations of Necrosis in Eighty-Four Cases of Choroidal Sarcomas, Arch Ophth **11** 998 (June) 1934.

cases the original site could not be located because the tumor occupied the entire globe. The posterior location of these tumors indicated that the metastases were arrested in the larger vessels of the choroid.

SARCOMA OF THE CILIARY BODY

In this series of tumors there were 4 (2 per cent) sarcomas of the ciliary body alone and 9 (4.5 per cent) of the ciliary body with extension to the iris. Fuchs found the percentage to be the same in his series. It was difficult to determine definitely the site of origin of the sarcomas of the ciliary body and iris and to state how many extended from the ciliary body to the iris or vice versa. It seemed most probable that the place of origin was the ciliary body. Histologically, all tumors of this group were found to be mixed round and spindle cell sarcomas. Of the total number of 13 tumors, only 1 was not pigmented. The average age of the patients in this group was 50.1 years.

SARCOMA OF THE IRIS

In this series there were 9 (4.5 per cent) sarcomas of the iris. Histologically, they were mixed sarcomas. All 9 were pigmented. The average age of the patients was 40.3 years. Fuchs¹ in his series found the average age to be 31 years. The average age of persons with sarcoma of the iris has shown itself to be much lower than that of persons with sarcoma of the choroid.

SUMMARY AND CONCLUSIONS

In regard to the topographic distribution of choroidal sarcomas, it was found that most tumors are localized posteriorly and especially temporally.

In the present series 12.8 per cent of all enucleated eyes were removed because of uveal sarcomas.

Eighty-eight per cent of all uveal sarcomas are located in the choroid.

The average age of persons with sarcoma of the choroid was found to be 52.6 years.

Sarcomas of the choroid are distributed equally in the two sexes.

Most choroidal tumors (76.5 per cent) are pigmented.

The spindle cell sarcomas predominate over the round cell type.

The most common choroidal sarcomas on the basis of shape are those shaped like a loaf of bread and a mushroom.

Detachment of the retina occurs in more than 75 per cent of all cases of choroidal sarcoma.

Almost all circumpapillary sarcomas cause detachment of the retina (92.8 per cent).

One half of the eyes enucleated for sarcoma showed glaucomatous changes. The topographic distribution does not influence the frequency of glaucoma.

Extraocular extensions were found in 36.1 per cent of all cases, in 61.4 per cent there were no extensions. The site of the sarcoma has no bearing on the frequency of extensions.

Metastatic tumors of the choroid appear principally in the posterior segments.

Sarcomas of the ciliary body frequently involve the iris. They are generally nonpigmented.

The average age of patients with sarcoma of the iris is lower than that of patients with sarcoma of the choroid, that is, 40.3 years.

INOSITOL IN THE OCULAR TISSUES

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AND

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The knowledge of the chemical composition of most tissues is yet unsatisfactory. A few organs have been studied intensively, and their composition is relatively well known, on the contrary, for many other organs systematic data are still lacking. To undertake a study of physiologic chemistry before having a sufficient knowledge of "anatomic chemistry" results in incomplete or even false interpretations.

The analyses of the ocular tissues are fairly complete. This is particularly true for the lens. For 100 Gm. of lens, the composition of about 98.5 Gm. is known. The remaining 1.5 Gm. contains substances which, acting as catalysts, play an important role in spite of their low concentrations. Other substances enter the lens by diffusion, they do not act as metabolites, and their role is unimportant. But although the unknown fraction of the lens is a small part of the total weight, investigation of it may reveal the presence of substances reaching high concentrations, even higher than that of dextrose. Inositol is such a substance and is the subject of this article. This investigation, however, has not been limited to the lens but has been extended to all ocular tissues.

Inositol (cyclohexanhexol) was discovered in 1850 by Scherer. Because of its sweet taste, this substance was for a long time considered as a sugar and was called inosite or *Muskelzucker*. It is known to be widely distributed in both animal and plant tissues. A great interest arose in this substance when it was discovered that phytin was the phosphoric ester of inositol and that the enzyme, phytase, occurred in many animal tissues. Later, inositol was found in the urine of persons with diabetes insipidus and diabetes mellitus. The discovery of inosituria resulted in contradictory interpretations. It is actually well demonstrated that any polyuria, with or without glycosuria, is accompanied by inosituria. Inosituria is a consequence of the polyuria, not of the glycosuria.

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The role of inositol in the tissues, although still actually unknown, seems to be important in the biologic processes. Its presence and its distribution in the ocular tissues have not yet been the subject of experimental investigation. For this reason this work has been undertaken.

EXPERIMENTAL PROCEDURE

Normal eyes from 3 or 4 year old cattle were immediately removed after slaughter of the animal, dissection of the ocular tissues was started as soon as possible. An unavoidable delay of a few hours was, however, necessary before the extraction was begun. Since inositol is stable, its destruction was unlikely, on the contrary, a slight increase arising from postmortem changes is possible.¹

The method of Young² for the determination of inositol was used. Samples of corneal epithelium and conjunctiva weighing from 20 to 30 Gm, of vitreous weighing from 500 to 1,000 Gm and of other tissues weighing from 60 to 150 Gm were used.

The samples were extracted during two periods of twenty-four hours with a 70 per cent aqueous solution of acetone. For the first extraction, sufficient acetone was added to make a 70 per cent aqueous solution, depending on the amount of water in the tissue.

The acetone of the extract was removed by evaporation on the water bath. The remaining aqueous solution was extracted twice with ether and then evaporated to 100 cc on the water bath. Extraction with acetone and ether gives better results than hydrolysis with hot alkali because it is more specific. All the tissues were extracted in this manner with the exception of the vitreous humor, which was desiccated before extraction.

Many interfering substances were removed by precipitation with mercuric sulfate in acid solution. After neutralization, the solution was filtered. The mercury was removed by means of hydrogen sulfide and filtration.

Barium hydroxide and 95 per cent ethyl alcohol were used to precipitate inositol. The precipitate was separated by centrifugation and was hydrolyzed by heating after acidification. The barium was removed by filtration, and the clear filtrate was reduced to a small volume. The inositol was precipitated by means of acetone and ether.

The precipitate formed under these conditions was not pure inositol. A certain amount of salts passed during these different procedures, depending on the tissue. The percentage of inositol was determined by a method of titration which is based on a reduction of potassium iodomercurate in alkaline solution. For this titration, an aliquot of solution of the precipitate containing 2 to 5 mg of inositol was used. After the addition of sulfuric acid, an excess of iodine was added and titrated with thiosulfate.

RESULTS

The accompanying table shows the distribution of inositol in ocular tissues.

1 Winter, L. B. Inositol Metabolism in the Mammalian Heart, *Biochem J* **28** 6, 1934.

2 Young, L. Determination of Inositol in Animal Tissues, *Biochem J* **28** 1435, 1934.

The high concentrations of inositol in the lens and in the optic nerve permitted the isolation of the substance in almost pure form. The inositol obtained from the lens at the second crystallization had a melting point of 220.5 C (uncorrected), that of the optic nerve had a melting point of 222 C (uncorrected). The melting point of a commercial inositol was 223 C (uncorrected). The inositol isolated from the lens was optically inactive like that isolated from the brain.³

The amount of inositol in the vitreous humor was small. As a rule, the figures obtained by titration were lower than 2 mg in 100 Gm of vitreous humor. It was questionable whether this slight reduction was due to inositol or to compounds liberated during the procedure. An attempt to isolate it was unsuccessful. Inositol in the vitreous humor, if present, was probably a product of diffusion and not a metabolite.

Inositol in Ocular Tissues in Milligrams per Hundred Grams of Tissue

Whole Cornea		Corneal Epithelium		Corneal Stroma		Conjunctiva		Iris			
Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry		
8.0	42.9	26.5	132.5	5.3	28.2	6.05	33.4	22.0	118.0		
6.5	34.8	17.2	86.6	5.1	27.2			27.5	147.5		
		15.9	76.6					35.0	187.5		
Lens		Vitreous Humor		Retina		Choroid*		Sclera		Optic Nerve	
Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry	Wet	Dry
150	395	Traces	—	13.4	107.5	35.5	206.8	14.0	50.5	103	342
172	450	Traces	—	16.6	133.0	32.5	187.8	10.5	37.8	96	321
154	405	Traces	—	17.0	136.2	29.2	168.5	13.5	48.6	89	298
126	330	Traces	—	14.5	116.3					111	372
177	465	Traces	—							90.5	303
149	392										

* The retinal pigment epithelium was removed with the choroid.

COMMENT

An excellent survey of the literature up to 1926 has been made by Needham.⁴ In spite of rather large amounts of information, knowledge concerning metabolism of inositol is still scanty.

Since inositol is widely distributed in animals and plant tissues, the amount daily ingested in the food is relatively high. However, inositol as such is destroyed by the intestinal bacteria, and only a small fraction is absorbed through the intestinal walls. The inositol is likely absorbed in the form of phytin.

³ Momose, G. On the Inositol of Brain and Its Preparation, *Biochem J* 10:120, 1916.

⁴ Needham, J. Die physiologische Bedeutung der Cyclosen, *Ergebn d Physiol* 25 1, 1926.

Inositol in the blood reaches only low concentrations⁵ The concentrations in tissue are much higher and cannot be explained by simple diffusion From beef brain, Thudichum⁶ has isolated 10 Gm of pure inositol from 50 pounds (22,679 Gm) of tissue, or 44 mg per hundred grams For human brain, the figures given by the same author are higher 193 mg per hundred grams in the gray matter and 217.2 mg per hundred grams in the white matter Since this early publication, the data found in the literature show a wide range of variation depending on the method used and the experimental conditions According to recent determinations, inositol can reach concentrations higher than 170 mg per hundred grams in the brain About 85 mg per hundred grams is found in fresh heart muscle, and about 125 mg per hundred grams is found in the same tissue some hours after death The amount of inositol in skeletal muscle seems to be much lower⁷

Furthermore, previous investigations show that the tissues are able to synthesize inositol for neither suppression of inositol from the food for a long period nor polyuria with inosituria is able to decrease the amount of inositol in the animal tissues Since the injection of dextrose increases the amount of inositol in the incubating egg and since the injection of insulin decreases the sugar in the blood and increases the inositol in the tissues, it may be supposed that dextrose plays a role in the synthesis of inositol⁸

The question arises as to the nature of the inositol in the tissues Since the concentration of inositol increases with the autolysis, the hypothesis of a precursor of inositol, inositogen, has been suggested⁹ It is possible that the inositol itself is partly free and partly bound with a molecule containing phosphorus The present methods do not allow more than an estimation of these two fractions¹

The final fate of inositol is unknown It has been suggested that inositol is changed into glycogen dextrose or perhaps into lactic acid in the vertebrate cardiac muscle¹⁰ However, inositol in the food does

5 Needham, J Studies on Inositol II The Synthesis of Inositol in the Animal Body, *Biochem J* **18** 891, 1924

6 Thudichum, J L W Die chemische Konstitution des Gehirns des Menschen und der Tiere, Tubingen, Franz Pietzcker, 1901, pp 40 and 276-278

7 Gregory, R A A Modification of Young's Method for Determination of Inositol in Animal Tissues, *Biochem J* **29** 2798, 1935 Winter¹ Young²

8 (a) Needham, J Studies on Inositol III The Metabolic Behaviour of I-Inositol in the Developing Avian Egg, *Biochem J* **18** 1371, 1924 (b) Needham, J, Smith, W, and Winter, L B Insulin and Inositol, *J Physiol* **57** LXXXII, 1923

9 (a) Rosenberger, F Weitere Untersuchungen uber Inosit, *Ztschr f physiol Chem* **64** 341, 1910 (b) Needham, J Studies on Inositol I A Method of Quantitative Estimation, *Biochem J* **17** 422, 1923

10 Boyland, E Chemical Changes in Muscle II Invertebrate Muscle, III Vertebrate Cardiac Muscle, *Biochem J* **22** 362, 1928

not increase the respiratory quotient,¹¹ and perfusion of tissue with inositol gives no formation of dextrose or lactic acid¹² The experiments done with the Warburg apparatus are not consistent¹³

It has been suggested that inositol has no other role than that of bringing phosphorus into the organism¹⁴ If this were true, why does the organism show an active synthesis of inositol and why is there a constant concentration of inositol in the tissue when the diet is free from inositol and or when there is prolonged polyuria with inosituria? Hypotheses on the subject will not add anything more, for experimental facts are missing

The results of the investigations show that the concentration of inositol varies greatly with the type of ocular tissues It is high in the lens and in the optic nerve Further experimental work will perhaps give a clue to the relation of metabolism to these high concentrations

CONCLUSIONS

Inositol was found in each of the bovine ocular tissues, namely conjunctiva, corneal epithelium, corneal stroma, iris, lens, retina, choroid, sclera and optic nerve The presence of inositol in the vitreous humor is questionable

Its concentration is relatively constant in a definite type of tissue but varies broadly in different tissues The concentration is remarkably high in the lens (150 mg per hundred grams of wet tissue) and in the optic nerve (100 mg per hundred grams of wet tissue)

The role played by inositol in these tissues is still unknown

To consider inositol as a stable form of metabolite in the tissues which have no glycogen or a small amount of it is a good working hypothesis

11 Anderson, R. J. The Utilization of Inosite in the Dog, *J. Biol. Chem.* **25** 391, 1916

12 (a) Oppenheimer, S. Ueber die Milchsäurebildung in der künstlich durchströmten Leber, *Biochem. Ztschr.* **45** 30, 1912 (b) Griesbach, W., and Oppenheimer, S. Ueber Milchsäurebildung im Blut, *ibid.* **55** 328, 1913 (c) Embden, G., and Griesbach, W. Ueber Milchsäure und Zuckerbildung in der isolierten Leber I. Ueber den Abbau der d-Sorbose, II. Ueber das Schicksal des d-Sorbit und einiger anderer Hexite, *Ztschr. f. physiol. Chem.* **91** 284, 1914

13 (a) Das, N., and Guha, B. C. The Biological Oxidation of Inositol, *Current Sc.* **3** 157, 1934, abstracted, *Chem. Abstr.* **29** 1110, 1935, (b) Die Unwandlung von Inosit durch Rattengewebe, *Ztschr. f. physiol. Chem.* **231** 157, 1935 (c) Young, L. Inositol and the Respiration of the Brain, *Proc. Soc. Exper. Biol. & Med.* **35** 507, 1936

14 Starkenstein, E. (a) Die Beziehungen der Cyklosen zum tierischen Organismus, *Ztschr. f. physiol. Chem.* **58** 162, 1908, (b) Die biologische Bedeutung der Inosit Phosphorsäure, *Biochem. Ztschr.* **30** 56, 1911

Clinical Notes

A SPECIFIC TREATMENT FOR HERPES ZOSTER

J R WALKER, M D, AND B F WALKER, M D
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Herpes zoster of the eyes and forehead and sometimes of the side of the nose is one of the most stubborn diseases with which the ophthalmologist has to deal. The pain is often excruciating and sometimes cannot be relieved except by the administration of large doses of morphine.

For many years it has been known that when herpes occurred along the distribution of the fifth cranial nerve the eye of the side affected became involved. The sensory nerve fibers that supply the iris and some other parts of the eyeball are branches of the nasociliary nerve which also supply the cornea, sensory branches of the fifth nerve also supply the eyelids, the conjunctiva, the caruncle and the lacrimal sac. Since herpes zoster follows the course of the fifth nerve and its numerous branches, one can see how the vesicles can be so widely distributed on the side of the face.

Herpes zoster is comparatively rare and may be mistaken for erysipelas. Such a mistake should not be made, especially by one who has ever seen a case of herpes. The symptoms are different. Herpes zoster usually develops to its entire extent at once and is seldom accompanied by fever, while erysipelas usually starts from one point and spreads and is accompanied by fever, which may reach from 103 to 106 F. Herpes involves only one side of the face, while erysipelas may spread over the entire face, the neck and the head. The onset of herpes is usually sudden, and the vesicles do not spread. Some patients suffer from pain for months after the eruption has subsided. After the vesicles have healed, there is a permanent scar. Sometimes paralysis of some of the ocular muscles occurs.

It has been our experience that we do not see patients with herpes zoster at the onset of the disease unless there is involvement of the cornea or the lids. Most patients are treated by their family physician and consult an ophthalmologist only after the condition is of one or two months' duration. However, in the past few months we have seen 2 patients with herpes in the acute initial stage.

ETIOLOGY

As to the cause of herpes zoster we are still ignorant. We do not know whether it is of central or of peripheral origin. Some authors have suggested that it is caused by cold and some that it is caused by internal toxins. Stein¹ stated that it is caused by an infection passing from the sensory ganglion down the course of the sensory nerve to the

1 Stern, E. S. Brit J Dermat 49 263, 1937

skin. According to him, herpes zoster and chickenpox are the result of the same infection. He scarcely mentioned treatment. The condition is said to have been known to ophthalmic surgeons since 1866. A good description may be found in any textbook on ophthalmology, but most all of them deal briefly with the treatment.

TREATMENT

We recently reviewed the literature on herpes zoster and found that most writers consider medication to be unsatisfactory. Many report what they deem to be good results with the use of a solution of posterior pituitary. The library package of the American College of Surgeons gives little on the treatment. We have just reviewed the literature on the treatment of herpes zoster as sent us in the package library of the American Medical Association and find the following methods of treatment outlined.

Barksdale² reported good results with autohemotherapy. Vorhaus³ used vitamin B₁ without positive results. Somers and Pouppirt⁴ obtained good results with a solution of pituitary. Ruggles⁵ in 1931 reported what seem to us better results with sodium iodide than with any other type of treatment. Beeson⁶ reported excellent results with autohemotherapy. Franchini⁷ reported a case of herpes zoster of the epiglottis. His treatment consisted of drugs to relieve pain. Pituitary preparations are used by a great many physicians, but the results are usually slow. Niles⁸ reported several cases in which a solution of posterior pituitary was used, 5 of the patients were well in eleven days, 1 was improved but the pain continued, 4 were no better, and the results for 6 were unknown. We have never seen recurrent herpes zoster. In Norris and Oliver's⁹ "System of Diseases of the Eye" there are three lines about treatment. Gifford¹⁰ in his book on ocular therapeutics gave only a short outline on treatment.

About twenty years ago we began a treatment which has been used by us to the exclusion of all others, and that is the injection of diphtheria antitoxin. We administer 5,000 units of the antitoxin, and if necessary we give another dose of the same amount two days later. Usually the pain ceases after the first dose, and the inflammation disappears rapidly. In only 2 cases have we given a third dose, and in 1 we do not believe

2 Barksdale, E. E. *Virginia M. Monthly* **64** 378, 1937.

3 Vorhaus, M. G. *Am. J. Digest. Dis. & Nutrition* **3** 915, 1937.

4 Somers, M. R., and Pouppirt, P. S. *California & West Med* **42** 370, 1935.

5 Ruggles, E. W. *Apparent Specific Effects of Sodium Iodide in Herpes Zoster*, *Arch. Dermat. & Syph.* **23** 472 (March) 1931.

6 Beeson, B. B. *Autohemotherapy in the Treatment of Herpes Zoster*, *Arch. Dermat. & Syph.* **18** 573 (Oct.) 1928.

7 Franchini, Y. *Semana med.* **1** 1323, 1937, *Rev. Asoc. med. argent.* **51** 53, 1937.

8 Niles, H. D. *New York State J. Med.* **32** 773, 1932.

9 Norris, W. F., and Oliver, C. A. *System of Diseases of the Eye*, Philadelphia, J. B. Lippincott Company, 1897-1898.

10 Gifford, S. R. *A Handbook of Ocular Therapeutics*, ed. 2, Philadelphia, Lea & Febiger, 1937.

that it would have been necessary. Some of the patients had had the pain for months. In a recent case pain had been present for nine months in spite of other treatment, but relief was obtained after the first dose of 5,000 units of diphtheria antitoxin. Other physicians whom we have induced to use the treatment report the same results. Some have asked if it was not the horse serum that was responsible for the cure. Since the use of concentrated diphtheria antitoxin, the results have been just as good.

While this paper was in preparation, we saw a patient with the most severe herpes zoster that it has been our lot to see.

W. R., a white man of about 50 years of age, was seen on Jan. 15, 1938, with extensive herpes involving both eyelids on the left side and extending up over the brow and forehead to the crown of the head. The blisters were the largest we had ever seen. The man was suffering from intense pain. The attack was of three days' duration when we saw him. We immediately gave him 5,000 units of diphtheria antitoxin. We saw him again on January 17, the acute pain was gone, and there were no new blisters. We gave him 5,000 units of the antitoxin, and on January 18 we gave him 5,000 more units. At that time the pain had ceased, and the blisters had begun to dry up. From then on he had no more pain and no increase of the herpes. We did not administer any more antitoxin, and the patient made a rapid recovery.

CONCLUSIONS

In our experience diphtheria antitoxin, as used by us, has proved to be a specific treatment for herpes zoster in the acute as well as in the chronic stage. It usually gives relief from pain within twenty-four hours, and the cure is permanent.

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Ophthalmologic Review

DETACHMENT OF THE RETINA

SUMMARY OF THE MODERN OPINIONS

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To comprehend satisfactorily the status of the new and modern theories concerning detachment of the retina, one must have a reasonable understanding of the growth of the knowledge of this subject. To this end a few facts and opinions have been gleaned from encyclopedias and numerous reviews of the subject as well as from many original articles.

The early recognition of this condition, before the ophthalmoscope was invented, will be considered first. According to numerous authors, de Saint-Yves¹ in 1722 was the first to describe anatomically a condition called *amotio sive sublatio retinae*. After the appearance of this description, numerous articles were published in which the condition was mentioned, but no real advance was made for nearly a century. Ware² in 1805, Wardrop³ in 1818 and Panizza⁴ in 1821 are credited with histologic descriptions. These students examined specimens and differentiated *hydrops subchoroidal* from *hydrops subsclerotic*.

In 1830 Mackenzie⁵ wrote of a watery fluid being present between the sclerotic and the choroid in cases of sclerotic staphyloma. He stated

There are also good grounds for believing that a similar effusion forms occasionally between the choroid and the retina. If the fluid is not evacuated by puncturing the staphyloma, it may accumulate to such a degree as to press the retina before it, and having at last produced, by means of its continued pressure, an absorption of the vitreous humor, it will gather the retina into a cord.

The condition was treated by thrusting a broad cataract needle toward the center of the vitreous, but not deeper than $\frac{1}{8}$ inch (0.32 cm).

This review is based on a course given at the American Academy of Ophthalmology and Otolaryngology.

1 de Saint-Yves, C. *Nouveau traité des maladies des yeux*, Paris, P. A. Le Mercier, 1722, pp. 8 and 331.

2 Ware, J. *Chirurgical Observations Relative to the Eye*, ed. 2, London, J. Mowman, 1805, vol. 1, p. 168.

3 Wardrop, J. *Essays on the Morbid Anatomy of the Human Eye*, ed. 2, London, A. Constable & Co., 1818.

4 Panizza, B. *Annotazioni anatomico-chirurgiche sul fungo midollare dell'occhio*, Pavia, P. Bizzoni, 1821.

5 Mackenzie, W. *A Practical Treatise on the Diseases of the Eye*, London, Longman [and others], 1830, p. 460.

The first person seemingly to recognize retinal detachment in situ was von Chelius,⁶ who in 1839 observed it through a dilated pupil as a white membrane

After the invention of the ophthalmoscope, Coccius, van Trigt, Arlt⁷ (1853) and von Graefe⁸ (1854) described the ophthalmoscopic appearances of detachment of the retina

It is interesting to note that in the first article by von Graefe on this subject, which appeared in the first volume of the *Archiv für Ophthalmologie*, trauma is noted as the cause of retinal detachment in certain cases, also chorioretinitis and a tendency to bleeding (hemorrhoids, epistaxis and other conditions in which bleeding is often severe) This article contains one of the first descriptions of retinal detachment after the invention of the ophthalmoscope Von Graefe noted the color of the detached areas as well as the position of the detachment Some of the areas were yellow, some white and some red He expressed the belief that some detachments must be due to hemorrhage, the blood subsequently absorbing, at least in part In this he differed with Arlt, who had contended that a chorioidal effusion was the causative force

Stellwag⁹ disproved this hemorrhagic theory by his anatomic investigations

In 1857 Muller¹⁰ called attention to the existence of contracting connective tissue in the vitreous, which he considered an important etiologic factor In the same year von Graefe¹¹ drew attention to the association of retinal detachment with myopia He expressed the belief that the outer tunics of the eye were more distensible than the retina, which was therefore pulled away as the eyeball enlarged with the development of myopia

In 1869 Knapp¹² described a hole in the macula, and the following year de Wecker¹³ emphasized the frequent occurrence of tears in cases of spontaneous detachment of the retina

"Leber noticed tears and detachment occurring together in eyes with retained foreign bodies He introduced, aseptically, metallic bodies into the vitreous and watched the formation of retinal tears and after a few

6 von Chelius, M J Handbuch der Augenheilkunde, Stuttgart, E Schweizerbart, 1839, vol 2, p 366

7 Arlt, F Die Krankheiten des Auges, Prague, F A Credner & Kleinbub, 1855, vol 2, p 158

8 Von Graefe, A Arch f Ophth (pt 1) 1 362, 1854

9 Stellwag von Carion, C Die Ophthalmologie, vom naturwissenschaftlichen Standpunkte aus bearbeitet, Erlangen, Ferdinand Enke, 1856, vol 2, p 100

10 Muller, H Ztschr f wissensch Zool 8 1, 1857

11 von Graefe, A Arch f Ophth (pt 2) 2 277, 1857

12 Knapp, J H Arch f Augenh 1 1, 1869

13 de Wecker, L Traite des maladies du fond de l'oeil, Paris, A Delahaye, 1870, p 153

days, extensive detachments of the retina”¹⁴ Lindner¹⁵ recently remarked “He [Leber] was the first to say that vitreous detachment precedes detachment of the retina and is caused by peripheral choroiditis” Leber^{15a} divided retinal detachments into three categories (a) acute, characterized by shrinkage of the vitreous and an outpouring of fluid through retinal tears produced by traction of strands of vitreous, (b) chronic, characterized by shrinkage of the vitreous without tears and without fluid passing through the retina, and (c) a type characterized by the formation of a cystic membrane with shrinkage of the vitreous

From time to time increments were added to the knowledge of the subject, but it was not until 1887 that Nordenson¹⁶ made a fairly satisfactory grouping of cases, consideration of these groups led him to develop an interesting theory He stated that examination of 119 persons with separation of the retina revealed one rent or more in over one third of the group According to his theory, the primary lesion is located in the uveal tract, the uveitis is followed by fibrillary metamorphosis and subsequent shrinkage of the vitreous The vitreous becomes firmly adherent to the retina and in contracting not only detaches the retina from its choroidal bed but tears it, through these openings, often minute, the posthyaloid effusion passes If the detachment occurs in the upper part of the globe, the fluid gravitates downward and further tends to increase the detachment

Leber's first theory¹⁴ was based on previously established facts, among these were Arlt's finding of fluid back of the vitreous in cases of advanced myopia and Elschnig's qualification that this occurred in less than half of these cases (Therefore, detachment is not a rule in cases of high myopia) According to Leber's first theory, myopia plus degeneration of the vitreous and contraction of the degenerated fibrils of the vitreous which are fastened to the retina and to the pars plana of the ciliary body cause tearing of the retina, fluid passes through the tear and elevates the retina The degenerative changes develop as follows Far forward on the ciliary body small inflammatory lesions are accompanied by a proliferation of epithelial cells which migrate onto the fibrils of the vitreous, here they have too little nourishment to maintain life, so degenerate and shrink—“fibrillary degeneration of the vitreous” When sufficient shrinkage occurs, the fibrils contract and

14 Leber, T *Tr Internat M Cong* (1881) 3 15, 1882, quoted by Anderson, J R *Detachment of the Retina*, New York, The Macmillan Company, 1931, p 64

15 Lindner, K *Lectures before the Chicago Ophthalmology Society*, 1935

15a Leber, cited in Wood, C A *American Encyclopedia and Dictionary of Ophthalmology*, Chicago, Cleveland Press, 1919, vol 15, p 11212

16 Nordenson, E *Die Netzhautablosung Untersuchungen über deren pathologische Anatomie und Pathogenese*, Wiesbaden, J F Bergmann, 1887, p 4

tug on the retina, this tugging produces the pre-detachment photopsia. Then when a fluid exists behind the vitreous some jerky movement of the eye causes these fibrils to tear the retina.

Leber's second theory, that of "pre-retinitis" (which theory was evolved in association with von Hippel¹⁷ in 1900 and 1908), was developed to explain the presence of small inflammatory masses, occasionally seen, uniting the vitreous and the internal limiting membrane. According to this theory, the shrinkage of the pre-retinal exudate wrinkles the retina, and fluid then gathers beneath it, either through a retinal tear or ex vacuo by transudation.

It is often difficult, it has seemed to me in this study, to determine exactly how one theory differs from another, this is due in part to the point of view of the author considered and to his understanding of the point of view of previous authors, and in part to the fact that the later point of view is different from that of the earlier authors whose works are reviewed here. Also, many reviewers inaccurately attribute to one man a theory propounded by another author. Thus, for example, Siergrist, of Berne, Switzerland, in his discussion of a paper by Gonin which appeared in 1919, gave Gonin credit for the idea of primary chorioretinitic foci in the periphery of the fundus leading to the formation of adhesions of the vitreous, with subsequent shrinkage and the development of retinal folds and tears. These ideas were part and parcel of several previous theories.

Having established this foundation, I shall next consider Gonin's¹⁸ theories, although in so doing some forward steps are necessarily being omitted. Gonin, however, by startling the world in 1919 and the next few years, focused attention on his contribution, which sums up and gives point to many of the previous findings.

Gonin tried to differentiate the various kinds of detachment and the factors producing them and called attention to the usual lack of understanding of these several factors. A careful history of the patient and a detailed examination of retinal detachments at consecutive stages led to a better understanding of the pathologic process, and to the proper treatment. Gonin divided detachments into the following four large groups on the basis of the etiologic factors: (1) those due to pulsion (*soulèvement*), (2) those due to traction, (3) those due to distention and (4) those due to depression (diminution of the size of the globe). According to him, treatment is seldom successful for the last two types of detachment, for the first type, the treatment is mainly directed to the cause. Most of this paper was devoted to a discussion of this type of detachment. In other papers, Gonin reviewed many previous state-

¹⁷ von Hippel, E. Arch. f. Ophth. **51** 132, 1900, **68** 38, 1908.

¹⁸ Gonin, J. Ann. d'ocul. **156** 281, 1919.

ments, agreeing with some and disagreeing with others. His principal contribution to the subject was that the reapplication of the retina is permanent only when the traction exerted on the retina by the vitreous has ceased or is counterbalanced by a sufficient adherence of the retina to the choroid. According to him, such a reapplication can be conceived of only after the closure of the one or more tears of the retina. Such closure was the aim of the treatment, especially closure brought about by the production of an extensive chorioretinal adhesion involving all of the edges of the tears. Gonn's classic method of treatment is well known. He himself has modified his theory from time to time in more or less important details, other investigators have added their quota also, many have overemphasized the importance of these modifications.

I shall now touch on some of these modifications that seem to be of value and shall especially consider the various views on the production of the hole, it having been agreed that a hole is a necessary prerequisite for detachment. In Arruga's¹⁹ recent work on "Detachment of the retina" (published in Spanish and in English), three theories of this mechanism are illustrated, Gonn's, Lindner's and Arruga's. The differentiation appears in parallel columns

Gonn	Lindner	Arruga
19 Retraction of the vitreous with its separation from the retina mainly superiorly	23 Liquefaction of the vitreous in the posterior part	27 Adherence of the vitreous to the retina. It may exist for a long time before energetic trepidation of the vitreous or the motions of the eyeball produce the retinal rent
20 Retraction of the vitreous with a vitreoretinal adhesion superiorly	24 Detachment of the vitreous with adhesion above	28 Ocular movements compel the vitreous to strike the edges of the hole, thus forcing the vitreous behind the retina
21 Formation of a retinal rent due to increased traction of the vitreous	25 Formation of a tear with a flap	29 An elevation of the retina caused by choroidal exudate. This elevation is localized by a chorioretinal adherence
22 Displacement of subretinal fluid downwards	26 Formation of a retinal hole with floating operculum	30 A rupture of the retina at the point where adhesion between the choroid and retina was present. Ocular movements facilitate the progress of the detachment

¹⁹ Arruga, H. Detachment of the Retina, translated by R. Castroviejo, New York, B. Westerman, 1936, pp. 26-27.

In brief, Gonin stated that the shrinking vitreous causes no trouble unless it is fastened securely to the retina (not to the choroid) in a favorable spot, so that in shrinking it will pull the retina loose and tear it at that spot, allowing the retrovitreous fluid to pass through the tear. Lindner emphasized the frequency with which slit lamp examination reveals an optically empty space behind the vitreous, especially in cases of myopia. According to him, if the shrinking vitreous is adherent to the retina, it may pull a flap loose, causing a horseshoe tear, or it may pull off a small button of retina, causing a hole. Arruga expressed an inclination to minimize the effects of shrinking vitreous and emphasized the effect of sudden motions of the eyeball on a diseased retina which is in one or more places slightly adherent to the vitreous. He stated

As a result of previous inflammation (chorioretinitis)²⁰ or degenerative processes (atrophy and cystoid degeneration), the retina becomes adherent to the vitreous in certain places. At the same time it becomes thinner, weaker and retractile, a condition found in all tissues following inflammation or degenerative atrophy. Then, a blow on the head or the eye, even movements of the eyeball may make the adhesion of the vitreous produce a rent in the retina.

It is difficult to decide who really was the first to emphasize the effect of motion, certainly, almost simultaneously the use of pinhole disks in treatment was adopted by nearly all clinics. Arruga further stated that several favoring conditions have to be present for a retina to become detached: (1) hole, (2) retractibility of the retina, (3) adhesion of the retina and the vitreous and (4) an inciting cause, such as a blow on the head or a sudden movement of the eyeball. He emphasized the fact that holes are often present without detachments resulting, e. g. after equatorial sclerotomy (therapeutic) the retina does not detach itself. Detachment associated with pregnancy and nephritis ordinarily occurs without the formation of holes, and spontaneous healing results.

Anderson,²¹ in summing up many opinions, brought out the relation between the degenerative changes in the retina, particularly the anterior third, in cases of myopia and those in cases of retinal detachment. In both conditions the retina is thin and atrophic, especially in the region of the ora serrata, where the formation of a hole is a frequent finding. Also the vitreous is abnormal, degenerate. Anderson was undoubtedly influenced in this opinion by the accumulated experience of many leaders who from the time of Blessig, in 1855 to that of Kopmczynski, in

²⁰ A footnote in Arruga's article at this point reads as follows: "If, experimentally, an inflammation of the membranes of the eye is produced by irritating the sclera, not only do the membranes become adherent to each other, but the vitreous body also becomes adherent at the point where there was inflammation."

²¹ Anderson, J. R. *Detachment of the Retina*, New York, The Macmillan Company, 1931, p. 39.

1929, reported on cystoid degeneration of the retina. Hanssen²² and others collected considerable data on the prevalence of cystoid degeneration of the retina and reasoned that when these cysts broke holes would form. Hanssen expressed the belief that the cysts would break easily if the internal limiting membrane covering the retina were adherent to the vitreous and a slight injury were received at that spot. In this he agreed with Gonnin's statement of four years before.²³

In 1929 Pressburger²⁴ reported on an extensive study of the cystoid changes in the retina. He stated that they could occur at any age but were most frequent in senile and myopic eyes, and that they were seldom found farther than 7 mm from the ora serrata. According to him, vitreous pulling on such degenerated tissue would cause tears and holes, especially if the cyst were unusually large.

The part played by trauma has been stressed by some authors. Gifford²⁵ expressed the belief that detachment may occur many years after an injury. Others have stated that there are few patients who cannot, if pressed, recall some injury, for example, a blow on the head or eye, rubbing of the eyes, sudden motion of the eyes, such as one makes when an unexpected noise causes a person to look around suddenly, or a fall or a coughing spell. Recently Walker²⁶ pointed out the effect of a sudden movement of the eyes up and to the right on the upper outer quadrant of the left retina, as caused by the combined action of the left internal rectus muscle and the inferior oblique muscle acting against the superior oblique muscle. He expressed the opinion that a sudden jerk of the eyes as in flight would easily whip a retina loose if certain favorable factors were present. Yet if these favorable factors are not present, a detachment does not result, as, for example, in cases of old choroidal tears and in most cases of cataract extraction.

And finally I shall add a few words about the hole in the macula and its relation to detachment. As previously stated, Knapp¹² in 1869 was the first to describe a hole in the macula, but its significance was not appreciated even by Middleton,²⁷ who in 1919 reported detachment of the retina and a macular hole in a soldier who gave a history of trauma. Vogt²⁸ in 1924 reported a case of retinal detachment and uveitis in a man in whom the retinal tear became suddenly larger and

22 Hanssen, R. *Klin Monatsbl f Augenh* **74** 778, 1925, **75** 344, 1925

23 Gonnin, J. *Klin Monatsbl f Augenh* **67** 316, 1921

24 Pressburger, E. *Ztschr f Augenh* **68** 331, 1929

25 Gifford. Personal communication to the author

26 Walker, C. B., in discussion on Spiegel, E. A., and Scala, N. P. *Ocular Disturbances Associated with Experimental Lesions of the Mesencephalic Central Gray Matter*, *Arch Ophth* **18** 614 (Oct) 1937

27 Middleton, A. B. *Am J Ophth* **2** 779, 1919

28 Vogt, A. *Klin Monatsbl f Augenh* **72** 335 1924

was associated with acute hypotony. The following year ²⁹ he established the clinical entity "cystoid macular degeneration," or honeycomb macula. This is similar to Blessig's cystic degeneration of the peripheral portion of the retina. This condition is not easily seen in the macula without the aid of the red-free light. These holes are assuming more importance because of Lindner's recently developed "undermining" method of chemical cautery.

The macular holes may be primary, the result of contusion either directly, such as is caused by a blow from a stone or fist or from a bullet or stick, or indirectly, with an interval during which inflammatory or degenerative processes develop, or they may be secondary to the detachment itself, being caused by toxemia of the interretinal fluids or by wavy motions of the detached retina as the eye moves. Since the macula is the most delicate part of the retina, the retroretinal fluid washing against it would cause a tear in this portion first. Naturally, such a secondary hole must be treated just as any secondary or primary hole must be treated, that is, by the production of seclusive choroiditis.

²⁹ Vogt, A. *Munchen med Wchnschr* **73** 1101, 1925

Correspondence

A NEW METHOD FOR REBUILDING A LOWER LID

To the Editor —An article by Dr Wendell L Hughes entitled "A New Method for Rebuilding a Lower Lid" in the June 1937 issue of the ARCHIVES (page 1008) has just been called to my attention by one of my associates

I am surprised that this article has escaped my notice for so long, but it seems impossible to see all of the contributions on reconstructive surgery appearing in the various journals

Dr Hughes offered this technic as an original contribution and, undoubtedly, has received much credit for an ideal method on this basis It is, however, a faithful description of the method presented before the Société française d'ophtalmologie by Dr L Dupuy-Dutemps, ophthalmologist at Hôpital Saint-Louis, Paris, in May, 1927

The method is described in detail, with full illustration of the various steps of the procedure and a photograph of a completed operation, on pages 161 and 162 of my text "Reconstructive Surgery of the Head and Neck," published by Thomas Nelson & Sons, New York, in 1928

Dr Hughes' only variation of this method is the replacement of the skin resulting from the excision of the lid He elevates the facial skin beneath the eye to cover the new lower lid instead of utilizing normal skin from the upper lid He adds grafted eyelashes after the method described in detail by John Wheeler in a paper entitled "Plastic Operations About the Eye, Suggestions for Several Important Conditions" in the 1922 *Transactions of the International Congress of Ophthalmology* (page 356), but fails to mention Dr Wheeler

FERRIS SMITH, M D, Grand Rapids, Mich

To the Editor —Dr Smith's letter has come at a time when I am in the midst of conducting a more thorough search of the literature When this is completed, it will be offered for publication

There are several methods for reconstruction of the lid in which the structures of the lid are used, one of the earliest being that of Landolt, followed by that of Kollner and of Dupuy-Dutemps The method reported in my paper differs from each one previously described

I wish to thank Dr Smith for calling my attention to Dr Dupuy-Dutemps' procedure, which was not mentioned in the list of references supplied me from the Library of the Surgeon General's Office at Washington, D C

WENDELL L HUGHES, M D, Hempstead, L I

SIMPLIFICATION OF THE O'CONNOR CINCH OPERATION

To the Editor —The article by Dr M E Smukler on simplification (?) of my cinch operation in the June issue of the ARCHIVES (page 930) is most pleasing to me because it indicates that after twenty-seven years interest in it has reached the Atlantic Coast and because in the

discussion Dr L F Appleman emphasized some of its advantages, especially the *absence of reaction*. Most eastern physicians in mentioning the operation emphasize just the opposite idea.

In devising his instrument to facilitate (?) the passing of the deimal shortener, Dr Smukle has fallen into the common error that I always divide the tendon into four strips. In describing my operation I have shown the tendon split into four strands merely for diagrammatic purposes. I often split it into five six or even up to ten or twelve strips, depending on the result desired. Hence a four-pronged separator really does not simplify the operation. Moreover, when Dr Smukle learns the knack of passing the shorteners, I think that he will admit that the time spent in placing his separator is little less than the time spent in passing the shorteners. This knack consists merely in pulling aside the strip that has been looped first, then picking up the exposed second strip and so on across the entire width of the tendon.

As to length of metallic X coating, I like the $\frac{1}{2}$ inch (1.3 cm) length for the same reason that one does not use $1\frac{1}{2}$ inch (3.8 cm) needles for conjunctival or corneal sutures. The shorter coating, when bent $\frac{1}{4}$ inch (0.6 cm) from its end goes between the strips of tendon with less discomfort to the patient.

In general, the fewer special instruments needed to do a particular operation the better. The flat hook is really the only special instrument needed in the cinch operation, and it has come into such universal use in all types of operations on muscles that at the present date it can hardly be called a special instrument.

RODERIC O'CONNOR M D, San Francisco

News and Notes

UNIVERSITY NEWS

Postgraduate Courses, Harvard University Medical School.—Postgraduate courses in ophthalmology at the Harvard University Medical School are divided as follows (1) courses open to general practitioners, dealing with the use of the ophthalmoscope and ocular complications in general disease, (2) a course in the fundamental sciences in ophthalmology, open to beginners in the specialty and devoted to a study of the essentials of embryology, anatomy, pathology, physiologic optics, physiology, chemistry and pharmacology, (3) courses in clinical ophthalmology, ocular pathology and physiologic optics, with a four weeks' intensive course on recent advances in ophthalmology, open to advanced students

Obituaries

JAMES A SPALDING, M D

1846—1938

Dr James A Spalding was born at Portsmouth, N H, Aug 20, 1846, the son of Lyman Dyer and Susan Parker Spalding, and died at his home in Portland, Me, on Feb 27, 1938, in his ninety-second year. He was a grandson of Dr Lyman Spalding, the founder of the Pharmacopeia of the United States, which was responsible for effecting uniform writing of prescriptions. After graduating from Dartmouth College, he received the degree of Doctor of Medicine from Harvard in 1870. He had a defect in hearing, which became gradually worse, and he was advised to specialize in diseases of the eyes and of the ears. After he had studied these specialties abroad, he started to practice in Portland in 1873. He became ophthalmologist and otologist to the Maine General Hospital and took an active part in the development of the practice of these two specialties and helped to found the ophthalmologic and otologic clinics at Augusta, Bangor and Portland.

He was a member of the American Ophthalmological Society, the American Academy of Medicine, the American Academy of Ophthalmology and Otolaryngology, the Maine Medical Association, of which he was once president, and the Cumberland County Medical Association.

Dr Spalding was the author of many medical articles and also wrote several books, including "Maine Physicians in 1820." He married Miss Sarah Chase Shepley, of Boston, in 1882.

In addition to his interest in the two specialties which he practiced, he was a linguist, being unusually proficient in German, Italian and French and also having studied Spanish and Gaelic. He had an analytic mind, and few things escaped his keen sense of humor.

He early came under the influence of Dr Herman Knapp and through many years was of the greatest help in the publication of the ARCHIVES OF OPHTHALMOLOGY and the *Archives of Otology*.

Notwithstanding his loss of hearing, his career has been a most distinguished one. On the occasion of a dinner in his honor, on Sept 19, 1931, Dr Edwin W Gehring stated that Dr Spalding was the "Nestor of the medical profession of Maine, a man of broad culture, profound learning, and wide human sympathies, whose perennial youth startles and delights us."

ARNOLD KNAPP

Abstracts from Current Literature

EDITED BY DR WILLIAM ZENTMAYER

Anatomy and Embryology

HISTOLOGY OF BOWMAN'S MEMBRANE IN CASES OF GLAUCOMA
G TALBOT, Brit J Ophth 22: 210 (April) 1938

In 1937 Wolff and Lyle reported finding hemispherical bodies in Bowman's membrane in an eye excised for chronic glaucoma. In 14 cases, in all of which there were other evidences of glaucoma, Talbot found these bodies in Bowman's membrane. In 4 cases in which there were evidences of glaucoma the bodies were not found, nor were they observed in any case in which tension was presumed to be normal.

The bodies are best brought out by Verhoeff's stain or by phosphotungstic acid and hematoxylin.

Their appearance is constant. They are small structureless hemispheres, measuring about 10 microns, at the base, which is attached to the corneal epithelium. Sometimes one gets the impression that they exceed a hemisphere and that at least two thirds of a globe can be seen.

The presence of such bodies is indicative that the tension is still elevated and is of at least three days' standing.

Wolff suggested that the bodies might be of a hyaline nature in connection with the nerves penetrating Bowman's membrane. The author wonders if they are lacuna-like spaces around the nerve fibers.

One illustration accompanies the article.

W ZENTMAYER

Biochemistry

SEROLOGIC RESEARCH. SPECIFIC PROPERTIES OF THE LENS. E
WOLLMAN, P GONZALES and P DUCREST, Compt rend Soc de
biol 127: 668, 1938

Confirming the work of Uhlenhuth, the authors found that a mammalian lens antiserum though inactive against a lens suspension of the same species precipitates in dilute suspension lenses from all other mammals and birds but affects a suspension of fish lens slightly, if at all. However, if the lens antiserum is derived from fish, the result is positive against all vertebrate lenses, though in varying degree—a dilution of 1:80,000 for the lenses of ray, 1:40,000 for the lenses of codfish and 1:10,000 for the lenses of mammals and birds. On the other hand, the lens of the cephalopod does not react at all with vertebrate lens antisera, or vice versa. The chemical constitution of the lens is hence probably somewhat similar throughout the vertebrate kingdom.

J E LEBENSOHN

Conjunctiva

LYMPHOID REACTION OF THE BULBAR CONJUNCTIVA. REACTION TO
TUBERCULIN. J L PAVIA, Rev oto-neuro-oftal 12: 36 (Feb)
1937

After reference to 4 similar cases previously reported by him, Pavia presents 4 new cases of a peculiar vitreous-like formation in the bulbar

conjunctiva of patients with active tuberculous lesions and a susceptibility to tuberculin injected intradermally

The process is characterized mostly by a slight photophobia and lacrimation, a superficial vascular injection at the site of the lesion, dilatation of the lymphatics accompanying the blood vessels, a slight localized chemosis, semitransparent formations in the vitreous (from 0.3 to 3 mm in diameter), which are easily overlooked, and rarely some circumcorneal injection

Histologic examination in 1 case showed a diffuse lymphoid infiltration, and biopsy of an accompanying follicular infiltration of the lid showed a scattered similar slight follicular formation

In all except 1 case the intradermal injection of tuberculin was followed by a marked local reaction and a prompt disappearance of the ocular lesion. In the case in which there was a negative Mantoux reaction after three trials the ocular lesion promptly disappeared after the injection of tuberculin. In this case there was a preexisting elevation of temperature and a persistent cough

C E FINLAY

Cornea and Sclera

A PRELIMINARY REPORT OF A CASE OF KERATOCONUS, SUCCESSFULLY TREATED WITH ORGANOTHERAPY, RADIUM AND SHORT-WAVE DIATHERMY. H. L. HILGARTNER, H. L. HILGARTNER JR and J. T. GILBERT, *Am J Ophth* 20. 1032, 1937

A case of keratoconus associated with hypothyroidism is reported in an 18 year old girl. Short wave diathermy, the use of drops of ethylmorphine hydrochloride and mercurochrome at home, radium treatment and thyroid therapy resulted in improvement of the vision in the right eye from hand movements at 1 foot (30 cm) on Jan 2, 1937, to 20/100 on February 19. The authors consider the diathermy responsible for the improvement

W S REESE

TREATMENT OF MOOREN'S ULCER WITH LIVER EXTRACT. C. J. CANTILON, *Bull M J* 1 71 (Jan 8) 1938

Two cases of Mooren's ulcer are reported in which an injection of liver extract was given daily and then every other day for three weeks. In addition, Metri's cautery (from 80 to 85 F) was applied to the margins of the ulcer for one minute every second day.

In the second case the treatment was interrupted, and a relapse occurred. Treatment was again begun and continued with fortnightly injections.

ARNOLD KNAPP

SENILE DEGENERATION OF THE SCLERA AT THE INSERTION OF THE OCULAR MUSCLES. REPORT OF A CASE. H. GASTEIGER, *Klin Monatsbl f Augenh* 98 767 (June) 1937

Gasteiger refers to the publications of Pillat, Kiss and Kreibitz on this subject and reports the case of a woman aged 78 with bilateral senile degeneration of the sclera at the insertion of the extrinsic ocular muscles. Dark, glassy looking bands, running fairly perpendicularly,

were observed in the right eye near the insertion of the horizontal and the inferior rectus muscle. These bands had a straight outline toward the cornea, but they were slightly humpy toward the temples. Slit lamp examination showed the unevenness to be produced by tendons of the muscles. A similar condition existed in the left eye, but for the absence of the band near the inferior rectus muscle. Scleral transillumination showed a weak red reflex over the temporal band of each eye, whereas the other bands remained dark. This picture differs from that reported by Pillat, who observed illumination of all bands, the appearance being that of bright red windows. Experiments on pigs' eyes dehydrated with glycerin showed that this change was gradual but not essential. Gasteiger concludes that conditions of dehydration are responsible for these phenomena in human eyes also.

K. L. STOLL

Experimental Pathology

EXPERIMENTAL DINITROPHENOL CATARACT IN ANIMALS. H. SOHR, *Arch f Ophth* 138: 332 (Feb) 1938

The chief American literature on dinitrophenol cataract is reviewed. The author fed dinitrophenol to guinea pigs for two and a half months, giving from 30 to 40 mg per kilogram of body weight by stomach tube two or three times a week. Of the 25 guinea pigs treated in this way 17 died during the feeding period. The remaining 8 animals were observed for four months, during which time 5 more died. The remaining 3 animals retained macroscopically and microscopically normal lenses.

P. C. KRONFELD

THE PERMEABILITY OF THE CAPSULE OF THE LENS AND THE DEXTROSE CONTENT OF BLOOD, AQUEOUS AND LENS IN ANIMALS WITH GALACTOSE CATARACT. T. SASAKI, *Arch f Ophth* 138: 365 (Feb) 1938

Certain phases of the carbohydrate metabolism of rats fed galactose and suffering from galactose cataract were studied. The reducing sugars of the whole blood, of the aqueous and of the lens were determined by the method of Hagedorn and Jensen, and the results were expressed in terms of dextrose. The cataractous lenses were incubated in saline solution for five hours, and the amounts of dextrose which were used up by the lenses during this period (glycolysis) and the amounts which diffused out of the lenses into the saline solution were determined. The dextrose content of the whole blood and that of the aqueous were found to be considerably higher for the rats fed galactose than for the control animals. The cataractous lenses, however, contained about the same amount of simple sugars as the lenses of the control animals. From these data the author concludes that in the animals with galactose cataract the osmotic pressure of the aqueous was higher than that of the lens, which difference might play a part in the production of the cataract. Incubated in saline solution, the cataractous lenses gave off more sugar than normal lenses, a phenomenon which indicates increased permeability of the capsule of a cataractous lens. If dextrose was fed young rats instead of the galactose of the cataract-

producing diet, no cataract developed, the dextrose contents of blood, aqueous and lens were found to be increased. No difference in osmotic pressure prevailed between aqueous and lens, and the permeability of the capsule of the lens remained normal, as far as its condition could be judged from the loss of sugar during incubation.

P. C. KRONFELD

General

THE SIGNIFICANCE OF HEREDITY IN OPHTHALMOLOGY. PRELIMINARY SURVEY OF HEREDITARY EYE DISEASES IN TASMANIA. J. B. HAMILTON, *Brit J Ophth* 22: 19 (Jan), 83 (Feb), 129 (March) 1938.

After a brief historical sketch and some vital statistics on Tasmania, Hamilton tabulates the associated hereditary defects of the eye with hereditary defects in other parts of the body and also possible hereditary ocular defects.

These articles constitute a treatise on the significance of heredity in ophthalmology and do not lend themselves to abstracting.

W. ZENTMAYER

USE OF CONTACT GLASSES FOR COSMETIC PURPOSE. S. K. MUKERJEE, *Brit J Ophth* 22: 43 (Jan) 1938.

Unsuccessful attempts had been made to darken the cornea of a girl aged 16 in order to conceal leukomatous changes. When she was seen by the author the cornea was slightly staphylomatous, and the iris tissue was entangled in the wound. An excellent cosmetic effect was obtained with the use of a contact glass, which was painted on the inside to match the color of the other eye.

The article is illustrated.

W. ZENTMAYER

INFLUENCE OF LIGHT ON ARTERIAL TENSION. H. VIGNES, *Compt rend Soc de biol* 127: 768, 1938.

Some obstetricians have been clinically impressed with the adverse influence of light in cases of eclampsia and have insisted on keeping their patients in a darkened room. Experimentally, sudden passage from darkness to a well lighted room (300 watts at 2 meters) induced an increase in the blood pressure of from 5 to 15 mm, especially in pregnant women, among whom those menaced by eclampsia were particularly sensitive.

J. E. LEBENSOHN

General Diseases

DYSOSTOSIS MULTIPLEX WITH SPECIAL CONSIDERATION OF THE OCULAR CONDITION. REPORT OF A CASE. H. GASTEIGER and L. LIEBENAM, *Klin Monatsbl f Augenh* 99: 433 (Oct) 1937.

Dysostosis multiplex, a rare osteodysplasia, is a degenerative dysostosis of a hyperplastic type. One of 2 female twins aged 13 was normal, the family history was unimportant. The other twin was born in breech presentation, but developed normally up to the age of 2½ years, when

her size remained behind that of her sister. The growth in height stopped entirely when she was 7 years old. She had a massive head and was disproportionably dwarfed, a deformed thorax, a gibbus in the upper lumbar portion of the spine, a protruding abdomen, deformed extremities and a clumsy gait were noted. The function of the joints was limited, and the mouth could not be fully opened. The inner organs were normal. A detailed description of the roentgenograms is given. No changes of the skull were found aside from bony connections between the anterior and the middle clinoid process and between both anterior and posterior clinoid processes. There was an absence of ocular changes except for corneal nubeculae. Under the slit lamp these nubeculae appeared to be composed of numerous small, dot-shaped grayish-white opacities, they were evenly distributed in all layers of the cornea, but least so in Bowman's membrane. The transparency and sensitiveness of the cornea and the field of vision were normal, vision was 5/5 after correction of hyperopia of 5 and 6 D, respectively. No further pathologic changes were observed later. Pain in the back and dyspnea disappeared, while the function of the extremities increased under systematic active and passive exercises. Ample space is devoted to the differential diagnosis, and the etiologic factors are discussed. The authors conclude that dysostosis multiplex is a congenital disturbance which leads to disproportionate dwarfness. The causal genesis of the disease is unknown.

K L STOLL

Glaucoma

THE ASSOCIATION OF CYCLODIALYSIS WITH FISTULIZING OPERATIONS IN CERTAIN CASES OF GLAUCOMA. M G BESSO, *Ann di ottal e clin ocul* 65: 625 (Aug) 1937

On a number of eyes with secondary, subacute or chronic absolute glaucoma in which vision had been reduced to zero or nearly zero the author performed corneoscleral trephining according to the method of Elliot. However, on completion of the sclerotomy, he inserted the Elschnig spatula in the trephine opening and separated the ciliary body from the sclera in an area corresponding to that covered by the usual cyclodialysis. This was followed by basal iridectomy, after which the flap was sutured. The tension in eyes with the subacute and chronic forms of glaucoma was usually maintained below normal by this procedure, while in those with glaucoma secondary to recent uveitis, exacerbations of inflammation occurred, which prevented the desired result. Reasons are given which may lead to the adoption of the procedure for other types of glaucoma. Patients on whom this operation was performed have been followed for from six to twelve months.

S R GIFFORD

Injuries

SUPERFICIAL AND DEEP-SEATED DISCIFORM KERATITIS IN CAISSON WORKERS AFTER EXPOSURE TO HYDROGEN SULFIDE. REPORT OF CASES. K HARTMANN, *Klin Monatsbl f Augenh* 99: 456 (Oct) 1937

In the first chapter of this publication Hartmann refers to ocular lesions produced by hydrogen sulfide as a disease observed more frequently in recent years. It occurs in workers in factories producing

artificial silk, sugar, soap and glue and also in those employed in tanneries, dye works, mines, and sulfur baths. The injuries produced by this chemical have been recognized as an occupational disease through the effort of Thies. Previous publications in point are recorded.

Six cases of this condition in caisson workers are reported. The location in which the lesions occurred is described at the beginning of the second chapter and a detailed clinical history of each case follows. The men were working at a depth of from 8 to 13 meters below the surface of the North Sea, the ocular disease occurred mostly when they were working in a layer composed of loam and shells, in which sulfurous gases abounded. Recovery followed when local treatment was administered and the workers were placed in well aired surroundings, the ocular symptoms recurred as soon as the men returned to the lower strata. The symptoms consisted chiefly of punctate defects and vesicular eruptions in the cornea, localized within the area of the palpebral fissure. The sensitiveness of the cornea was reduced. No complications of the intraocular organs developed. A resistance to the fumes of hydrogen sulfide could not be acquired.

The prognosis and course were favorable in all but 1 case, in which a deep-seated disciform keratitis left a dense scar. Haitmann discusses the possibility of an infection with the herpes virus in this case, because the lesions produced by hydrogen sulfite are usually limited to the corneal epithelium.

Suggestions are given for the prevention of this type of keratitis. Among them are: elimination of those workers who have previously suffered from diseases of the respiratory tract and corneoconjunctival diseases, proper ventilation and fresh air containing not more than 0.05 per cent of sulfurous gases, air-tight goggles, and shifts of not more than three or four hours.

K. L. STOLL

Lens

SPHEROPHAKIA, LUXATION OF LENSES, AND SECONDARY GLAUCOMA
RELIEVED BY EXTRACTION OF LENSES. I. JACOBS, *Am J Ophth*
20: 1042, 1937

Jacobs discusses spherophakia and dislocation of the lens and the aggravation of glaucoma in such cases by the use of miotics. He cites the case of a 17 year old boy who had bilateral dislocation of the lens. Both lenses eventually became dislocated into the anterior chamber, and secondary glaucoma ensued, which was relieved by the administration of a few drops of a 1 per cent solution of atropine. Both lenses were successfully removed.

W. S. REESE

PSYCHOSES COMPLICATING RECOVERY FROM EXTRACTION OF CATARACT
P. PREU and F. GUIDA, *Arch Neurol & Psychiat* 38: 818 (Oct)
1937

Four cases of psychosis following cataract extraction are reported. In 3 cases a diagnosis of psychogenic "experiential" panic was made. This condition is characterized by restlessness, apprehension, fear, "experiential" confusion and in some cases paranoid misinterpretation of environment. In the fourth case a diagnosis of senile arteriosclerotic

mental disorder was made. This condition is characterized by sensorial and intellectual defects, with fluctuating disorientation and impairment of memory but no striking emotional disorder.

In the first type of psychosis the operation, the subsequent care and especially the unfamiliar setting of darkness are all so out of the realm of the patient's past experience as to produce an "experiential" confused state. This usually comes on within the first twenty-four hours after operation and is relieved within forty-eight hours after the removal of bandages.

In the second, or organic, type of psychosis, the operation plays no etiologic role, and removal of the bandage has no effect.

The authors emphasize the importance of recognizing the second type preoperatively by some inquiry into the intellectual status of the patient, for example, by memory tests. Administration of sedatives usually increases the already present clouding of consciousness and so are contraindicated. Frequently a combination of both types of psychosis is encountered. The authors suggest familiarizing the patient with the procedure and surroundings preoperatively, and in some cases in which mental difficulty is definitely anticipated, they even suggest operating in the home if possible.

R IRVINE

CATARACT AND LATENT TETANY M. MECCA, *Ann di ottal e clin ocul* 65: 609 (Aug.) 1937

A woman of 25 had noticed failing vision for two years. Falling of the hair and fragility of the nails had also been observed. Vision was reduced to 3/50 in the right eye and 1/10 in the left eye. Both lenses showed numerous white punctate, needle-shaped opacities and also a ring of larger round or oval opacities. The subcapsular layers of cortex and the nucleus were involved anteriorly and posteriorly, leaving a clear zone near the equator of each lens. Neurologic examination showed signs of latent tetany. The blood calcium was 9 mg per hundred cubic centimeters and the potassium was 19 mg. After discussion and corneal expression, the course was practically normal, with recovery of vision to 7/10. The literature on so-called endocrine cataract is reviewed, which indicates that bilateral progressive subcapsular cataracts of this type are most commonly due to a glandular imbalance involving especially the parathyroid glands.

S. R. GIFFORD

CONCERNING CATARACT FORMATION AFTER REDUCTION OF WEIGHT WITH DINITRO-BODIES T. HELMINEN, *Acta ophth* 15: 490, 1937

The 5 cases of cataract reported here occurred in Finland. All the patients were otherwise healthy women below the usual age for cataract. Two had used dinitrophenol, 2 had used dinitroorthocresol, and 1 had used both drugs. The usual therapeutic dose was never exceeded, and the formation of cataract did not seem dependent on the amount of the drug taken. The interval between the completion of the reducing treatment and the full development of the cataracts varied from seven to fourteen months. Prodromal disturbances of accommodation occurred in 3 cases. The cataracts developed over a period of from three to seven months. In 4 cases secondary glaucoma occurred shortly before the extraction of the lenses.

There were no operative or postoperative complications. The author tried unsuccessfully to produce changes in the lens by feeding the drugs to rabbits.

O P PERKINS

Lids

LICHEN PLANUS OF THE EYELIDS H E MICHELSON and C W LAYMON, *Arch Dermat & Syph* 37:27 (Jan) 1938

Some areas of the body seem immune to the invasion of certain diseases. This appears to be true of the eyelids in lichen planus. However, Michelson and Laymon saw 5 patients in whom the eyelids were unmistakably the seat of such lesions. Three types of lesions were observed: (1) the classic lilac-colored slightly delled papules with filigree scaling, associated with similar lesions on the body; (2) papules so arranged as to make the typical annular or small medallion-like plaques which are often seen on the glans penis (patients with this type had other annular lesions on the body) and (3) some unique lesions. The third type was noted in 2 brunette women, aged 29 and 32. The upper eyelids were discolored with a sepia retiform eruption, which was not made up of elevated papules, although in the skein of the network were small knobs which could have been papules previously. The eruption caused no symptoms but gradually extended to all four lids and became darker, constituting a cosmetic deformity. In 1 of these cases a small bit of tissue was excised, and on microscopic examination it showed the characteristic bandlike infiltration of lichen planus. The rest of the skin and the mucous membranes were free from eruption in both cases. The eruption on the eyelids was identical with areas of melanotic staining seen in certain patients with lichen planus in the late stages of regression. This form of lichen planus of the eyelids could easily be ignored as a variant of that disease or might be diagnosed as erythema ab igne. Conjunctival lesions are identical with those seen on the buccal or on the genital mucosa.

J A M A (W ZENTMAYER)

PALPEBRAL MANIFESTATIONS IN DELAYED HEREDITARY SYPHILIS R GOZBERK, *Ann d'ocul* 174:837 (Dec) 1937

Delayed hereditary syphilis often gives rise to such ocular lesions as keratitis, iritis, iridochoroiditis and neuritis, but its localization at the site of the lids is exceptional. Laurence reported a case in which signs of hereditary syphilis were present on the lids, lips and cheeks of a boy of 14 years. In 1888 Hutchinson described palpebral commissures, and von Michel in 1900 reported a gumma on the upper lid of a girl of 11 years. Other similar reports are recorded.

Delayed manifestations appear during childhood, adolescence and adult life. As a rule they develop spontaneously, but they sometimes appear during convalescence from infectious diseases or after trauma. Except in rare cases, these late appearances coexist with congenital dystrophies, which are of aid in making a diagnosis. Delayed hereditary syphilis may attack any part of the lid, skin, conjunctiva, subcutaneous tissue and tarsus. It appears in one of three forms: cutaneous gumma, subcutaneous gumma and gummatous infiltrations bordering on ulcer-

ation The ulcerations may be so extensive as to bring about destruction of the lid

Two cases are reported The first was that of a boy of 11 years in whom a subcutaneous gumma developed in the parts surrounding the eye after an injury In a few weeks an ulcerative process developed, causing irreparable mutilation of the lid In the second case that of a man of 30, syphilitic gummas appeared on the lid and on the face In this patient, who was insufficiently treated, an injury revealed the latent syphilis Two illustrations and a bibliography accompany the article

S H McKEE

Methods of Examination

OPHTHALMODYNAMOMETRY AND ITS IMPORTANCE IN CLINICAL MEDICINE T R YANES, *Rev cubana de oto-neuro-oftal* 5 5 (Jan-Feb) 1936

After a historical survey and a description of the technic of using the ophthalmodynamometer, the author stresses the importance of dividing patients with a high retinal arterial pressure into two groups, those with a coincident elevation of the general arterial pressure and those in whom the pressure is normal, and of studying in the latter group conditions of meningocephalic, ocular and vascular origin

C E FINLAY

MEASUREMENTS IN THE FUNDUS W KUHN, *Arch f Ophth* 138:129 (Oct) 1937

With a heliometer built into the ophthalmoscope of Gullstrand (*Arch f Ophth* 133:153, 1934), Kuhn measured the width of the retinal vessels of 225 healthy persons and of a number of persons with vascular diseases and diseases of the optic nerve In every case the micrometer scale of the heliometer was first standardized by measuring with it a fairly constant distance in the fundus, namely, the horizontal diameter of the disk, then the width of the vessels near the margin of the disk was measured in the thus standardized scale units For healthy persons, the relation to the caliber of the artery to the caliber of the vein and to the horizontal diameter of the disk was 1 12 14 to 16 (1 expressing the value for the arterial diameter) In absolute terms, the caliber of the artery varied from 0.086 to 0.138 mm and that of the vein from 0.100 to 0.159 mm In a case of occlusion of the central artery, the width of the latter was 0.03 mm, whereas the vein had a caliber of 0.125 mm

P C KRONFELD

Neurology

A CASE OF EPILEPSY ASSOCIATED WITH MENINGIOMA OF THE OPTIC NERVE SHEATH COMPRESSING THE OLFACTORY CENTERS, DURAL CALCIFICATIONS AND THALAMIC LESIONS J W PAPEZ and R W RUNDLES, *Arch Neurol & Psychiat* 39 150 (Jan) 1938

The authors present a case of epilepsy with an olfactory aura in a physician who died at the age of 76 and summarize it as follows

"Twenty years prior to death there developed retrobulbar optic neuritis on the right, which resulted in blindness. An olfactory aura appeared, and five years later epileptic attacks began. A meningioma of the right optic nerve sheath which compressed the basal olfactory centers was observed at autopsy. A tumor of the breast had been removed surgically. The adrenal glands were greatly enlarged. Degenerations of various fiber tracts caused by the meningioma, dural calcifications and lacunar softenings in the left thalamus and elsewhere are described."

R. IRVINE

CONTRIBUTION TO STUDY OF BLINDNESS OF HYPOPHYSIAL ORIGIN
FOUR CASES OF ADIPOSOGENITAL SYNDROME WITH RETINAL
DEGENERATION AND MENTAL BACKWARDNESS. P. PESME and G.
HIRTZ. *Gaz. med. de France* 44: 833 (Oct 15) 1937.

Pesme and Hirtz recently observed 4 cases of a curious hereditary and familial syndrome that is characterized by adiposogenital dystrophy with mental backwardness and retinal degeneration. The authors describe these cases not only because of their rarity but because they show the role of the hypophysis in the development of the syndrome and particularly the ocular lesions. The authors direct attention to the similarity of the disorder to the Laurence-Biedl syndrome. The described cases differ from those designated as the Laurence-Biedl syndrome by the absence of malformations of the members (syndactylism or polydactylism). There seem to be three symptoms that characterize the typical as well as the atypical cases: obesity of the hypophysial type, mental backwardness and retinal disorders. The retinal symptoms are especially noteworthy. In all cases there was a degeneration of the retina, with discoloration and night blindness. One of the patients was given extracts of the entire hypophysis for a year, but this form of treatment failed to produce the slightest improvement. After a new examination, it was decided to treat the patient with extracts of the anterior lobe of the hypophysis and with several other glandular preparations (thymus, thyroid, adrenal and whole pituitary). Under the influence of this treatment the patient lost weight and increased in height. Moreover, the genitalia developed to normal size, the hands and fingers which had been extremely short, assumed a normal shape, and the nails lost their friability. The mental aspects likewise changed in that the boy became more active and bright. Even more remarkable was the change in the ocular defects. The boy was able to fix his vision without being disturbed by nystagmus, and the photophobia disappeared. Measurement of the visual acuity revealed hardly any increase, but the boy was better able to utilize his visual powers. On the basis of this observation the authors conclude that the hypophysis influences not only the fat metabolism and the development of the genital organs but the retina. Such a relation between the retina and the hypophysis had already been suggested by other investigators, and the authors think that treatment with hypophysial hormones may perhaps prove valuable in some congenital retinal defects.

J. A. M. A. (W. ZENTMAYER)

THE CHANGES IN THE EYE IN HERPES ZOSTER OPHTHALMICUS A N
MURZIN, *Vestnik oftal* 11.758, 1937

Three cases of herpes zoster ophthalmicus with serious complications are reported

CASE 1—A woman aged 30 had ocular changes consisting of diffuse corneal opacities in the superficial and deep layers, numerous retinal hemorrhages and marked inflammation of the optic disk, which gradually became atrophic, with resulting loss of vision. There was also paralysis of the abducens nerve on the side affected. Murzin believes that the herpes virus most likely penetrated the optic nerve and the retina through the sheaths of the ciliary nerves after having caused changes in the trigeminal nerve.

CASE 2—A man aged 23 had herpes zoster of the region of the second and the third branch of the trigeminal nerve, keratoiritis and optic neuritis. Vision improved from 0.08 to 0.6 within one month.

CASE 3—A man aged 63 had herpes zoster of the first branch of the trigeminal nerve with involvement of the sympathetic nerve, as Horner's syndrome was present. Murzin discusses the causation and the clinical and pathologic pictures of herpes zoster.

O SITCHEVSKA

Ocular Muscles

FUNDAMENTAL PLAN UNDERLYING OCULAR MOVEMENTS I THE
CONJUGATE HORIZONTAL MOVEMENTS J OHM, *Arch f Ophth*
138.1 (Oct) 1937

Summing up thirty years of his widely known work, Ohm gives a comprehensive presentation of his views concerning the innervation of ocular movements. At first the physical and the nervous mechanism of vestibular nystagmus are described. On slow rotation of a normal person in the turning chair in a dark room the eyes show no nystagmus but a tonic deviation in the same direction as the current of the endolymph which is caused by this rotation. On more rapid rotation, nystagmus with a slow and a quick phase occurs. The tonic deviation is independent of either phase of this nystagmus. Stimulation of the neuroepithelium of a single semicircular canal is sufficient to cause nystagmus. Nystagmus to the right, for instance, may be caused by an ampullopetal lymph current in the right horizontal semicircular canal or by an ampullofugal current in the left horizontal semicircular canal, by irrigation of the right external auditory meatus with hot water or of the left external auditory meatus with cold water and by destruction of the left labyrinth or of the left vestibular nerve. Ohm considers nystagmus with slow and quick movements as a process of discharge of energy by the vestibular nuclei which is not conditioned on the integrity of the vestibular nerves. In the state of rest that is in the state in which no strong stimuli are acting on the vestibular nuclei, there is a mutual exchange of energy between the two nuclei through intranuclear fibers until an equilibrium is reached. An ampullopetal lymph current increases the charge of the homolateral nucleus, whereas an ampullofugal current lowers the charge.

One of the cardinal points in Ohm's views is the assumption of a main stimuli-emitting station (*Hauptaugenmuskelsender*) for the ocular muscles which is located in the nucleus of the vestibular nerve. Even the impulses for voluntary ocular movements which are produced in the frontal center of gaze pass through the vestibular nuclei.

The pathways for optokinetic nystagmus and its disturbances are reviewed in great detail. The fiber tracts of the central nervous system which are concerned with ocular movements are graphically represented in three diagrams.

P. C. KRONFELD

ESOPHORIA AND ANAPHORIA IN TWINS OF THE SAME SEX J. STREBEL,
Klin Monatsbl f Augenh 98: 788 (June) 1937

Twins sisters aged 18, of monolike resemblance, had eyes of identical appearance and muscular asthenopia. They complained of pain in the forehead, which disappeared when the hyperphoric right eye was covered for work at close range. Vertigo was absent. The eyeballs were free from pathologic changes. Slight esophoria, positive hyperphoria, anaphoria of 4 prism diopters in the right eye and of $\frac{1}{3}$ in the left eye and homonymous double images of about from 10 to $\frac{1}{12}$ prism diopters were found. As prism lenses had afforded only transient relief recurrences of the upper half of the medial rectus muscle, and of the nasal half of the superior rectus muscle were done under phorometric control. Complete disappearance of the disturbance resulted.

K. L. STOLL

Orbit, Eyeball and Accessory Sinuses

DISLOCATION OF THE EYEBALL AS A COMPLICATION OF OXYCEPHALY
J. SHERNE, Brit M J 1: 565 (March 12) 1938

A child 8 months old was first seen at the General Infirmary in Leeds on July 12, 1935, at which time there was marked protrusion of the forehead and exophthalmos was present. The roentgenographic appearances were typical of oxycephaly. The child was seen again at the age of 2 years. Vision was supposed to be defective. On April 8, 1937, the left eye became suddenly completely dislocated forward and was immobile. After anesthetization, the eye was gently levered back into position with a spatula, and to prevent a recurrence, a lateral tarsorrhaphy was done. Examination of the right fundus showed slight pallor of the disk. The child recovered promptly, and there has been no return of the dislocation.

In attempting to explain this condition, the author assumed that the proptosis was so marked that the action of the orbicularis muscle was largely nullified or reversed. In a fit of crying, the spasm of the orbicularis muscle was so great as to dislocate the globe.

ARNOLD KNAPP

AN INTERESTING CASE OF THROMBOPHLEBITIS OF THE CAVERNOUS
SINUS A. FRANCES, Rev oto-neuro-oftal 12: 79 (March) 1937

A case of thrombophlebitis of the cavernous sinus of dental origin with a fatal outcome is reported. The author stresses the possibility of this origin, as it is usually overlooked.

C. E. FINLAY

CLINICAL VALUE OF ROENTGENOGRAPHIC STUDIES OF THE OPTIC FORAMEN B J FARBEROV, *Ztschr f Augenh* 89:208 (June) 1936

Roentgenographic studies were made of the optic foramina of 300 persons. The technic was not described in detail, but to be of any value identical procedures must be carried out for both the right and the left foramen. Pathologic conditions of positive clinical value were found in only 22 per cent of the cases. Normal roentgenograms were of definite negative clinical value in 6 per cent.

Of the pathologic conditions, enlargement of the canal was found in 17 cases. In 4 of these there was no general pathologic process to account for the enlargement. Unilateral unexplained papilledema was associated with enlargement in 1 case, which three years later showed no change. The enlargement in the other cases was caused by various types of tumors of the optic nerve and orbit.

Narrowing of the canal was found in 9 cases, caused by trauma, tumors, congenital atrophy of the optic nerve, Crouzon's disease and syphilis.

Deformities of the canal were noted in cases of congenital anomalies of the skull. General thickening of the wall of the canal was caused by osteoblastic metastatic tumor, congenital atrophy of the optic nerve, syphilis and hemangioma of the orbit. Thinning of the wall of the canal was caused by tumors of the optic nerve and fibromatosis of Recklinghausen. Farberov feels that roentgenographic studies should be obligatory in cases in which there are atrophy of the optic nerve, exophthalmos, retrobulbar neuritis, unilateral papilledema, trauma to the skull, and unusual atypical pathologic changes in the visual field or fundus.

H GIFFORD JR

Physiology

THE NATURE OF THE AQUEOUS HUMOR J D ROBERTSON, *Brit J Ophth* 22:79 (Feb) 1938

Robertson considers the several points raised by Duke-Elder in an article criticizing his paper concerning the theories on the formation and exit of the aqueous humor. He comes to the following conclusions.

The canal of Schlemm is not a vascular capillary in any sense, physiologic or anatomic.

Further study of the urea and the sugar equilibrium shows that the aqueous humor cannot be formed by dialysis because (a) the concentration of urea in the aqueous is lower than that in the blood and (b) the concentration of sugar in the aqueous is also lower.

As secretions and dialysates are both isotonic with blood, observations on the osmotic pressure are of no value in differentiating the one from the other.

A physical equilibrium does not exist between blood and aqueous, and the equilibrium level of the intraocular pressure is not maintained by the hydrostatic force in the capillaries minus the difference in osmotic pressure between blood and aqueous.

This is a further argument against the theory of dialysis. In summary, it may be stated that the observations which the author reported

originally refute the suggestion that the aqueous humor is a dialysate, and Duke-Elder's most recent contribution does not cause him to alter this view

W ZENTMAYER

Retina and Optic Nerve

RETINAL MYELIN FIBERS A GARCIA MIRANDA, *Ann d'ocul* 174 744 (Nov) 1937

Retinal conditions in man and in the higher animals present a great problem which experimental biologic research is endeavoring to solve. A study of embryologic and hereditary factors has helped to clear in an important manner this not thoroughly explored field. Among unexplained retinal conditions, one of the most interesting is that found represented by retinal myelin fibers.

The fibers were discovered by Vuchow, who in 1856 first observed white patches in the neighborhood of the papilla. The year before Jaeger observed myelin fibers in the retina on ophthalmoscopic examination and had considered them to be an enlargement of the papilla. Nowadays numerous authors stress the familial origin of this condition. Mauthner observed it in 2 sisters, and Kiso observed it 6 times in a family during two generations. Schueck concludes that it is a hereditary condition.

The ophthalmoscopic appearances are well known. In typical cases brilliant white patches are seen branching laterally above the papilla. At the periphery these have the appearance of silk thread, near the papilla they are more opaque and more prominent. The vessels appear in some parts to be covered by these, in others, they lie in the myelin fibers. The fibers rarely pass the borders of the papilla, but this varies considerably. Usually the fibers come just to the border without going beyond, appearing slightly opaque. Sometimes the papilla is affected in only a small degree, while at times it is completely involved, with the retinal vessels visible only in the retina. One illustration and a bibliography accompany the article.

S H McKEE

PSEUDO ALBUMINURIC RETINITIS IN CASES OF TUMOR OF THE BRAIN
I J MERKULOW, *Acta ophth* 15 406, 1937

Merkulow records his observations on 8 patients, all of whom on ophthalmoscopic examination showed the type of lesion in the fundus which is ordinarily associated with renal disease. One patient proved to have an abscess in the temporal lobe. The other patients all had tumors of the brain. Examination of the urine and blood and a test of the renal function eliminated involvement of the kidney in each case. In no case was the blood pressure elevated.

The author theorizes as to the mechanism of the production of pseudo albuminuric retinitis. He reviews briefly the classic theories as to the production of real albuminuric retinitis, paying most attention to the opinion of Volhard, who holds that the retinitis is not the result of hypertension as such but of the ischemia resulting from vascular changes. In the cases under consideration the author believes that the cycle of

events is as follows. Even in the absence of high systemic blood pressure there may be intracranial hypertension. This gives rise to a choked disk. The choked disk causes a disturbed retinal circulation, with eventual changes in the vascular structure and permeability. Deposition of fatty elements and the other changes of pseudo albuminuric retinitis result, but only if an additional factor is present. The additional factor is held to be a toxic product in some way elaborated by the tumor of the brain.

O P PERKINS

Uvea

PATHOGENESIS, ETIOLOGY AND TREATMENT OF GLAUCOMATOUS IRIDOCYCLITIS. L WEINGOTT, *Arch d'opht* 53: 672 (Sept) 1936

In this paper the author offers an explanation for the occurrence of persistent hypertension in some cases of iritis. He reviews the work which has been done and the theories proposed to account for the secondary glaucoma which sometimes persists in spite of treatment. As a result of his observations, the author suggests that persistent hypertension in cases of iridocyclitis is due to degenerative changes which are commonly the result of syphilis and alcohol. He believes that alcohol increases microbic virulence and causes chronic anatomic changes. Inasmuch as gonococcal infection has been found in so many instances, the treatment he advocates includes the administration of vaccine in addition to the usual therapeutic procedures.

S B MARLOW

Society Transactions

EDITED BY W L BENEDICT

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

CHARLES R HEED, M D , *Chairman*

Dec 16, 1937

ALEXANDER G FEWELL, M D , *Clerk*

INTRACAPSULAR CATARACT EXTRACTION REPORT OF A FURTHER SERIES OF CASES DR LEIGHTON F APPLEMAN

This article was published in full in the April issue of the ARCHIVES,
page 548

BILATERAL SUPERFICIAL LESIONS OF THE CORNEA DUE TO ALLERGY REPORT OF A CASE DR GEORGE J DUBLIN

A white woman aged 48 complained of pain and inflammation of the eyes for one week. She had suffered a similar attack four years previously.

Vision in each eye equaled 6/6. Both eyes showed moderate bulbar injection, the corneal epithelium contained multiple punctate infiltrates, which were thickened and slightly elevated and stained with fluorescein. The eyegrounds were normal. The patient complained of severe discomfort to light.

Medical examination disclosed slight involvement of one of the sinuses and secondary anemia. The condition in the sinus cleared, but the pain and discomfort continued in spite of usual treatment. Tests for allergy were made, and the patient had a strong 4+ reaction to orris root. All other tests gave negative results.

The patient was advised to discontinue the use of cosmetics containing orris root. Without treatment, relief was instantaneous, and within twenty-four hours there were only 5 stained areas in the right cornea and 20 in the left cornea, whereas there had always been approximately from 100 to 200 such areas in each cornea for the past four months. The patient was seen frequently during the following three months, and in this time there was no recurrence of symptoms.

DISCUSSION

DR WILLIAM ZENTMAYER. At the time I saw this patient the conjunctiva of the upper lids was quite papillary, and there was vascularity of the limbus together with the other changes in the corneas described by Dr Dublin.

The condition somewhat resembled trachoma, and I thought that this possibility should be borne in mind, although there was also a

likelihood that it could be due to cosmetics I think that Dr Dublin has conclusively shown that the latter was correct

DR LEIGHTON F APPLEMAN I believe observations of this kind are of considerable importance Occasionally one sees in hospital practice patients with inflammation involving the lids and conjunctiva which resist all usual forms of medication I have at times been at a loss to know what medication to use next I believe that many of these inflammations may be of allergic origin and that the patient should be subjected to the usual tests to determine this

ALEXANDER G FEWELL, M D, *Chairman*

April 21, 1938

WARREN S REESE, M D, *Clerk*

MUSCLE SPASM CAUSED BY EYEGLASSES DR LEO F McANDREWS

A man 41 years of age presents this interesting finding As soon as he attempts to wear his glasses, a distinct, hard mass appears over the region of each mastoid

Palpation indicates that the mass is evidently a contraction of the occipitofrontalis muscle

If the patient persists in wearing the glasses, the entire right side of the head and right ear become painful, and the neck becomes stiff Removal of the glasses or firm pressure on the bridge of the nose causes the mass and discomfort to disappear

The condition has been present for about four years Thyroidectomy was performed three months ago, but the patient is still nervous and emotionally unstable

There is no anatomic explanation for this condition The probable explanation is that it is a neurotic manifestation associated with the toxic goiter and can be cured by appropriate psychotherapy

DISCUSSION

DR WALTER I LILLIE It is my impression that this is a spastic condition Its duration of four years is suggestive of a postencephalitic syndrome When the man puts his glasses on he has marked twitching of the eyelids In a case of postencephalitic syndrome, especially if changes in the respiratory tract are present, any sudden touch to any part of the body stops the attack It would be interesting to follow this patient to see if a parkinsonian syndrome develops

PALSY OF THE OCULAR MUSCLES IN A CASE OF TOXIC GOITER DR C E G SHANNON

This is a supplementary report on a case of toxic goiter complicated by palsy of the ocular muscles A preliminary report was made at the meeting of the section in April 1937

H. D., a man aged 47, was admitted to the Jefferson Hospital on April 2, 1936, with a diagnosis of exophthalmic goiter. His chief complaints were tremor of the hands, palpitation of the heart, bulging of the eyes, diplopia, excessive perspiration and loss of weight. The blood count was normal, and the Wassermann and Kahn reactions were negative. The basal metabolic rate was 45 plus. Motility of the larynx was normal, with no apparent narrowing of the tracheal airway.

Subtotal thyroidectomy was performed, and the patient was discharged ten days after the operation in good condition and relieved of all symptoms of toxic goiter aside from the proptosis and the diplopia.

Briefly, the ocular history of the patient is as follows. In March 1936, about a month prior to the removal of the thyroid gland, diplopia first developed, it was followed shortly by swollen and edematous lids and later by exophthalmos.

The diplopia and proptosis persisted, but the edema of the lids receded immediately after operation.

At the first examination of the patient, on Dec. 4, 1936, the following notes were made:

Vision in the right eye equaled 6/9 +, that in the left eye equaled 6/9 (mostly). The pupils were equal and reacted freely to direct and consensual stimulation. The tension was normal in each eye. The media and fundi were normal. The eyes were proptosed, the exophthalmometer showing 23 mm of proptosis on each side. The left eye appeared definitely deviated inward and on a lower plane than the right eye. The Maddox rod test showed an esophoria of 30 degrees and left hypophoria of 26 degrees.

Diplopia was obtained in all the cardinal directions except in the immediate lower field, i. e., about from 12 to 14 inches (30 to 35 cm) from the eyes, indicating involvement of all the extrinsic muscles of the eyeballs. It was interesting to note that a wide separation of images at 20 feet (609 cm) diminished steadily as the light was brought toward the eyes, until at a comparatively near point the diplopia nearly disappeared, suggesting a paresis of divergence. The power of accommodation was affected. The patient read Jaeger's test type 14 at 4 inches (10 cm) on the right side and at 5 inches (12 cm) on the left side.

Various theories have been advanced in connection with the development of the exophthalmos, but so far none has proved entirely satisfactory.

The causation of palsy of the ocular muscles in cases of toxic goiter is still under discussion. Various theories have been presented by different authorities. When the preliminary report of this case was made an immediate operation to correct the muscular deviation was considered. In the discussion that followed Dr. Zentmayer suggested wisely that an operative procedure should be delayed until the congestion and the proptosis had further subsided. He cited the report by Naffziger of cases of malignant exophthalmos with blindness in which enucleation was followed by death.

Two months later, with the patient under ether anesthesia, tucking of the superior rectus muscle and recession of the inferior rectus muscle were carried out. In addition, tucking of the external rectus muscle and the recession of the internal rectus were performed. There

was considerable reaction to these procedures, as might be expected, but with the use of ice compresses the eyes quieted, and binocular single vision was exhibited and still maintains

CATARACT OPERATION TO REDUCE THE INCIDENCE OF PROLAPSE OF THE IRIS DR FRANK C PARKER (by invitation)

This article will be published in full in a later issue of the ARCHIVES

CLINICAL SIGNIFICANCE OF THE RETINAL CHANGES IN LEUKEMIA DR GLEN G GIBSON

This paper will be published in a later issue of the ARCHIVES

EXTERNAL ORBITOTOMY DR EDMUND B SPAETH

(A motion picture in colors was presented, demonstrating the technic of orbitotomy by the external route without resection of the bone for the removal of retinobulbar tumor) The tumor was in the muscle cone. Microscopic examination after its removal showed it to be a glioma of the nerve. In spite of the successful orbitotomy, it was thought wise in this case to do an enucleation for the satisfactory implantation of radium

NEW YORK ACADEMY OF MEDICINE, SECTION
OF OPHTHALMOLOGY

JAMES W WHITE, M D, *Chairman*

Feb 21, 1938

RUDOLF AEBLI, M D, *Secretary*

SUPRASellar MENINGIOMA DR KAUFMAN SCHLIVEK

A case of suprasellar meningioma is presented to demonstrate how proper visual fields, correctly interpreted, can make an elusive diagnosis straightforward

L K, a woman aged 51, complained of loss of vision in her left eye for one and one-half years. Aside from controlled diabetes and treatment for obesity, her past history was unimportant. Physical examination gave essentially negative results. Vision was reduced to 3/200 in the left eye, that in the right eye was 15/15. The field of vision of the left eye showed a temporal hemianopia and that of the right eye showed reduction of the upper temporal quadrant for color. Roentgenograms were normal. The patient had been treated for retrobulbar neuritis and was encouraged in the belief that the condition was improving. Other oculists suggested vascular disease and a macular pathologic process as the cause. However, on proper analysis of the case I concluded that the lesion was a suprasellar tumor. This was confirmed by encephalography and proved by successful operation.

The vision and the visual fields showed marked improvement. At the last examination, several months after operation, vision in the right eye was 15/15 and in left eye 15/30 + +, and the visual fields were practically completely restored.

TOTAL SYMBLEPHARON WITH PLASTIC REPAIR DR W P GRIFFEY
(by invitation)

Anterior, or partial, symblepharon may be readily repaired by excising the adhesions from the globe and covering the denuded area with a sliding or pedunculated conjunctival graft. Posterior, or complete, symblepharon is a more difficult problem and at times is not correctable by surgical means.

Aside from facial disfigurement, symblepharon may give rise to loss of sight and interfere with the ocular movements.

The case reported here is one of posterior, or complete, symblepharon involving the right lower lid. Considerable loss of motion was observed in the visual field away from the adhesions. There were also some astigmatic changes in the affected eye.

Mr J O, a white man aged 45, suffered from a lime burn of the right eye in May 1937, while working with plaster. The eye was described at that time as showing a burn of the margins of the upper and lower lids, nasally. There was marked destruction of tissue about the lower canaliculus. The conjunctiva on the nasal side of the globe, inferiorly, and on the lower lid was destroyed. The cornea was ulcerated from 3 to 7 o'clock at the limbus and inward to beyond the center of the pupil.

Six months later, at which time the patient was operated on, a firm adhesion had formed between the lower lid and the eyeball. Fibrous tissue extended into the cornea from 3 to 5 o'clock peripherally and centrally to the margin of the pupillary area. The inferior punctum was drawn laterally to a point near the limbus.

The patient complained of a sensation of constant pulling on the eye and diplopia on gazing to the right. The diplopia was most annoying when he looked up and to the right. The vision in the right eye was 20/30 and in the left eye 20/20.

The adhesion was excised from the cornea, local anesthesia being used. Then, by means of scissors, the fibrous mass was separated from the globe well down to the lower fornix. The bare area on the globe was covered by means of sliding conjunctival flaps. A free conjunctival graft, 1.25 by 0.75 cm, was taken from the upper lid of the right eye and placed on the denuded inner surface of the lid. This graft was anchored in place by sutures, and a pressure dressing was applied. At the end of seven days the sutures were removed, and the eye was uncovered.

The cosmetic and functional results were satisfactory. The visual acuity was corrected to 20/20 by means of lenses.

SUBCONJUNCTIVAL CYST SIMULATING PROLAPSE OF THE LACRIMAL
GLAND DR JAMES W SMITH

The patient was presented before this section in May 1929 with bilateral dislocation of the lacrimal glands. The glands were subse-

quently successfully replaced into the lacrimal fossae by operation. The technic employed was described in the 1933 *Transactions of the Section on Ophthalmology of the American Medical Association*, page 43.

Four months ago two small conjunctival cysts were seen along the border of the palpebral lobe of the left lacrimal gland. The cysts ruptured during an attempt at removal. They were found attached to the scar of operation performed in June 1929. Two months ago the left upper lid appeared full, and a firm, movable, almond-shaped mass was palpated in the temporal quarter, which could be replaced on pressure into the orbit. A diagnosis of recurrent prolapse of the lacrimal gland was made, and removal of the gland was advised.

A transverse section of the lid exposed a large cyst attached by a broad pedicle to scar tissue and the septum orbital just inferior to the lacrimal fossa. The lacrimal gland was found to be in normal position. Pathologic examination revealed that the cyst was probably formed from remnants of the lacrimal gland left behind at the time of operation eight years before. The original description of the replacement operation indicated that a small portion of the palpebral lobe contiguous with the fornix conjunctivae could not be freed and doubtlessly remained outside the fossa, with the resultant formation of a subconjunctival cyst.

SUBCONJUNCTIVAL SECTION OF THE DUCTULES OF THE LACRIMAL GLAND AS A CURE FOR EPIPHORA. DR. P. CHALMERS JAMESON, Brooklyn (by invitation)

Section of the ductules of the lacrimal gland in cases of epiphora is more advantageous than the more heroic operation of extirpation of the lacrimal glands. The postorbital region is not invaded, and satisfactory cutting off of the desired amount of secretion is obtained by a comparatively easy subconjunctival procedure. The anatomy should be visualized.

Whitnall states that the ductules seldom exceed a total of 12. Nearly all open separately along a line about 4 or 5 mm. above the upper convex border of the tarsal plate. (They open when the lid is everted.)

If these ductules are severed subconjunctivally in this region, communication between the glands and the conjunctival sac is disestablished. The product of the lacrimal gland, after sectioning, is discharged beneath the conjunctiva and does not enter the sac.

It must again be remembered that the region just described is not above, but below, the tarsal curve when the lid is doubly everted.

The technic for section of the ductules follows.

The lid is doubly everted by forceps. The fornix is thoroughly exposed and put on stretch. An incision 4 mm. long is made at the fornix of the outer canthus. Stevens' scissors are introduced in this opening, and a subconjunctival separation of the conjunctiva from its basal tissues is made, measuring about 7 mm. broad and 14 mm. long. This sections the ductules. The scissors can be seen through the translucent conjunctiva, and the secretion of the ductules sometimes can be seen to bulge the conjunctiva.

In making this separation, the distinctive landmark is the upper tarsal curve. The breadth of this conjunctival separation should extend from the curve to a line 7 or 8 mm. below.

The reaction is slight. Edema of the orbital tissues is not noticeable. The cheek on the side operated on swells slightly, showing that the secretion is discharging into the tissues. This rapidly disappears. The patient can go home immediately.

The after-treatment consists of ice compresses and sterilization of the sac, if there is any reaction.

DISCUSSION

DR JAMES W. WHITE: I was fortunate enough to see one of the patients to whom Dr. Jameson referred. I saw the second eye operated on, the patient was so satisfied with the result in the first eye that he came back for an operation on the other eye. This was the second case reported by Dr. Jameson.

DR LEWIS WEBB CRIGLER: I should like to ask if Dr. Jameson thinks that this procedure might be advantageous immediately after extirpation of the sac in cases of chronic dacrocystitis. It would be a more simple procedure than the Toti operation or one of its modifications.

DR P. CHALMERS JAMESON: I think that this procedure might be used to an advantage in cases of chronic dacrocystitis, the only disadvantage would be in not knowing just how the secretion is going to react after the operation. I should be inclined to do the double procedure at the time of removal of the sac, possibly not on all the ductules. I think that one of the great advantages of this method is that one can grade it, one can section only half of the ductules, or less, by cutting down the area of operation.

DR JULIUS WOLFF: May I ask whether Dr. Jameson thinks that possibly an external canthotomy might aid in everting the lid and making the operative area more accessible?

DR P. CHALMERS JAMESON: I think that a canthotomy might be of help, but I do not think that it is necessary as the entire area of operation is so easily exposed.

DR JAMES W. WHITE: That is my impression, I did this operation after seeing Dr. Jameson do it, and a canthotomy seems unnecessary.

OBSTETRIC OPHTHALMOLOGY DR DEWEY KATZ (by invitation)

(The subject was discussed in the light of the present knowledge under the headings of changes in the visual field, ophthalmologic aspects of the toxemias of pregnancy, retinal detachment in pregnancy, ophthalmologic indications for the termination of pregnancy and sterilization, birth injuries, ocular disease in the puerperium and lactation period and ocular disease of the fetus and of the newborn.)

DISCUSSION

DR ALVIN J. B. TILLMAN: I hope you have all enjoyed Dr. Katz's paper as much as I have. I for one have been entirely unfamiliar with a great deal of what has been said here, and in discussion I shall confine myself to a few of the great number of topics covered by Dr. Katz. As an internist I have for several years been interested in the toxemias of pregnancy, and in the course of a study of these I have collected data

on a large number of cases in which a long follow-up study was made. For this meeting I went over the records of these cases from two points of view—first, for a description of the retinal findings and, second, for the results of follow-up study. I have been able to study 109 cases in which the patients were followed on the average of seven years, and of these, 67 per cent showed hypertension. Of 26 patients who showed no retinal damage during the toxemia, 43 per cent showed hypertension later. The entire series of cases was analyzed as to vascular change, edema of the disk and the presence of retinal hemorrhage or of exudate, and the following facts were noted. Of a total of 99 patients, 71.7 per cent of those who showed vascular change alone had hypertension later. Of those with edema of the disk, 63.8 per cent later showed hypertension after an average of six and one-half years. Of the patients with retinal hemorrhage and exudate, 85 per cent showed hypertension after a period of from seven to eight years.

In agreement with what has been said here, there were 16 patients who showed vascular change before the seventh month, and of these 16, all showed hypertension later, and 2 died four and four and one-half years, respectively, after the toxic pregnancy.

Thirteen patients in the series showed vascular change, marked retinal hemorrhage and exudate and edema of the disk, and after an average of from six to seven years later, none was normal and 1 had died. Of these 13 patients with toxemic pregnancies, 70 per cent had stillbirths, that is, 9 of the 13 had stillbirths, and the remaining 4 had premature deliveries with a dubious outcome for the babies. The correlation between this type of retinal picture and the poor, dubious or fatal outcome can be seen.

I have had 5 patients with retinal detachment, and in these, too, there was an extremely poor outcome. Three of the patients had stillbirths and 2 had premature deliveries, and during the follow-up study, which ranged from three to eight years, only 1 patient was found to be free from hypertensive disease. Four of the 5 patients showed severe hypertension, 1 was normal.

There is one point on which I differ from the ophthalmologist, that is, his advice to the obstetrician in the presence of retinal hemorrhage in the vomiting of pregnancy. I have seen 3 patients with this picture. Of these 3, 2 died and 1 recovered. One of the 2 who died had had the pregnancy terminated two days after the finding of the retinal picture. The other patient, as a result of what happened to the first patient, did not have the pregnancy terminated, and she died too. The third had the pregnancy terminated before the onset of retinal hemorrhage, but this developed a few hours later. The patient survived, but paraplegia developed, which lasted for several months, and I believe now, four years after this event, that she still shows evidence of it. In spite of the fact that vitamins B and C are used intravenously in the treatment of such patients, I am against allowing pregnancy to continue. Vitamins B and C have their place in the treatment of pernicious vomiting long before retinal hemorrhage takes place. The vitamins need time to act, and the immediate condition of this type of patient is critical. If in spite of this treatment retinal hemorrhage develops, the outcome may be unfavorable. A few months ago I had a patient of this type who was

treated in this manner and was permitted to continue with pregnancy. Two months after the decision, and in spite of adequate doses of vitamins, hemiplegia developed.

In summing up the few points I have made I should like to say that as a result of this meeting I have added considerably to my knowledge in this small field of the toxemias of pregnancy. This knowledge should be helpful in formulating prognostications in this type of case. Judging by the cases that I have seen, hypertension will eventually develop in the patient with complete neuroretinitis, whereas only from 40 to 50 per cent of those with other types of neuroretinitis will have this sequel. It is therefore highly important to study the retina in the active phase of toxemia and in the process of evolution, though one may be tempted to forego this in the presence of severe illness of the patient in the effort not to disturb her. Still, in the interests of the outcome, retinal examination is important.

DR FRANCES RICHMAN Dr Katz has thoroughly and concisely reviewed the various ophthalmologic conditions associated with pregnancy and has brought out the need in these conditions for consultation and cooperation between the ophthalmologist and the obstetrician.

In a consideration of the ophthalmologic conditions of the newborn due to birth stresses, to which I shall limit my discussion, it will be seen that cooperation between the two branches of medicine is especially important if the infant is to be assured of perfect visual acuity at birth.

In an ophthalmoscopic study of 1,500 newborn infants here and abroad, I have found their classification simplified by separating them into two groups: those whose eyes have apparently successfully withstood the stresses of birth and appear normal in all respects and those whose eyes have been unable to withstand the stresses of birth and therefore show ocular changes as a result. The term "birth injuries" in this connection is to be avoided. It has a connotation signifying trauma due either to accident or to inexperience and should be reserved for the extremely rare cases of such nature reported in the literature. On the other hand, the term "birth stresses" signifies all factors associated with pregnancy and birth which constitute a menace to the health of the mother or the child. Against these the careful obstetrician can take suitable measures, for they lend themselves to analysis and study.

Birth stresses affecting the eye are biochemical and mechanical, the former beginning with conception and operating incessantly until labor begins, the mechanical stresses then predominate until the child is born and the cord tied. The fetal nutriment conveyed by way of the maternal blood stream may at one time or another during gestation lack necessary elements or actually contain substances deleterious to the vitality of the ocular structures. Any impoverishment of the maternal blood, whether it be due to an avitaminosis, to a hidden focus of infection or to other factors which Dr Katz has pointed out, may leave an imprint on the infant's eye that ill prepares it to cope with the innumerable mechanical stresses to which it is subjected during birth, be this ever so normal. The immediate effects produced by the interaction of both groups of stresses, passive and active, so far as they have affected the eyes of the infant, can be readily ascertained by ophthalmoscopic examination, the eye being unique among sense organs in that it yields its secrets to inspection.

The presence of hemorrhage in the fundus is the most common sign that an eye has been unable to withstand the stresses of birth. Hemorrhages are of various types and extent and location and can be classified according to the retinal layer which each involves. Bright red stipitate extravasations, evidently situated in the nerve fiber layer, are absorbed rapidly and leave no trace of their presence, hence the necessity for early examination, on the first or the second day. A preretinal type of hemorrhage is next in frequency. This is dark red and shiny and is well circumscribed and long lasting. At the summit of each collection of blood is seen the tiny reflex of the ophthalmoscopic light, which definitely shows that the hemorrhages lie in front of the retina, the inference is that in spite of appearance and duration they may have the least damaging effect. Diffusely round, dull red hemorrhages situated in the layers deeper than the nerve fibers may be the ones having the greatest potential damaging power. A type of hemorrhage combining flame-shaped extremities and spindle-shaped white centers of serum is often seen.

Aside from these, other signs of stress are edema of the disk, retina and macula, stretching of the disk, ruptures of Descemet's membrane, and subconjunctival hemorrhages.

A routine "eyepoint" of every newborn infant, as I have described it elsewhere, containing a detailed description of the normal as well as the pathologic conditions seen at birth, would enable the obstetrician to study his case in retrospect as to possible causes of stress and their avoidance in future, it would furnish ophthalmologists with information of extreme value in studying and eventually clarifying ophthalmologic problems of a controversial nature.

DR MARTIN COHEN. In these cases of toxemia of pregnancy I am much interested in how long the high blood pressure had existed prior to the toxemia.

DR ALVIN J. B. TILLMAN. It is impossible to say with any degree of accuracy. Some patients were admitted to the hospital for emergency measures and were seen for the first time when the hemorrhages were already present. No information as to antecedents could be obtained on these cases. I have more data on the 16 patients I mentioned who had vascular change prior to the seventh month, and I should say that about one half of this group had had hypertension over a variable period of time.

DR DEWEY KATZ. I make no claim for originality or priority of any of the statements made this evening. My main purpose has been to try to bring some sort of order out of chaos in the field of obstetric ophthalmology and to plead for a closer cooperation between the ophthalmologist and the obstetrician.

Book Reviews

A Text-Book of Ophthalmic Operations By Harold Grimsdale, M B, F R C S, Consulting Ophthalmic Surgeon, St George's Hospital, London, and Elmore Biewerton, F R C S, Consulting Ophthalmic Surgeon, Metropolitan Hospital, London Third Edition Price, \$6 Cloth Pp 322, with 105 illustrations Baltimore William Wood & Company, 1937

The third edition of "A Text-Book of Ophthalmic Operations" is larger in format than the second edition and less in pagination. There are 12 chapters, in which are discussed in rotation operations on the muscles, operations for ptosis, operations on the lids, operations on the conjunctiva, enucleation, operations on the lacrimal apparatus, operations for cataract, after-cataract and glaucoma, respectively, and operations on the cornea, the sclerotic and the iris and for the removal of foreign bodies.

The content has been well arranged. The grouping of procedures, not always easy to accomplish, has been carefully worked out, as has also the division of operations into their respective stages. The latter serves as an excellent simplified guide for the student.

The illustrations, with the eye blocked in by a border to represent the lids, are adopted in this edition as previously. Though this manner of presentation is striking and novel and may accentuate the picture portrayal, it does not add beauty to the volume.

The artist has done his work well. Being an artist, however, he has frequently introduced a large triangular corneal reflex, which in some instances may confuse the operative procedure for the student.

The anatomic and physiologic prefaces to the chapters are excellent, well thought out and simply described. They constitute one of the strong contributions to the book.

While the descriptive text is brief for the most part, it is clear, and every chapter is supplemented by a complete bibliography. Modesty on the part of the authors is probably the reason why so little of their personal work is portrayed or described. Comment also in regard to procedure is discreet, although in some cases pointed.

The presentation of a large number of operations without evaluation may leave the student confused.

The first chapter on muscles gives brief, but lucid, descriptions of various operations, among which are the newer procedures. It might be mentioned that two of the most popular operations of advancement and shortening are omitted—one, the Worth procedure (described in the previous edition), probably the most popular operation of this type in Great Britain, and the other, the Reese method (shortening), the most frequently practiced in Eastern America.

More space might have been given to the special corrective capabilities of advancement and recession, the degree of correction obtained by each and the specific value of each in its own sphere, with emphasis on the necessity of using the operation best suited to the type of case at hand. Correlated with this, one might wish there had been stressed the necessity of exact diagnosis based not on deviation alone but on a careful computa-

tion and investigation of underlying conditions before and after exposure of the field, this to include with deviation an estimation of the innervation, muscularization and fascial conditions in a given case

In the second chapter on ptosis the student will find a goodly array of operations from which to choose

The desire of the authors to eliminate largely historical matter, as mentioned in the preface, has, to a certain extent, been carried out. It has not been altogether accomplished, however, as this would be impossible in a well rounded volume. This may explain why 3 pages are devoted to a certain operation for ptosis of the skin. There is little actual benefit the student could accrue from the performance of this procedure, as the deformity left after such a plastic operation is usually worse than the defect.

In chapters III and IV 50 pages are devoted to operations on the lids. Guides to selection are made plainer by the excellent preface to this chapter. The operations are well, though briefly, described, and the illustrations are good.

In the section on conjunctival operations, stereotype procedures are portrayed. This type of work presents many problems, so that much can be left to the ingenuity of the operator.

The necessities for enucleation, with the varying technics, and also obliteration of the socket and opticociliary neurectomy are ably discussed. The technic of insertion of glass balls, implants, foreign material and paraffin, and the technic of exenteration are described.

The operative procedures dealing with the lacrimal apparatus are well selected. The technic of the extirpation of the lacrimal glands is described. The innocuous subconjunctival section of the ductules will probably render these more dangerous procedures unnecessary in the future.

Sixty-five pages are devoted to cataract. This chapter is well done and well balanced. Surgeons may find some omissions of their favorite procedures. The reviewer is much enamored with the bridge operations, which are not mentioned, and an important omission may be the through and through needling of Ziegler for juvenile cataract.

The closing chapters are devoted to the surgical treatment of glaucoma and to operations on the cornea, sclerotic and iris and for the removal of intraocular foreign bodies.

The preface to the chapter on glaucoma is most excellent. In this chapter the principles are briefly and simply described and cover the field.

A short but clear outline of the work done to date for retinal detachment is inserted toward the end where the work of Gonin, Guist, Laïsson, Lindner, Šafář, Szily, Vogt and Weve is described. This brings the volume to a close.

As already stated, there are possibly some omissions on major subjects, and the surgeon-at-large may not find his favorite procedure or technic mentioned, but if this were done for all, it would take a library to hold the volumes.

The student will find a large range of operations briefly but well described. These can always be supplemented by a profuse and excellent bibliography.

P CHALMERS JAMESON

Untersuchungen über die Augenhöhlen des Menschen in verschiedenen Lebensaltern. By Per Pallin Pp 107, with 22 figures and 12 tables Stockholm Isaac Marcus Boktryckeri-Aktiebolag, 1937

In his introduction Pallin states that the present study was undertaken because of the well known fact that a convergent strabismus of moderate or low degree often decreases or even disappears at about the age of 15 and that some patients who have been operated at an early age often eventually have divergent strabismus

The author has measured 240 skulls of newly born infants, young children, older children and youths As a control, the measurements of 200 adult skulls (100 of male and 100 of female origin) were used

Among the measurements made were the inclination of the two orbits to each other, the angle between the outer walls of the orbits, the angle between the inner walls of the orbits, the depth of the orbit, the biorbital width, the interorbital width, the distance between the optic foramina and the distance between the midpoints of the orbital openings

Each group of similar measurements has been treated statistically to obtain the mean, the mean deviation and the mean deviation of the mean, with a discussion of the extent to which the differences between the various means can be used as a basis for ascribing any changes noted to growth

The author also reports the measurements of the orbits of 6 fetuses, which were 63, 86, 105, 185, 210 and 330 mm in length

In his summary Pallin states that starting about the middle of fetal life and continuing after birth there is a gradual turning forward of the large wing of the sphenoid bone The depth of the orbit also increases, as does the distance between the midpoints of the orbital openings The angle between the orbital axes becomes smaller, and there is a corresponding decrease in the angle between the axes of the muscle cones

After late childhood there is no further increase in the depth of the orbit At this time, owing to the relatively greater increase in the biorbital and the interorbital width, the midpoints of the orbital openings turn out more than the optic foramina do, so that there is an increase in the divergence of the orbital axes This increased divergence, however, is slight when compared with the diminution of the biorbital angle which occurs after birth

During further growth there is a further increase in the biorbital angle, which ceases at about the age of 20

The biorbital angle has the following values in late fetal life, 61 degrees, in newborn infants, 59.73 degrees, in young children, 49.98 degrees, in older children, 52.79 degrees, in youths, 52.90 degrees and in adults, 54.14 degrees One must assume that there are corresponding changes in the angle between the axes of the muscle cones

The medial orbital walls converge posteriorly before birth Owing to the development of the ethmoid cells, which bulge more and more into the orbit, increasing the width of the interorbital region posteriorly, these walls become approximately parallel by late childhood and maintain this position in later life

The external orbital walls also change their position. During the latter part of fetal life there is a turning forward of the orbital surfaces of the great wings of the sphenoid bone, which continues until late childhood. Also, the frontal processes of the malar bones gradually diverge anteriorly, with regard to their orbital surfaces, until adolescence, with a corresponding definite enlargement of the orbital width.

Pallin has not only done a tremendous amount of work in measuring skulls and tabulating his results, but, from his statistical treatment of the data, he has also indicated which changes are significant, which are of questionable value and which are of no significance, at least for the number of skulls studied.

This is another one of the excellent monographs which have recently been issuing from the Scandinavian countries. The work was carried on at the University of Uppsala under the directions of Professors Frederik Berg and Erik Agduhr. In my opinion it is a valuable contribution to the developmental anatomy of the orbit, and it should be of great value to future workers in the same field.

W F DUGGAN

Vitamine und Hormone in ihren Beziehungen zur Augenheilkunde (Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten, no. 25). By Prof Dr Kurt Wachholder, Prof Dr Werner Kyrieleis and Dozent Dr Reinhard Braun, with an introduction by Prof Dr C Behr. Price, Swiss francs 9.40. Pp 80, with 21 illustrations. Berlin: S. Karger, 1938.

Three surveys which were presented in a symposium on vitamins and hormones and their relations to ophthalmology at the last meeting of the North-West German Ophthalmological Society are reprinted from *Zeitschrift für Augenheilkunde*. This publication for ophthalmologists is most timely in view of the importance of this research and of the rapid progress of the knowledge in this field. Wachholder's introductory survey is a basic study of the role played by vitamins and hormones in the animal economy as a whole. He calls attention to what Lumière has called the renaissance of humoral pathology and to how this has colored clinical study as well as laboratory research and experiment. While vitamins are correctly named, as they are nutritional elements essential to life, only one is, strictly speaking, an amine. The hormones, chemical messengers having no nutritional function, are concerned with the medication of neural impulses. The physical, neurologic and chemical nature of these reactions is analyzed and presented in detail and with admirable clarity. It is shown that vitamins are closely allied to enzymes and may act either as stabilizers or as catalyzers. They may be produced in the animal organism, or at least prepared in the form of provitamins, while substances which are practically hormones are found in plants. Carbonic acid, acetylcholine and a substance closely akin to epinephrine are general (*Allgemein*) hormones which can be isolated even from unicellular organisms. Kyrieleis discusses ocular symptoms in diseases of the glands of internal secretion. He notes the interdependence of the various members of the endocrine system and considers the factor of imbalance quite as important as hyperfunction or hypofunction of a single organ. The role of the vegetative nervous

system is also considered, as is that of the cerebrum, especially the midbrain. The last-named structure is considered at length and most instructively in connection with the ocular manifestations of hyperthyroidism, such as Graefe's, Stellwag's, Mobius' and Dalrymple's sign, in exophthalmic goiter and in other diseases, such as epidemic encephalitis, postencephalitic parkinsonism and myotonia congenita (Thomsen). A detailed study of the cause and pathomechanism of exophthalmos then follows. The occurrence and the characteristic form of cataract in cases of tetany, latent and manifest, as well as in cases of certain closely allied diseases, such as myotonia dystrophica, are described in detail. Acromegaly, Addison's disease, diabetes and the various dystrophies associated with pathologic changes and dysfunction of the pituitary gland are also described. Tumors of the hypophysis and their mechanical effect on the optic nerve and in the causation of increased intracranial tension and possible endocrine factors in corneal dystrophies and in certain syndromes, such as those which bear the names of Crouzon, Hand-Schuller-Christian, Friedreich and Wilson, are considered briefly. This survey contains a wealth of clinical information and data of biopathologic research. It is amply documented and beautifully and profusely illustrated. The third and concluding article on vitamins and the eye, by Braun, although short, is particularly valuable in that it takes up the thread of discussion from the generalizing introductory remarks of Wachholder's basic study and analyzes the role of the individual vitamins in various ocular conditions, physiologic as well as pathologic. This includes the subject of the intraocular production of vitamins, the chemism of visual purple and the factors of photosensitization and light adaptation. Braun lays stress on the importance of recognizing the deleterious action of vitamin imbalance, just as in a similar discrepancy affecting the endocrine system. The interrelations and reciprocal actions, while less thoroughly understood, are without much doubt of equal practical significance.

PERCY FRIDENBERG

Stereoskopische Bilder für schielende Kinder By Prof C H Sattler, Königsberg. Third edition. Price, 6 marks. Fifty-four double plates. Stuttgart. Ferdinand Enke, 1937.

The first edition of these plates appeared in 1928 and the second edition in 1933 (reviewed in the October 1934 issue of the ARCHIVES, page 625). The present edition consists of 54 split cards for diagnosis and therapy.

In the accompanying book of instructions are given suitable questions for use with the various slides as well as the procedure to be followed in using the plates.

The appearance of a new edition testifies to the popularity of the Sattler stereoscopic pictures.

W F DUGGAN

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Place Gulfport, Miss Time May 8, 1939

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Acting President Dr N Darrell Harvey, 112 Waterman St, Providence
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 second Thursday in October, December, February and April

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 September to May

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 October to April, inclusive

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Chairman Each member, in alphabetical order
 Secretary Dr Sam H Sonders, Medical Arts Bldg, Memphis, Tenn
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 D C
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 Place Episcopal Eye, Ear and Throat Hospital Time 8 p m, first Monday
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ARCHIVES OF OPHTHALMOLOGY

VOLUME 20 (old series Volume 77) SEPTEMBER 1938

NUMBER 3

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INTRAOCULAR TENSION IN CASES OF SARCOMA OF THE CHOROID AND CILIARY BODY

JOHN H DUNNINGTON, MD

NEW YORK

This study of the intraocular tension in eyes containing new growths was undertaken in an endeavor to determine the diagnostic value of this test in the differential diagnosis between a serous detachment of the retina and one due to an intraocular neoplasm

The occurrence of secondary glaucoma as a late manifestation of intraocular sarcoma is universally recognized, but the behavior of the intraocular tension in the early stages of the disease is not well known. The literature on this phase of the subject is confusing, for some authors have made no mention of it, while one stated "Tension is a valuable diagnostic point, in retinal detachment due to a serous collection it is generally decreased, in choroidal sarcoma it is usually normal at first and becomes increased later" ¹ Still another ² stated "Too much reliance cannot be placed upon the tension of the eyeball as a distinguishing sign between sarcoma and retinal detachment because intraocular tension may be unaltered in each instance, although as C Devereux Marshall has shown it is probably never diminished in undoubted cases of choroidal sarcoma while it may be reduced in cases of sarcoma of the ciliary body"

The earliest reference to hypotony in cases of intraocular neoplasm was made by Laurence ³ in 1866. He reported a case in which the eyeball was very soft. The condition was not diagnosed until the eye was enucleated after the patient had previously been subjected to iridectomy for glaucoma with subsequent hypotony. Laurence cited this case

From the Institute of Ophthalmology, Presbyterian Hospital

Read before the New York Academy of Medicine, Section of Ophthalmology, May 16, 1938

1 Ball, J M, cited by Findlay, E K. Tumors of the Choroid, in Wood, C A. The American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol 3, p 2180

2 de Schweinitz, G E. Diseases of the Eye, ed 10, Philadelphia, W B Saunders Company, 1924, p 405

3 Laurence, J Z. Ophth Rev 2 378, 1866

as an exception to von Graefe's dictum that "the degree of tension of the eyeball is a guide to diagnosis" In commenting on this unusual finding, he stated "Mr Nunneley of Leeds told me he had frequently met with soft globes in the first stage of malignant disease of the eyeball" The first real study on this subject was made in 1889 by Marshall,⁴ who concluded that the intraocular tension was seldom lowered in globes containing new growths He found the tension increased in 66.6 per cent, normal in 30.3 per cent and lowered in 3.03 per cent Carrying his studies still further, he reported in 1896 on 100 cases in which he "compared the pathological state with the condition they presented before excision" From these studies he concluded that "diminished tension by no means uncommonly occurs when the ciliary body is involved, but I have been unable to find an undoubted case in which the tension has been reduced when the choroid only is affected"

In 1898 Leber and Krahnstover⁶ reviewed the literature on hypotony in cases of intraocular sarcoma They reported on 37 such cases, adding the reports of several of their own to those previously recorded No attempt was made to measure the intraocular tension of both eyes, and the series of cases was largely made up of those in which phthisis bulbi was a late manifestation In 11, there was profound hypotony without actual shrinkage of the eyeball, but the authors contended that these eyes were in a transitional stage They showed that phthisis bulbi is usually not the result of a perforation and concluded that the hypotony and shrinkage of the eyeball come from a chronic plastic inflammation due to bacteria rather than from the toxins formed by necrosis of the tumor They speculated that the frequency of endogenous infection may be due to the fact that necrotic tumor tissue forms a good medium for bacterial growth

Franz⁷ in 1920 was the first to record the intraocular tension of both eyes in his report on hypotony as an early manifestation of choroidal sarcoma He reported the detailed observations in 3 such cases and concluded from his study that the lowered intraocular tension was not due to bacterial infection, uveal inflammation or necrosis He discussed the causes of hypotony in cases of detachment of the retina and stated that the same causative factors were responsible for its occurrence in association with tumor The tension of the affected eye was 6 mm of mercury lower than that of the other eye in 1 of his cases, 8 mm in 1 case and 12 mm in another

In the same year Francis⁸ reported an interesting case of intraocular sarcoma in which the patient was followed for five years on account

4 Marshall, C D Roy London Ophth Hosp Rep **15** 57, 1889

5 Marshall, C D Tr Ophth Soc U Kingdom **16** 155, 1896

6 Leber, T, and Krahnstover A Arch f Ophth **45** 164, 1898

7 Franz, G Klin Monatsbl f Augenh **64** 348, 1920

8 Francis, L M Am J Ophth **3** 872, 1920

of a macular lesion. When he was first seen, the intraocular tension was 20 mm of mercury, while at the time of enucleation it was only 5 mm. Pathologic examination failed to reveal a perforation, and the tumor was located in the choroid near the optic disk. So while the existence of hypotony in association with intraocular sarcoma has been known for many years, no recent attempt has been made to establish the frequency with which it occurs in the first stage of the disease.

ANALYSIS OF CASES

No case was considered in this analysis unless the clinical diagnosis was confirmed by a pathologic examination of the enucleated eye. Tonometric readings with a Schiotz tonometer were made on both eyes in every instance. Fifty-five cases of sarcoma of the choroid and the ciliary body have been analyzed, and the results are shown in table 1.

In 9 of the 15 cases in which the intraocular tension of the affected eye was higher than that of the sound eye, the disease had progressed

TABLE 1—*Comparative Tension of the Affected Eyes*

Tension	No. of Cases	Percentage
Higher	15	27.3
Equal	11	20.0
Lower	29	52.7

to the second stage, as there was definite secondary glaucoma present. With the exclusion of these 9 cases from consideration, it was found that the intraocular tension was either equal to or less than that of the fellow eye in 86.9 per cent of the remaining 46 cases. A definite lowering of the intraocular tension was recorded in 63 per cent of these cases. The difference in the tonometric readings for the two eyes varied from 2 to 10 mm of mercury, the average being 5 mm. The average tension in these 46 cases was 11.9 mm of mercury. It is interesting to note that in a previous report Macne and I⁹ found the average intraocular tension in 55 cases of serous detachment of the retina to be 11.1 mm of mercury. In none of these cases in which a lowered intraocular tension was found had the condition progressed through the glaucomatous state into one of phthisis bulbi. In every instance the diagnosis was made prior to the onset of glaucoma, i. e., in the first stage of the disease. It can therefore be said that in at least from 60 to 65 per cent of cases of sarcoma of the choroid and ciliary body a lowered intraocular tension is manifested in the early stages of the disease.

The 55 cases studied here were grouped according to the location of the primary site of the tumor, as shown in table 2.

⁹ Dunnington, J. H., and Macne, J. P. Detachment of the Retina. Operative Results in One Hundred and Fifty Cases, *Arch. Ophth.* **13**: 191 (Feb.) 1935.

TABLE 2—*Classification of Cases on Basis of Primary Site of Tumor*

Primary Site	No of Cases	Tension		
		Higher	Equal	Lower
Choroid	47	14 (29.8%)	8 (17%)	23 (53.2%)
Ciliary body	8	1 (12.5%)	2 (37.5%)	4 (50%)

TABLE 3—*Analysis of Fifty-Five Cases of Sarcoma of the Choroid and the Ciliary Body*

Case No	Eye Involved	Tension		Location of Tumor
		O D	O S	
Group 1 Higher Intramuscular Tension in the Affected Eye				
1	O S	12	13	Choroid
2	O D	27	23	Choroid
3	O S	15	18	Choroid
4	O D	18	16	Choroid
5	O S	12	14	Choroid
6	O D	23	19	Choroid
7	O S	19	70	Choroid
8	O S	15	25	Choroid
9	O S	12	33	Choroid
10	O S	10	52	Choroid
11	O D	44	18	Choroid
12	O D	70	16	Choroid
13	O S	16	49	Choroid
14	O S	15	48	Choroid
15	O S	12	33	Choroid
Group 2 Equal Intraocular Tension in Both Eyes				
16	O S	10	10	Choroid
17	O D	10	10	Choroid
18	O D	8	8	Choroid
19	O D	14	14	Choroid
20	O D	14	14	Choroid
21	O D	20	20	Choroid
22	O S	8	8	Choroid
23	O S	20	20	Choroid
24	O D	15	15	Ciliary body
25	O D	10	10	Ciliary body
26	O D	8	8	Ciliary body
Group 3 Lower Intraocular Tension in the Affected Eye				
27	O D	8	14	Choroid
28	O D	8	10	Choroid
29	O S	18	15	Choroid
30	O S	20	12	Choroid
31	O S	16	12	Choroid
32	O D	7	13.5	Choroid
33	O D	10	12	Choroid
34	O S	16	12	Choroid
35	O S	14	9	Choroid
36	O D	10	16	Choroid
37	O D	5	14	Choroid
38	O D	12	14	Choroid
39	O S	17	10	Choroid
40	O D	11	14	Choroid
41	O D	7	13	Choroid
42	O D	14	18	Choroid
43	O S	12	8	Choroid
44	O D	12	14	Choroid
45	O D	8	13	Choroid
46	O S	23	12	Choroid
47	O S	16	7	Choroid
48	O D	18	23	Choroid
49	O S	18	14	Choroid
50	O D	18	23	Choroid
51	O S	18	14	Choroid
52	O S	12	8	Ciliary body
53	O D	9	12	Ciliary body
54	O D	8	14	Ciliary body
55	O S	20	10	Ciliary body

A study of the figures in table 2 reveals that of the 47 cases in which the disease was diagnosed as being of choroidal origin there was a lower intraocular tension in the affected eye in 25 (53.2 per cent). While in some of the cases secondary involvement of the ciliary body may have been present, in many of them the lesion was confined entirely to the choroid.

The number of cases of primary involvement of the ciliary body is too small to justify any comment, unless the relative infrequency of increased intraocular tension in this group is worthy of note, having occurred in only 1 of the 8 cases.

SUMMARY

Fifty-five cases of intraocular sarcoma have been analyzed in an endeavor to determine the frequency as well as the manner in which the intraocular tension is affected by the disease. In 9 of these cases definite secondary glaucoma had intervened.

In 29 (63 per cent) of the remaining 46 cases, the tension of the affected eye was found to be lower than that of the sound eye. In 11 (23.9 per cent) the intraocular pressure of the two eyes was equal, while in 6 (13.1 per cent), the affected eye showed a slightly higher tension. The amount of lowering of the intraocular tension varied from 2 to 10 mm. of mercury, the average being 5 mm. This decrease in pressure was encountered just as frequently when the disease originated in the choroid as when it started in the ciliary body. It is therefore felt that an initial drop in the intraocular tension is the rule rather than the exception in the early stages of sarcoma of the choroid and ciliary body.

Dr. John M. Wheeler allowed me to incorporate the data for many of his cases in this report.

CLINICAL SIGNIFICANCE OF THE RETINAL CHANGES IN LEUKEMIA

GLEN G GIBSON, M D

PHILADELPHIA

Since Liebreich's description of the retinal changes in leukemia, numerous ophthalmologists have recorded their experiences in contributions which take into consideration the description, the incidence, the prognostic significance and the histologic picture of the various ocular lesions observed in the several types of this condition

This report includes the results of a study of the retinal changes and the laboratory findings in 22 cases in which a diagnosis of leukemia was made by the members of the medical department of the Temple University Medical School. The clinical diagnosis was confirmed in 9 cases at autopsy and in 7 additional cases by biopsy. None of the eyes was studied microscopically. The ophthalmologic and clinical data in these cases conform in most instances to the observations of Wagener,¹ Goldback² and Frank.³ The objects of this report are to discuss the ophthalmologic diagnosis and prognosis of leukemia and to call attention to the close parallelism between the amount of hemorrhage in the retina and the degree of anemia which is associated with the leukemia. This relation was noted by Borgeson and Wagener¹ in their excellent paper on leukemia.

DIAGNOSIS

In many cases it is possible for the ophthalmoscopist to make a positive diagnosis of leukemia without the assistance of the clinical data merely by the appearance of the fundus. In this series the retinal changes were characteristic of leukemia in 2 cases before the changes in the blood and the clinical signs were sufficiently advanced to warrant a diagnosis. The ophthalmologic diagnosis was subsequently confirmed at a later admission to the hospital. Consequently, it does not behoove

From the Department of Ophthalmology of Temple University Medical School

Read before the Eye Section of the College of Physicians of Philadelphia, April 21, 1938

1 Borgeson, E J, and Wagener, H P. Changes in the Eye in Leukemia, *Am J M Sc* **177** 663-676 (May) 1929

2 Goldback, J. Leukemic Retinitis, *Arch Ophth* **10** 808-817 (Dec) 1933

3 Frank, J. Leukemic Retinitis, *M J Australia* **1** 364 (March 23) 1935

the ophthalmologist who finds a typical retinitis of leukemia to change his opinion if the clinician is unable to confirm the diagnosis. Twelve patients presented retinal changes which were sufficiently characteristic to permit a positive diagnosis of leukemia to be made. In 2 of the cases the diagnosis made on the basis of the fundal changes was blood dyscrasia, and in 1 case it was retinitis of anemia, in 2 of the cases no classification was made. The fundi were reported as normal in 5 cases.

The first and most frequently observed sign in the cases of leukemia in this series was a change in the appearance of the retinal veins. They became darker and fuller than normal. This was the most helpful sign in making the diagnosis. This observation is not in accord with that of R. Foster Moore,⁴ who stated that the venous changes are the least significant. The retinal veins were recorded to be darker and fuller than normal in all except the 5 normal fundi. This sign almost invariably precedes the retinal hemorrhages and persists after they absorb during remissions. In the most advanced and typical forms the veins become dilated and tortuous and constricted at intervals, so as to resemble roughly a chain of sausages. In every case of bilateral involvement in which the retinal veins appear darker and fuller than normal, leukemia should be suspected, and a careful study of the fundi should be made to exclude such types of venous changes as are associated with anemia, polycythemia, congenital heart disease and congenitally large veins. This is not always easy or possible, but frequently one is rewarded for careful study by helpful differential points. Congenitally large veins are normal in color and quite tortuous and are usually matched by a similar tortuosity of the arteries in an otherwise normal fundus. In cases of anemia the rest of the fundus, including the disk, is pale. In the cases of leukemia the combination of dark full veins and pallor of the disks was not observed without other associated lesions. In cases of polycythemia there is a definite cyanotic color, and the arteries are also larger and darker than normal. In cases of congenital heart disease the arteries usually appear normal, and the veins which are larger than normal, are extremely cyanotic. One should exclude a diagnosis of leukemia in cases in which one observes the combination of normal disks and arteries associated with relatively full and dark retinal veins. If, however, in addition to the changes just enumerated, one finds one or more retinal hemorrhages, the possibility of the condition being leukemia is enhanced materially. Retinal hemorrhages were observed in 13 of the 22 cases in this series at the first observation. In 2 cases localized areas of exudate without hemor-

⁴ Moore R. F. *Medical Ophthalmology*, ed. 2. Philadelphia, P. Blakiston's Son & Co. 1925. chap. 10, p. 104.

hage were observed, hemorrhages occurring subsequently in the course of the disease. While the most typical hemorrhage of leukemia is a large round hemorrhage with a white center, this type is seldom observed in the earlier stages of the retinal changes. One may encounter almost any size or shape hemorrhage, with the possible exception of the small punctate hemorrhages so frequently observed in cases of diabetes. The degree of hemorrhage may vary from one small hemorrhage to almost complete infiltration of the retina. The amount of the hemorrhage seems to be proportional to the degree of anemia associated with the leukemia, hence in certain cases of leukemia a retinal picture is observed that is indistinguishable from the retinitis of anemia. In 2 of the cases the hemorrhage was so extensive that it simulated bilateral thrombosis of the central retinal vein. If, in addition to the foregoing changes, one observes a narrow white line on each side of the vein, the condition is true retinitis of leukemia in the limited sense, with perivenous diapedesis of the white blood cells. This perivenous infiltration does not in any way deform the vein, and this is helpful in differentiating the condition from periphlebitis. In 2 cases there was edema of the disk. Rarely one observes that the arteries become slightly fuller than normal and that the reflex stripe on the artery is accentuated.

PROGNOSIS

While the ultimate prognosis in cases of leukemia is almost invariably fatal, it is difficult to attempt to judge the immediate prognosis by the retinal picture. Occasionally the disease runs its complete course without the development of ocular signs. Consequently, a normal ocular picture does not necessarily render the immediate prognosis good. Usually in fatal cases an extensive hemorrhagic infiltration of the retina develops before death. However, it sometimes happens that a patient with maximum retinal involvement will enjoy a spontaneous remission both from the retinal and the clinical standpoint. Consequently extensive retinal hemorrhage, though usually a grave sign, does not necessarily have an immediate serious significance. Aside from these exceptions, however, one gets the clinical impression that there is a fairly close relation between the amount of retinal hemorrhage and the clinical condition of the patient. As a rule, when the patient's general condition gets worse more retinal hemorrhages occur, and when the patient improves clinically, the hemorrhages tend to absorb.

CORRELATION BETWEEN RETINAL HEMORRHAGE AND ANEMIA

In the analysis of these cases there was one observation which seems to be of clinical importance, that is, the close parallelism between the amount of anemia and the extent of the retinal hemorrhage. For

descriptive purposes, the cases were divided into four groups on the basis of the amount of retinal hemorrhage. In group 1 were included those cases in which there were from one to three retinal hemorrhages (fig 1). Group 2 included those in which from about four to ten retinal hemorrhages occurred, while group 3 (fig 2) consisted of those in which the hemorrhages were too numerous to count, and group 4 (fig 3), those in which there was a maximum degree of retinal hemorrhage. The cases are arranged in order in figure 4, so that the ones in which the red cell counts were the lowest are at the top of the chart and those in which the red cell counts were the highest are at the



Fig 1 (group 1) —Early retinal picture in a case of leukemia. The veins are slightly full and dark, the hemorrhage is near the normal disk, the arteries are normal, and exudate is seen above the macula. The rest of the white spots are high lights.

bottom. The degree of hemorrhage was plotted on the basis of the foregoing classification. It is immediately apparent that the cases in which the red cell counts were the lowest were the ones in which the most extensive degree of retinal hemorrhage occurred. Cases of anemia of a moderate degree are in general the ones in which the more moderate amount of retinal hemorrhage occurs, whereas those in which the red cell counts are relatively high are the ones in which no retinal hemorrhage occurs. This close correlation indicates that there must be a close relation between the amount of hemorrhage and the degree of

anemia Further confirmation of this was furnished by examination of 3 patients who on first observation had no retinal hemorrhages and only a mild degree of anemia Subsequently, it was observed that retinal



Fig 2 (group 3) —Slight edema and pallor of the disk, as shown in the right and left eye of the same patient The veins are dark, full and tortuous, the arteries are normal, and the hemorrhages are too numerous to count

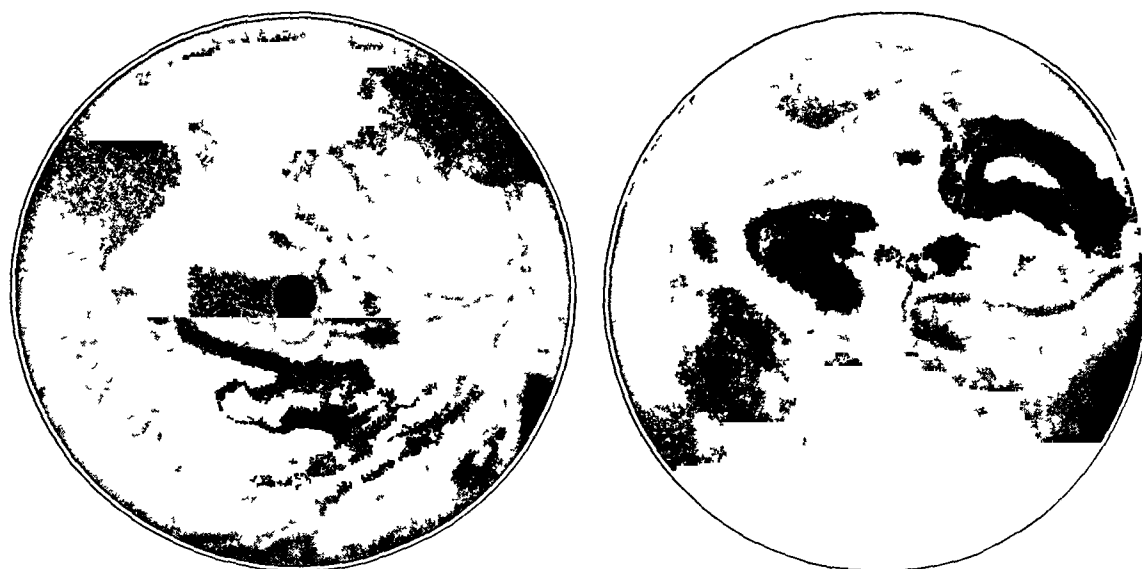


Fig 3 (group 4) —Maximum degree of retinal hemorrhage with white centers, as shown in the right and left eyes of the same patient Slight edema of the disk is present

hemorrhages developed and that this development was associated with an appreciable decrease in the number of red blood cells This decrease was out of proportion to the degree of the clinical hemorrhagic phe-

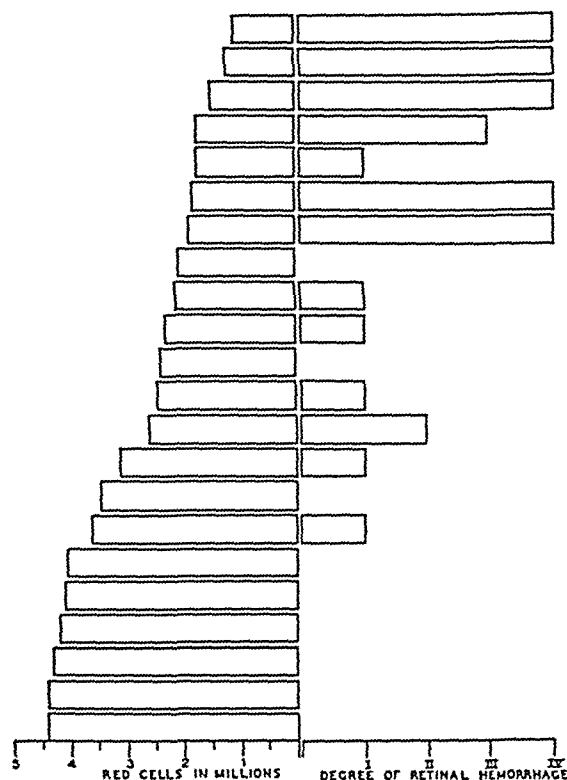


Fig 4—Correlation between anemia and retinal hemorrhage in cases of leukemia. The blocks to the left represent the red blood cells in millions in each of the 22 cases at the time of the first retinal examination. The adjacent blocks represent the amount of retinal hemorrhage. If there is no block adjacent to the red blood cell block, it indicates that there were no retinal hemorrhages. The extensive retinal hemorrhages occurred in the cases of severe anemia, the moderate degree of retinal hemorrhages occurred in the cases of moderate anemia, and no hemorrhages occurred in the cases of mild anemia.

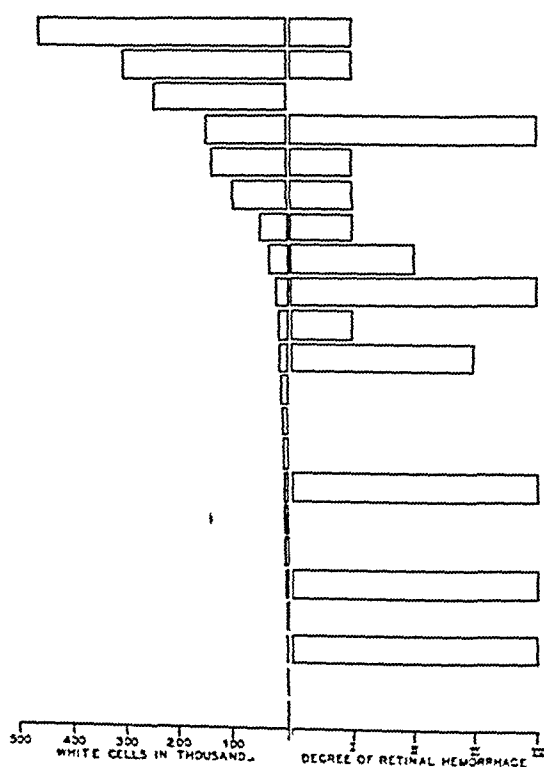


Fig 5—Comparison of the white cell count and retinal hemorrhage in cases of leukemia. The blocks on the left represent the white blood cell count in each of the 22 cases, and the blocks to the right represent the amount of retinal hemorrhage. There is no correlation between the leukocytosis and the amount of retinal hemorrhage.

nomenon In this series of cases, it seemed to make no difference whether the leukemia was of the myelogenous or the lymphatic type There is a similar but less consistent correlation between the hemoglobin and the retinal hemorrhages Interestingly, in the case in which the red cell count was the lowest the ophthalmologic diagnosis was retinitis of anemia instead of leukemia There is no such correlation between the white cell count and the amount of retinal hemorrhage as pointed out by Goldback ² In figure 5 the same cases are arranged in the order of increasing white cell count, and it is apparent that there is no relation between the amount of retinal hemorrhage and the degree of leukocytosis

CLINICAL IMPRESSION

Since there is such a close parallelism between the degree of anemia and the extent of the retinal hemorrhage as well as the clinical condition of patients with leukemia, it seems fair to conclude that evidence has been presented which suggests that therapeutic and investigative procedures could be more advantageously directed toward the factor of anemia instead of toward the leukocytosis in the unsolved problem of leukemia

DISCUSSION

DR FRANCIS HEED ADLER I should like to ask Dr Gibson if he has any idea what causes the white spots in the center of the hemorrhages in cases of leukemia Textbooks usually say that they are due to the presence of accumulated white blood cells, but I have noticed them in cases of leukemia when the white cell count was not materially elevated

DR GLEN G GIBSON In observing these cases one gets the impression that the white centers are due to a collection of serum and hemorrhagic debris rather than to a nest of white blood cells surrounded by red blood cells I regret that I have no microscopic confirmation of this impression

BILATERAL CONGENITAL ECTOPIA LENTIS WITH ARACHNODACTYLY (MARFAN'S SYNDROME)

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The condition which bears Marfan's name is characterized by the following symptoms lack of subcutaneous fat, poorly developed musculature, elongated extremities and curvature of the spine. In approximately half of the cases the following conditions are also found congenital cardiac disease, congenitally dislocated lenses, miosis and high myopia. The condition occurs with equal frequency in both sexes and usually is not recognized until the third or the fourth year, often it is not diagnosed until adult life has been reached. It is a hereditary condition which can be transmitted by males and females alike and is considered by most authorities to be transmitted by changes in a varying number of chromosomes. If only chromosomes concerned with mesodermal development are involved, the ocular symptoms are absent (congenital mesodermal dystrophy), if chromosomes concerned with ectodermal development are also affected, the suspensory ligament of the lens and sometimes the dilator muscle of the iris are involved, resulting in dislocation of the lens and a rigid, contracted pupil (congenital ectodermal dystrophy).

The mesodermal changes are characteristic. The patient is tall, due to the long legs, the hands and feet are extremely long and thin, the body and face are thin, due to the lack of subcutaneous fat, the fingers are delicate and long ("spider digits"—arachnodactyly), there is scoliosis or kyphosis or both, the skull and teeth are long and narrow, and the bony palate is narrow with a high arch.

TERMINOLOGY

Some of the mesodermal changes were first described by Marfan¹ in 1896, and the name of arachnodactyly was suggested by Achard² in 1902. Since then variations in the syndrome, including the cardiac and ocular changes, have been observed. In 1924 Ormond and Wil-

1 Marfan, A. B. Un cas de deformation congenitale des quatre membres plus prononcee aux extremités caracterisee par l'allongement des os avec un certain degre d'amincissement, *Bull et mem Soc med d hop de Paris* **13** 220, 1896.

2 Achard C. Arachnodactylie, *Bull et mem Soc méd d hôp de Paris* **19** 834, 1902.

hams³ first described the ocular changes which occur in about half the cases of this syndrome. The only American ophthalmologists who have paid any particular attention to this condition are Lloyd⁴ and Burch,⁵ whose articles appeared in 1935, 1936 and 1937. Burch's article is accompanied by a complete chronologic bibliography, which it is unnecessary to repeat here.

Weve⁶ reported on this condition in 1931 and added reports of 23 cases in 6 families from Holland. This is the first article to appear from that country. He placed all the signs on a mesodermal basis. Killmann⁷ reported 7 cases in which the picture was typical and 1 in which it was not quite typical, the latter type he called a *forme fruste*. His experience was the same as that of all other investigators in that he found no evidence of syphilis, alcoholism or endocrine disturbance. Franceschetti,⁸ and Waardenburg⁹ expressed the belief that the changes in the iris and lens are ectodermal in origin, and in 1 case he noted a coloboma of the lens.

Because of the congenital changes in the mesodermal and ectodermal tissues, with resultant dislocation of the lens, perhaps it would be advisable to coin a new name for the condition and call it dysmesectopia. The "dys" is for the dystrophy, the "mes" for mesodermal and the "ecto" for ectodermal, the "ecto" is also a part of the word ectopia (dislocation). If only the mesodermal tissue is involved, the name would be dysmesdactyly, the "dactyly" referring to the condition of the fingers, which is a prominent characteristic, and at the same time part of the term arachnodactyly, which has come into fairly common use, would be retained.

REPORT OF CASES

CASE 1—M. H., a white man, single, aged 50, was first seen in Dr. Fletcher's clinic at the Manhattan Eye, Ear and Throat Hospital in December 1936, at which time he requested an examination for glasses. The vision in the right eye was found to be limited to the ability to count fingers at 5 feet (152 cm), there was no improvement with glasses. The vision in the left eye was 20/200, which was

3 Ormond, A. W., and Williams, R. G. Case of Arachnodactyly with Special Reference to Ocular Symptoms, *Guy's Hosp. Rep.* **74** 385, 1924.

4 Lloyd, R. I. Arachnodactyly, *Arch. Ophth.* **13** 744 (May) 1935, A Second Group of Cases of Arachnodactyly, *ibid.* **17** 66 (Jan) 1937.

5 Burch, F. E. Association of Ectopia Lentis with Arachnodactyly, *Arch. Ophth.* **15** 645 (April) 1936.

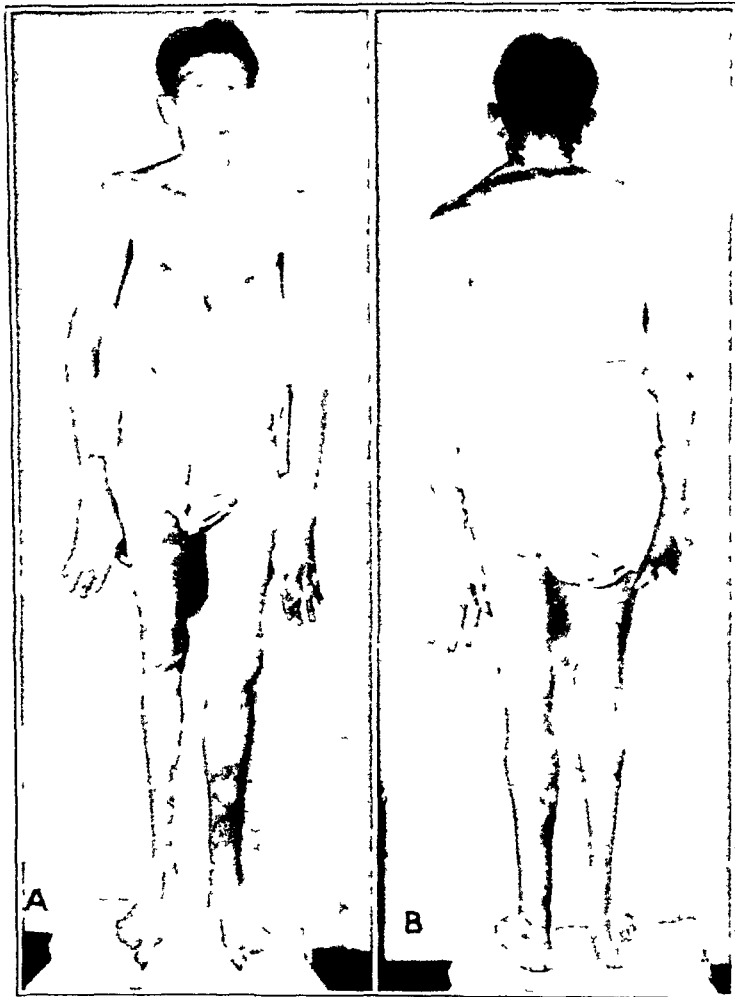
6 Weve, H. Ueber Arachnodactyly (Typus Marfan) Dystrophia Mesodermalis Congenita, *Arch. f. Augenh.* **104** 1, 1931.

7 Killmann. Angeborene doppelseitige Linsenektopie und Arachnodactylie, *Klin. Monatsbl. f. Augenh.* **92** 335, 1934.

8 Franceschetti, A. Marfanscher Symptomenkomplex und Coloboma lentis, *Klin. Monatsbl. f. Augenh.* **88** 686, 1932.

9 Waardenburg, P. J. Das menschliche Auge und seine Erbanlagen, Haag, Nijhoff, 1932.

improved to 20/70 + by a +1.00 D sph \ominus +2.00 D cyl, axis 15. The right eye showed a deep anterior chamber, with the face of the iris somewhat concave, an extremely small pupil and marked iridodonesis. After several drops of homatropine hydrobromide and atropine were administered, only slight dilatation of the pupils was obtained, but it was sufficient to determine that in the right eye the lens was in the lower part of the vitreous and marked choroidal pigment changes were present throughout the fundus. The left eye also had a small pupil, and the face of the iris was concave below and convex above. Moderate iridodonesis was present. A cataractous lens could be seen dislocated up and in, and on movement



Appearance of the patient in case 1

of the head in certain positions the edge of the lens could be moved partially out of the pupillary space. The patient was seen at regular intervals, and one year later the left lens was found entirely displaced from the pupillary space and lying in the vitreous below. The vision now was 20/30 with a +1.00 D sph \ominus +2.00 D cyl, axis 135. The fundus was clearly seen after slight dilation with homatropine hydrobromide. There were marked myopic changes, including a posterior staphyloma and choroidal atrophy.

The typical appearance of the hands and feet and the gaunt appearance due to the lack of subcutaneous fat are shown in *A* of the accompanying illustration. The extreme kyphosis is shown in *B*. In both *A* and *B* it will be noted that the

left lower extremity is more robust and more fully developed. This was due, I believe, to the kyphosis which resulted from excess weight being put on the left lower extremity.

According to the patient, his father and mother had no ocular trouble and wore glasses only for reading. He has two brothers, 1 of whom I examined and found to have normal eyes and no skeletal defects. The other brother is supposed to have normal eyes and has 2 children, one of whom is nearsighted.

CASE 2—D. H., a 10 year old girl, was first brought to Dr. Fletcher's clinic at the age of 6 years because of poor vision. When examined at the first visit she was found to have long hands and feet and looked emaciated. No subcutaneous fat was present, and there was marked lordosis of the spine. Her teeth were long and thin, and her palate was high and quite arched. The lenses were both dislocated, and the irides were tremulous. The pupils were small but were easily dilated with homatropine hydrobromide. The fundi showed no abnormalities. At the time of writing, four years later, both lenses are completely dislocated and lie in the vitreous below. With a +9 D sph vision of approximately 20/50 is obtained for each eye, but it is difficult to examine the child as she is not cooperative. Her father and mother and maternal grandmother have normal eyes and no skeletal defects. The child has no brothers or sisters. A request for a photograph was emphatically refused by the patient.

COMMENT

There has been a good deal of discussion about the condition of the dilator muscle of the iris in cases of dysmesectopia. Some say that the muscle is rigid and others are as certain that it is absent. In 1 of my cases (2) dilatation was easily obtained with homatropine hydrobromide, showing that the dilator muscle was not rigid, and moderate dilatation was obtained with cocaine hydrochloride, pointing to the presence of some dilator muscle fibers. In the other case (1) only a slight increase in the size of the pupil, 2 mm., was obtained with either homatropine hydrobromide or cocaine hydrochloride, but the dilatation was definite. I think that a conservative opinion would be that in some cases of dysmesectopia not only mesodermal structures are affected but ectodermal structures, such as the zonular fibers of the lens or the dilator muscle of the iris or both, may also be involved. This is what Vogt¹⁰ suggested, and many others agree that it seems correct in the light of the present knowledge.

SUMMARY

Two cases of congenital mesodermal and ectodermal dystrophy with anachnodyly and bilateral congenital ectopia lentis are reported, and a résumé of the various signs and symptoms is given. The terminology is discussed, and it is suggested that the term dysmesdactyly be adopted when the chromosomes concerned with mesodermal tissue are involved. When the ectodermal tissue is also affected, the term dysmesectopia is suggested.

10 Vogt, A. Marfan's Syndrome, *Klin Monatsbl f Augenh* 87:258 1931.

NATURE AND MANAGEMENT OF THE HETEROPHORIAS

J T MAXWELL, M D

OMAHA

At birth the eyeballs assume a position controlled by basic innervation which is present before environmental influences have become effective. This innervation is innate and has been called tonic innervation. At birth vision is not fully developed, nor is the relation between the two eyes developed. As growth proceeds, fixation, fusion, coordination, convergence and accommodation develop, become interrelated and assume their various roles in the positioning of the eyes. When the higher centers of the brain are inhibited, as in sleep, deep narcosis or coma, the eyes may assume a position which is irrelevant to that in effect when the nervous system is functioning normally. In fact, it seems from the positions of the eyeballs under the conditions named that the eyes are more or less set adrift to wander with little or no innervational control. With the return to normalcy, a flow of properly distributed innervation to the ocular muscles is resumed. In discourses on psychophysiologic mechanisms, the term position of rest is frequently used. The question arises as to what may be called rest. Shall the term be used when fusion alone is eliminated or when other stimuli which influence the position of the visual axes are rendered inactive?

TONIC INNERVATION

When a dissociation method, such as the screen test or the Maddox rod, is used to test the muscle balance for distant vision (assuming emmetropia), only the tonic innervation is in effect, because fusion is eliminated, and if the eyes are emmetropic, no accommodation is active. Under dissociation, there may be either orthophoria or heterophoria (exophoria, esophoria, hyperphoria or cyclophoria).

Exophoria may be defined as that position of the eyes, relative to the distance at which the test is made, in which the visual axes, under dissociation, do not meet before reaching the object of fixation. Esophoria is that position of the eyes in which the visual axes, under dissociation, meet between the eyes and the object of fixation. Hyperphoria and cyclophoria are likewise positions of the eyeballs controlled by the innate tonic innervation when fusion is made artificially impossible. In other words, heterophorias are examples of improper distribution of

tonic innervation It is the function of the fusion faculty to correct these anomalies by keeping the eyes in a position that will insure single binocular vision The adequacy of this mechanism depends on the degree of heterophoria and the extent of the fusional amplitude Exophoria or esophoria for distance vision of over 1 or 2 prism diopters which is not caused by accommodative convergence would be an example of an anomaly of tonic innervation

When the relation between accommodation and convergence is well developed, esophoria may result from accommodative convergence prompted by the effort of the accommodation to overcome an existing hyperopia The same condition may result from an overcorrection of myopia This would not be an anomaly of tonic innervation An esophoria of this type would disappear under the influence of a correcting sphere The adjustment might not occur at once, however, owing to the possible formation of habit

The detection and measurement of anomalies of tonic innervation are accomplished by dissociation methods at 6 meters In the presence of such an anomaly the power of the fusion faculty must be measured by prism convergence or prism divergence (duction tests) in order to ascertain the adequacy of the correcting force in overcoming the existing phoria

Heterophoria is not in itself necessarily of clinical significance It is the ratio of the correcting power of the fusion faculty to the heterophoria which is the deciding factor If the power of the fusion faculty is low in proportion to the amount of heterophoria present, single binocular vision will either be maintained under stress (eyestrain) or, if the proportional strength of the fusion faculty to the heterophoria is too low to produce single binocular vision, strabismus will result Exophoria of 5 prism diopters for distance vision would in all probability give no trouble if the eyes were able to overcome 10 or more prism diopters base out (adduction) and retain single binocular vision, whereas, if the positive fusional convergence were much less, the exophoria for distance vision might well be expected to produce symptoms It is the relation of the demand (heterophoria) to the available innervational supply indicated by the duction tests which decides the issue

By long persistent orthoptic training it is often possible to reduce a phoria, but the process is not an easy one In fact, if some simple method could be developed whereby a phoria could be readily changed, the greatest problem of ocular muscle imbalance would be solved

In contrast to the relative stability of the distribution of tonic innervation, the power of the fusion faculty may vary greatly, not only with the condition of the general system but as the result of orthoptic training and other influences

FUSIONAL RESERVE

If a heterophoria exists, the fusion faculty should be able to maintain single binocular vision, and in addition there should be a reserve power, measured by prisms. As an analogy, one might say that if a man expected to carry a weight of 50 pounds (23 Kg) for a city block, he should be able to lift a considerably greater load. If the fusion faculty were just barely able to compensate a phoria (no reserve) and produce single binocular vision, it would be unreasonable to suppose that the condition would be comfortable over an extended period of time.

When both eyes fixate an object (single binocular vision) and prisms, base in or base out, are interposed, the images begin to move off the fovea, and a reflex is set up which will, within certain limitations, replace the images on the fovea and maintain fusion. The important aspect of the situation is the limitations. When prisms are placed base out before the eyes, they will, in order to retain single vision, converge to a certain extent only, beyond which limit the addition of further prism power will produce either diplopia or suppression of vision in one eye. The same is true of divergence produced by base in prisms, except that the out-turning of the eyes is usually less in amount than the inturning.

It can easily be shown that the angular distance which the eyes may be turned by prism power while maintaining fusion is not a muscular limitation, because as soon as fusion is lost either eye will turn farther. It is evidently the association factor that imposes the limitations. The degree to which the fusion faculty is developed is apparently the controlling factor.

A strain is produced under prism action in proportion to the strength of the prisms used. Under such conditions a load is thrown on the fusion faculty in an effort to retain single binocular vision. Thus, by the use of prisms, the power of the fusion faculty is measured. That amount of prism power which can be overcome, in excess of that which is normally in use, has been called the "fusional reserve." Comfortable vision depends largely on the relation of the amount of fusion power in use to that in reserve. Experience has shown that eyestrain is likely to occur if over one half of the total fusion power is in use. Such a rule will have many exceptions and be subject to individual variation, nevertheless, it is a basis on which to work.

When, by orthoptic training, an attempt is made to produce a comfortable condition by building up the fusion faculty to compensate for a high degree of heterophoria, an acquired reflex is being artificially increased to compensate for an improper (innate) tonic innervation. It usually happens that even though the fusion faculty has been increased by training so that a heterophoria may be carried in comfort, the tonic innervation (heterophoria) will remain unchanged. I wish to emphasize particularly the fact that tonic innervation is stable and difficult to change.

INNERVATION AT THE READING DISTANCE

At the reading distance there must be considered not only the tonic innervation and the fusion faculty which are present for distance but the influence of accommodation and the proximity of the object of regard. In order to see near objects clearly and singly, the visual axes must converge, and accommodation must be in effect. Therefore, accommodation and convergence become associated, although either may act without the other. In the presence of uncorrected hyperopia, accommodation must be exerted for distant vision, and it can easily be shown with the dissociation tests that convergence is induced by this accommodation. Therefore, esophoria is likely to be present for distance vision in cases of uncorrected hyperopia. When the necessary plus lenses are added, accommodative esophoria will disappear. At the reading distance accommodative convergence is usually present. The difference between the exophoria for near vision and the total amount of convergence necessary to maintain single binocular vision for near objects indicates the amount of accommodative convergence which is in effect under dissociation. In persons with myopia, who accommodate little, if any, the exophoria for near vision will usually be high, because accommodation and convergence have not become as closely associated in such persons. Experience has shown that accommodative convergence for near vision in perfectly normal subjects may vary from 16 prism diopters (the total amount of convergence at 38 cm) to none at all. It is therefore evident that accommodative convergence (calculated by the exophoria for near vision) is not necessarily an important factor of comfort for near vision but is simply indicative of the established relation between accommodation and convergence.

Convergence may be observed when an object approaches the eyes and may also be demonstrated by covering one eye with a card while a test object is brought near the other eye. The eye behind the screen, in most instances, will turn inward. There must be a stimulus to prompt a response. The question arises as to the sources of innervation which produce convergence at the reading distance. In order to differentiate these reflexes, they must be dissociated as in the distance tests. Fusion may be prevented by means of the Maddox rod, or the double prism may be used, or a single vertical displacing prism may be placed over one eye.

In this demonstration, the test object, such as a capital E 3 or 4 mm high in the center of a card about 5 inches square, is placed on the arm of the phorometer at a distance of 38 cm from the eyes of the subject. A 6 prism diopter base down over one eye will usually be sufficient to produce diplopia and cause one image to appear on a higher level than the other. If the two images are vertically aligned, it is evident that

the same amount of convergence is in effect when fusion is eliminated as when single binocular vision is present. However, the images will not often be exactly one above the other. With the base down prism over the right eye, the upper image will most often appear to the left (less frequently to the right) of the lower image, thus indicating that the visual axes have, under dissociation, failed to converge the full 16 prism diopters. The amount of exophoria present for near vision is measured by the prism power base in which is necessary to align the images vertically. The difference between the total amount of required convergence (16 prism diopters) and the exophoria for near vision (assuming orthophoria for distant vision) must be attributed to the influence of accommodation and the proximity of the object. For example, if there is an exophoria for near vision of 6 prism diopters and a total convergence of 16 prism diopters, there will remain 10 prism diopters of convergence because of the accommodation and the proximity of the object.

For the purpose of further analyzing convergence under dissociation at the reading distance, a pair of plus 2.75 D spheres are added to relax accommodation. Any convergence present under these conditions cannot be due to either fusion or accommodation, since each eye sees a separate image from the effect of the prism (dissociation) and the spheres eliminate accommodation. It is reasonable to assume, therefore, that it is the psychic effect of nearness that induces the remaining convergence. For example, if after the plus spheres are added the images separate to such an extent that the exophoria for near vision is increased to 12 prism diopters, there is still left 4 prism diopters (16 prism diopters — 12 prism diopters) of convergence, which is attributed to the psychic effect of nearness of the object (proximal convergence).

With fusion eliminated, the visual axes converge only 10 prism diopters. When accommodation is inhibited by the plus spheres, 6 prism diopters more of convergence disappear, leaving the final 4 prism diopters to be attributed to proximity. Thus, it is seen that the factors which may produce convergence for near vision can be divided into fusional, accommodative and proximal. In practice, the proximal convergence and the accommodative convergence may be treated as one. They will be so considered in the following discussions.

At birth there is no relation present between accommodation and convergence. The accommodative convergence reflex is the result of a learning process. However, the degree of the development has little, if any, bearing on ocular efficiency or comfort. Proof of this premise is found in everyday practice, in which the ophthalmologist constantly encounters persons with low accommodative convergence who are perfectly comfortable. This reflex is simply the result of an association that has been present from infancy, when accommodation was necessary

to enable the person to see clearly and convergence became necessary to enable him to see singly. This being the case, it is not advisable to use the exophoria for near vision from which to compute convergence efficiency at the reading distance (38 cm), since the exophoria for near vision merely determines the amount of accommodative convergence.

Most difficulties from ocular muscle imbalance arise from deficient positive fusional reserves at the reading distance. When the condition of balance at the reading distance is considered, it must be remembered that 16 prism diopters of positive convergence (orthophoria at distance) is necessary for single binocular vision at 38 cm. The eyes should be able to converge at least double the amount needed for fixation. Therefore, if the prism convergence (adduction) for near vision is less than 16 prism diopters, treatment may be needed.

MANAGEMENT OF THE HETEROPHORIAS

There are many persons with ocular muscle imbalance for whom orthoptic training is the only treatment indicated, although it is frequently advisable to use both training and prisms. The use of prisms without orthoptic training is indicated for the correction of hyperphoria and for lateral imbalances on occasions when exercises are impracticable.

For deficiencies of the fusional power in lateral imbalance, orthoptic training is direct in its application, since a weak fusional power is strengthened by training. In the tonic forms of ocular imbalance (phorias), the treatment is indirect in that strength (fusion) is built up in one faculty as compensation for a faulty distribution of another faculty (tonic innervation). By this means a comfortable condition may be produced, but the tests in most instances will show that the exophoria or the esophoria still remains, indicating that the tonic innervation is unchanged.

The methods available for the treatment of ocular muscle imbalance when single binocular vision exists consist of orthoptic training, the prescription of prisms for constant or temporary wear or a combination of both types of treatment. The indications for treatment are the same, whether orthoptic training or prisms are to be used.

Prisms incorporated in spectacle lenses are prescribed for the correction of heterophoria when for any reason orthoptic training cannot be given or when both methods of treatment are used. When the two are used together, the prisms are prescribed for immediate relief with the hope that they may eventually be discarded when orthoptic training has been successfully used.

When prisms are placed base in (fusion present), the eyes assume a position whereby the images are placed on the maculas with the visual

axes relatively divergent in respect to the fixation point, that is to say, base in prisms lessen the amount of convergence. On the other hand, base out prisms produce the opposite effect, so that the amount of convergence necessary for single binocular vision is increased above that normally required. It is apparent that the application of prisms is not curative in effect, thus, prisms are similar to crutches in this respect, but they are often necessary.

Prisms are prescribed chiefly for insufficiency of convergence and for vertical imbalance. A person who is orthophoric for distance vision must converge approximately 16 prism diopters in order to attain single binocular vision at 38 cm (15 inches). Unless a reserve of converging power equal to at least the amount in use can be overcome without producing diplopia, it may be assumed that an existing eyestrain not otherwise accounted for is the result of insufficient positive fusional amplitude. If exophoria for distance vision is present, more than 16 prism diopters of convergence is necessary to fixate at 38 cm, if esophoria for distance vision is present, the amount of convergence in force to maintain single binocular vision for near objects would be proportionately less.

The question immediately arises as to how much prism power shall be prescribed (orthoptic training not available) to produce comfortable use of the eyes for near work. In attempting to establish a schedule, one is confronted with the difficulty which always arises when physiologic processes are reduced to mathematical formulas. The following procedure is suggested.

If single binocular vision is barely maintained at 38 cm, that is, if diplopia appears as soon as a small amount of base out prism power is placed before the eyes, the maximum amount of base in prism to be prescribed for the first trial lens should not exceed 2 prism diopters for each eye. When some fusional amplitude is present but not equal to double the amount required for single binocular vision at the given distance, the strength of prism power will vary in proportion to the deficiency. For example, if the patient is orthophoric for distance vision and can overcome only 8 prism diopters base out at 38 cm, he might be said to have a deficiency of 8 prism diopters, since he should have a minimum of 16 prism diopters of reserve converging power but has only 8 prism diopters. If one assumes that a total of 4 prism diopters is the maximum that can be worn with comfort, a total of 2 prism diopters would be prescribed, 1 prism diopter base in before each eye. For slight deficiencies, the required effect can often be attained by decentration of the lenses without the necessity of grinding the prisms.

Attention must again be called to the fact that no rule can fit all cases and that any set rule is definitely empiric. If the first attempt at relieving the symptoms does not prove satisfactory, a modification of

the prescription must be made according to the indications and the judgment of the refractionist. The discomfort produced by strong prisms is no doubt in part due to prism aberration.

A prominent group of persons who require prismatic help are those with presbyopia who have insufficient converging power for near vision. For such persons prisms may be incorporated in the near correction, either as glasses for reading only or in the segments of bifocal lenses. It is a good plan to select an occasion when the patient has time to read in the reception room for an hour or two with trial lenses in order to determine the effect of the intended prescription. The person without presbyopia who is emmetropic and has insufficient convergence is confronted with the necessity of using prisms only for near work if he refuses orthoptic training.

The prescribing of prisms for the correction of esophoria is of doubtful value. This is especially true of high esophoria for near vision, which is usually of systemic origin. Yet many persons with esophoria are often uncomfortable, and base out prisms are in some instances gratefully accepted. Base out prisms should be prescribed with the patient's full knowledge that they are not of certain value, so that too much will not be expected.

Prisms in spectacles are used extensively for the relief of hyperphoria, since orthoptic training for vertical imbalance seems to be of little value. The amount of prism power prescribed should always be less than the amount of the phoria and depends on the prism power which the fusion faculty is able to overcome in the opposite direction (infraduction). It is suggested as a first trial that a prism be prescribed equal in power to one half of the demonstrable phoria, if the infraduction is less than the hyperphoria. If the vertical duction is equal to, or greater than, the phoria, proportionately less prism power should be prescribed. Errors of less than 1 prism diopter of hyperphoria are seldom considered of sufficient importance to require correction. Hyperphorias of high degree, especially when they are of sudden onset, are usually of toxic origin, and, on account of their fluctuating character, respond uncertainly to correction with prisms.

Many practitioners feel that the prescribing of prisms for constant use is questionable and fraught with the possible danger of increasing the error. With this thought in mind, consideration will be given to just what prisms in spectacle lenses accomplish. It is evident that prisms allow single binocular vision with the visual axes intersecting either nearer or beyond the object of regard. For example, if base in prisms are prescribed, less positive convergence is necessary than would otherwise be the case. If the fusional convergence is later required to do full duty by the removal of the prisms, symptoms of fatigue might arise. In other words, the patient would feel the effort more than before relief

had been afforded. In this respect the condition might be said to be made worse, at least the subject might so contend. On the other hand, the basic situation, that is, the exophoria, would in all probability not be altered, and from that standpoint the condition would not be increased unless the distribution of tonic innervation (phoria) had changed.

MEASUREMENT OF CONVERGENCE EFFICIENCY

In conclusion, I wish to call particular attention to the fact that convergence efficiency for near work must be determined by prism convergence with the test card placed at the reading distance. This procedure has a decided advantage over the method which consists of causing a test object to approach the nose while the point at which diplopia occurs or when one eye deviates is noted. In the "approach-the-nose" method, factors are introduced which are not in effect with fixation at from 38 to 40 cm.

In the first place, excessive accommodation is required, which may act as a further stimulus for convergence. In the second place, when the near point of accommodation has somewhat receded, as in cases of presbyopia, the resulting blurred image inhibits fusion and produces diplopia sooner than would otherwise be the case. This factor varies with the age of the patient. In the third place, the image on the retina becomes larger as the distance between the object and the nodal point of the eye shortens, introducing another factor not present in the use of the eyes at the ordinary reading distance. It is also possible that the increased proximity of the object may act as an additional stimulus to convergence. Objection to the near point method of measuring convergence lies in the fact that it is difficult to analyze and is not readily adaptable to a scale of measurement which can be associated with the converging power for distance vision or with the procedure for treatment. It seems that no correlation has been established between the near point of convergence for different ages and the necessary prism power used as a corrective measure. Above all, it is common to find persons having difficulty in reading, despite the fact that the near point of convergence is quite close to the eyes, yet when convergence is induced at from 38 to 40 cm by means of base out prisms, it is found deficient. The last objection abrogates this test and relegates it to the discard as far as determining convergence efficiency is concerned.

OPTOCHIASMIC ARACHNOIDITIS

IMPORTANCE OF A MIXED TYPE OF ATROPHY OF THE OPTIC NERVE AS A DIAGNOSTIC SIGN

DERRICK VAIL, M D

CINCINNATI

In September 1926 a white man 25 years of age presented himself with a history of failing vision of six months' duration. He had suffered an occasional severe frontal headache ever since he had received an injury to the back of his head from a fall seven years prior to admission to the hospital. Roentgenograms of the skull showed no lesion, an examination of the sinuses revealed no pathologic process. The Wassermann reactions of the blood and spinal fluid were negative. Both optic nerve heads showed a mixed type of atrophy (visible lamina, sharply defined margin, and markedly reduced vessels, especially arteries). The fields of vision suggested a suprasellar lesion, the history of injury, a chronic serous meningitis or arachnoiditis involving the chiasm. The patient gave his consent for an exploratory craniotomy, which was performed on March 7, 1927, by Dr. George J. Heuer. The pia-arachnoid over the whole area of the brain exposed was much thickened and grayish white. Beneath it was a large amount of cerebrospinal fluid, which escaped in quantities when the tough pia-arachnoid was torn with a needle. Further investigation revealed a mass of adhesions about the anterior part of the chiasm, continuing forward around the optic nerve. The cisterna chiasmatis was distended with fluid, and its wall was thickened and opaque. The cisterna was broken into between the tuberculum sellae and the chiasm, and a large amount of clear cerebrospinal fluid escaped. As much of the wall of the cistern as possible was snipped away with scissors. With a blunt dissector, the adhesions around the optic nerves were separated so as to free the nerves as much as possible. They appeared attenuated and grayish. After these manipulations were completed, it was possible to elevate the chiasm, so that one could look into the sella turcica and see the diaphragm sellae. Nothing suggestive of a tumor was seen about the chiasm. The patient had a smooth convalescence. A month later the central scotomas and enlarged blindspots were smaller, and the headaches had disappeared. The vision progressively improved from ability to count fingers at 5 feet (152 cm) with each eye prior to the operation to 20/30 + in each eye one and one-half years later. A recent letter stated that the patient's vision has remained good and his health excellent. (This case was reported by Heuer and myself in 1931.)

This case is considered in detail because it was the first of its kind encountered by me and especially because most of the characteristics were present which have since been established as a disease entity. Chronic arachnoiditis, or arachnitis affecting the brain and spinal cord, is well known. The generalized form was first described by Quincke in 1893 and the localized form by Schlesinger in 1898. Since this time comprehensive reviews of the whole subject have been published by

Read before the Washington, D. C., Ophthalmological Society, April 18, 1938

many authors, more recently by Craig, Spurling, Horrax and Stookey. The most important contributions on this subject, however, have recently been published by French authors, notably the monograph by Bollack, David and Puech. Because this paper is limited to a discussion of arachnoiditis producing the chiasmic syndrome, it is not pertinent to consider arachnoiditis affecting other parts of the central nervous system, except to mention the etiologic and pathologic factors which are probably common to all types of this disorder.

The cause of chronic arachnoiditis is not definitely established. Heuer expressed the belief that when an etiologic factor is present, it falls into one of two groups: an antecedent trauma (fracture of the skull or spine) or an infection. The infections are of two kinds: an infection of the meninges or brain (encephalitis, meningoencephalitis or leptomeningoencephalitis) or an infection of an area adjacent to the brain (the middle ear, the mastoid or the various sinuses). Most writers on the subject have noted the same etiologic factors. The pathologic process has been described by many authors who are in general agreement regarding the process and *modus operandi*. A thickened arachnoid is connected to the adjacent nerve tissue by fibrous bands, which vary in size from small discrete lines of adhesions to dense processes extending over a large area. The adhesions may form pockets and block the free flow of spinal fluid, producing the so-called arachnoid cysts. Localized symptoms develop, depending on the location of these bands and cysts, and produce what the older writers called pseudotumors.

The pathologic process affecting the chiasm is similar to that affecting the arachnoid. The clinical signs point to a restricted lesion in the chiasmic region. However, it has been observed by all neurosurgeons that the entire adjacent structure of the brain is involved by a more or less generalized process. It is therefore probable that if it were not for the accumulation of spinal fluid around the chiasma and the pressure symptoms produced thereby, the disease might remain silent and unrecognized. If this is true, it is logical to believe that there are many cases of chronic arachnoiditis in which the disease has never led to symptoms or distress. In the article by Heuer and myself this point was commented on as follows:

It has been our experience generally that chronic arachnoiditis is a fairly widespread disease regardless of the fact that the local symptoms produced by it may be referred to the cerebellum, the motor cortex, the chiasmal region or a certain level of the spinal cord, in other words, localized arachnoiditis is, in our experience, identified by symptomatology rather than pathology.

In 1929 Manuel Balado, of Buenos Aires, reported the case of a man aged 35 who complained of progressive visual failure of six months' duration. At the time the patient was seen his vision was reduced to

perception of shadows and light. Both disks showed primary atrophy. The optic chiasm was exposed, and an area of circumscribed inflammation of the arachnoid was found surrounding it. A piece of the arachnoid was removed, and histologic study showed some proliferation of endothelial cells and small calcareous nodules. During the months after the operation the vision steadily improved. This is the first case of chiasmic arachnoiditis on record, and Alice has proposed, with some justice, I believe, that the chiasmic syndrome produced by this disease be given Balado's name. However, Cushing and Eisenhardt, in discussing meningiomas arising from the tuberculum sellae, apparently recognized and reported this disease entity nine months before Balado. They said:

Though this chronic cisternal arachnoiditis, apart from a postmortem examination, must always be a questionable diagnosis, cases nevertheless are encountered in which, due probably to a previous inflammation, a local accumulation of fluid trapped in a thickened arachnoid membrane may give symptoms of tumor. In a few cases, however, in which a suprasellar lesion was suspected on the grounds of the syndrome under discussion, nothing was found but a local collection of slightly xanthochromic fluid, the evacuation of which led to a subsidence of symptoms.

It is unfortunate that reports of these few cases were not collected and published under a proper isolated title, since then there would be no question of priority. It has been my good fortune and privilege to observe and study 8 patients with arachnoiditis affecting the optic chiasm. Seven of them were operated on, and the diagnosis was verified at the time of the operation and by the subsequent progress of the patient. The eighth patient was not operated on, but his progress, and especially the changes in the visual fields, justify me, I believe, in including this case in my series. The visual fields first showed bilateral central scotoma, then bitemporal contraction, with progressive loss of the visual field practically to zero, followed by recovery in the reverse order. The patient was treated with injections of a foreign protein (typhoid vaccine intravenously), large doses of sodium salicylate (from 100 to 150 grains [6.5 to 9.7 Gm.] per rectum) and frequent spinal tapping. Later, 15 grains (0.97 Gm.) of sodium iodide was given intravenously daily for a course of fifteen injections.

A study of my cases and of those reported in the available literature (a total of 134 in which the diagnosis was verified at operation) has allowed me to draw certain conclusions. The condition is a disease of young adults, over 80 per cent of the cases occurring in persons under the age of 40. Seven cases in which the patients were under 10 have been reported. The condition predominates slightly in the male. The vision in one eye may be affected long before the second eye is involved, in 1 case there was an interval of ten years (Vincent). Once the disease

begins, however, the loss of vision is likely to be rapid. Occasionally slight remissions are observed. The earliest symptoms are most frequently those of acute retrobulbar optic neuritis, and there is almost always a central or cecocentral scotoma found in the field of vision. Bitemporal, binasal or homonymous hemianopia are frequently associated with the scotoma. Several cases of a basofrontal syndrome have been reported, with papilledema on one side and atrophy of the optic nerve secondary to papilledema on the other. Several combinations of signs may occur (Craig-Lillie) (1) unilateral central scotoma with normal fundi, (2) unilateral central scotoma and pallor of the disk but normal conditions in the other eye, (3) bilateral central scotoma with pallor of both disks, (4) bilateral central scotoma with choked disks, (5) unilateral amaurosis with simple atrophy of the optic nerve and choked disk in the opposite eye, sometimes associated with a central scotoma, (6) central scotoma and various alterations of the peripheral fields due to secondary contraction resulting from choked disks, and (7) bilateral amaurosis with any of the foregoing changes in the fundus.

It is my personal experience and belief that the appearance of atrophy of the disk which lies between the primary and the secondary type of atrophy is highly suggestive of arachnoiditis involving the chiasm. It would be unwise to be bold enough to assert that this is a pathognomonic sign, yet one is struck by the frequency of cases in which the outline of the disk is sharply defined, the lamina cribrosa is visible, but the caliber of the vessels is markedly reduced. I have observed this sign even when there has been a preceding papilledema of 2 diopters with hemorrhages of the retina. On the other hand, the case reports in the literature, while not complete in regard to this point, are highly suggestive that many of the patients showed the mixed type of atrophy of the optic nerve characterized by a blurred margin of the disk, normal-sized vessels and the presence or absence of the lamina cribrosa associated with either complete or sector atrophy of the nerve head.

Puech and Mahoudeau considered that the early and rapid simultaneous papillary edema and pallor of the nerve head coinciding with an important lowering of the visual acuity are characteristic of optochiasmic arachnoiditis. In other words, a study of the cases reported in the literature, associated with the clinical findings in my own experience, suggests that the involvement of the optic nerve is a combination of papilledema and simple atrophy. Either one or the other may predominate, and the resultant atrophy will take on a mixed character.

An analysis of the 129 cases studied by Bollack, David and Puech brings out the following facts regarding the nerve head: papillary atrophy with a sharp outline, of the primary type, 38 per cent of the cases, atrophy with a blurred outline, 15 per cent, papillary stasis, 10 per cent, partial atrophy limited to the temporal segment, 7 per cent,

simple hyperemia, 7 per cent, partial horizontal atrophy, 4 per cent, normal disks, 10 per cent, and disks difficult of interpretation, 8 per cent. However, the so-called primary type of atrophy of the optic nerve reported in 38 per cent of their cases was apparently not described in a thorough fashion, which would include a description of the outline, the elevation and the color of the disk, the visibility of the lamina cribrosa and the size and shape of the blood vessels. A history of previous inflammatory illnesses is found in many cases. These include syphilis, sinusitis, infected tonsils, abscessed teeth, whooping cough, encephalitis and especially influenza. In only a few of the analyzed cases was there a definite history of trauma and in none of these was the injury presumably fracture of the skull. One is inclined to think, therefore, that trauma plays little or no part in producing this particular syndrome. Severe headache but rarely vomiting accompanies visual loss. Nystagmus has been observed in a few cases. Polyuria and polydipsia are occasionally complained of, indicating irritation if not actual inflammation of the hypothalamic area. Studies of the visual fields are of paramount importance, not only prior to surgical intervention but also afterward, since it frequently happens that recovery of the field of vision progresses by the same stages as the loss, but in reverse order. There is, however, nothing typical of the defects in the visual field to help much in diagnosing this condition. Many varieties of defects in the peripheral visual field are described, but a central scotoma associated with a hemianopic defect of the peripheral visual field, especially if bitemporal, is most helpful and suggestive. The central scotoma precedes the defect in the peripheral field as a rule. According to Rollet, the characteristic alterations of the visual field in cases of arachnoiditis are as follows:

marked disturbance of the superiolateral quadrant, less marked in the superior internal quadrant, relative integrity of the externoinferior quadrant. If the lesions are very accentuated, an inferiointernal island in the form of a crescent or L-shaped sector embracing the point of fixation in its concavity may persist. In tumors, it is frequently the superior internal quadrant which is the last to be preserved. These alterations are explained by the predominance of lesions on the posterior and inferior surface of the chiasma, these areas being the most exposed to meningeal infections.

A frequent observation is the remission of visual loss in the course of the disease. The ocular symptoms improve momentarily, and one finds spontaneous amelioration of visual acuity, but at the end of some time there is a new attack and loss of vision. This, of course, is a frequent finding in cases of multiple sclerosis.

In most cases the cerebrospinal fluid is found to be normal. In only a few has there been increased pressure. Likewise, one rarely encounters general neurologic signs. Roentgenograms of the region of the

sella turcica show no destruction. In a few cases calcified areas were observed, leading to an erroneous diagnosis of a tumor of Rathke's pouch. Ventriculography is of no value in diagnosis.

The differential diagnosis offers many difficulties. Cushing classified the ocular syndromes produced at or near the optic chiasm as follows:

1. Meningiomas, with a parasellar rather than a suprasellar point of origin. These generally occur in persons who complain only of impairment of vision, the visual fields become bitemporally constricted.
2. Adenomas of the pituitary gland. Roentgenograms showing enlargement of the sella turcica and signs of hypophysial dysfunction are the basis for diagnosis.
3. Congenital tumors arising from the cranio-pharyngeal pouch. These usually manifest themselves early in life, and roentgenograms reveal shadows caused by deposits of calcium in the wall of the cyst.
4. Gliomas arising from the chiasm or the third ventricle. These are relatively rare and often have an associated anterior pouching of the sella turcica, as shown roentgenographically.
5. Syphilitic meningitis, as revealed by a Wassermann test of the spinal fluid.
6. Aneurysms situated around the chiasm. These seldom have an audible bruit. The roentgenographic picture is often pathognomonic.

Since the earliest sign in all cases is retrobulbar neuritis, all causes of this condition must be considered in arriving at the diagnosis. The difficulties are enormous in the present state of knowledge, and several hours could be consumed in discussing retrobulbar optic neuritis from every angle.

Retrobulbar neuritis has been defined by Fraclik as a disease of the visual pathways characterized by a selective affinity for the papillo-macular fibers. It is a clinical entity irrespective of the ophthalmoscopic appearance of the optic disk, which may vary from normal to an extreme degree of swelling.

There may be gradual or rapid loss of central vision over a variable period of time, followed in most instances by a definite improvement of variable amount, regardless of the actual cause or the therapeutic measures used. The loss of vision may be partial or relative, complete or absolute, unilateral or bilateral and transitory several times a day. Headache or dull pain in the orbit is elicited on pressing or moving the eyes. This is not a common symptom and usually has to be elicited on interrogation. The light sense, particularly the perception of the light difference, is reduced. There is difficulty in seeing small red or green colored objects, and the vision is usually better when it is bilateral and is tested in a subdued light. I shall leave out of the discussion of retrobulbar optic neuritis types of purely exogenous toxic origin, such as those due to tobacco, alcohol, lead, carbon bisulfide, thallium acetate, fusel oil, etc., and likewise those due to endogenous poisons, such as are associated with diabetes, beriberi, cutaneous burns, internal hemor-

rhage and pregnancy Also there can be excluded, I think, those cases associated with Leber's disease, vascular sclerosis (aneurysm), excessive ingestion of quinine and cranial polyneuritis These etiologic factors are fairly well established or can be definitely determined without much difficulty

I should like to confine myself, then, to a few remarks concerning multiple sclerosis, other demyelinating diseases and sinusitis or focal infections, of which sinusitis is apparently the most important, as factors in producing retrobulbar optic neuritis I believe, furthermore, that one may be justified in formulating a hypothesis which will connect multiple sclerosis, sinusitis and chiasmic arachnoiditis

The controversy which has raged for many years, ever since the publication in 1817 of Beer's article "On Vicarious Blindness from Suppressed Snuffles, Without Evident Accumulation of Mucus in the Frontal Sinus" to the discussion of Campbell's recent article on "Relationship of Sinusitis to Optic and Retrobulbar Optic Neuritis," has shown a swinging of the pendulum back and forth between the two conditions as etiologic factors for retrobulbar optic neuritis Campbell's article bears careful attention by those who are interested in the subject Recent studies by Frost indicate an important relation between disease of the posterior sinuses and papilledema Other authors have reported acute blindness due to suppurative sinusitis Onodi in 1908, Loeb in 1909 and Schaeffer in 1920 pointed out the close anatomic relation between the optic nerves and the posterior sinuses The theory that the optic nerve is affected by direct extension through a thin pathologic bone from the sinus is probably not the entire story Batson's recent anatomic studies appear to throw further light on the subject In a review of the venous connections between the eye and the sinuses he stated that

the many direct connections of the veins of the nose and paranasal sinuses with the veins of the orbit and cavernous sinuses of the cranium seem to present the probable pathway for the involvement of the orbit in infectious conditions of the nose

Eagleton (1932) emphasized the close connection between otitis and infected vessels of the sphenoid sinus and the relation of otitis to meningitis as a result of showers of infected emboli Campbell, in speaking of the status of the sinuses as a causative agent, stated

This condition appears still to be in the stage where individual opinions and theories prevail, and a great deal of real clinical and probably experimental research will be necessary to place the status of the sinuses in disturbances of the optic nerve on a foundation acceptable to all

The question of the causation and pathogenesis of multiple sclerosis is as yet unsolved Nowadays many neurologists think that all demye-

inating diseases (acute disseminated encephalomyelitis, acute ascending myelitis, neuromyelitis optica and diffuse periaxial encephalitis or Schilder's disease) are but varieties of multiple sclerosis. Certainly retrobulbar optic neuritis is a common, if not the most common and early, symptom in these diseases. Various authors have estimated that retrobulbar optic neuritis is the first manifestation of multiple sclerosis in from 11 to 33 per cent of cases. The European authors fix it at 60 per cent of cases.

A recent study by Tracy Putnam, of Boston, is, I think, of much value in arriving at the pathogenesis of multiple sclerosis. His experiments show that venous obstruction leads to axon degeneration and glial proliferation distributed in patches throughout the white matter of nerve tissue. These patches of focal disturbance always occur in encephalitis, in the acute stage of postvaccinal encephalitis and in rabies. He also produced venous thrombi by allergic reactions and suggested that allergy may be a factor in the disease. Curiously enough, H. H. Stark in 1921 advanced the theory that anaphylaxis accounts for the involvement of the nerve from a focus of infection. He expressed the belief that there may be a sensitization of the tissues of both the sinus and the orbit by bacterial proteins, an allergy being produced which results in a localized anaphylactic reaction each time the person comes in contact with a fresh infection of the same bacteria in the nose, teeth or tonsils or some other part of the body. Ruedemann has under observation 2 persons who are sensitive to certain foods, in whom optic neuritis develops on ingestion of the allergin. Bedell has mentioned a case of petechial retinal hemorrhage in an allergic person. Therefore, I agree with Campbell that "the allergic state as a possible etiologic basis in some of these cases should not be dismissed without further thought and investigation."

But to return to Putnam's work. After many studies and experiments, he concluded that the patches of demyelination and glial proliferation seen in cases of multiple sclerosis are due to venous thrombi, which he has seen not only in the white matter but in other tissues of the body as well. He does not venture to guess why the thrombi are formed but limits himself strictly to pathogenesis.

In several cases of optochiasmic arachnoiditis, purulent sphenoiditis has been found. In 1 case reported recently by Worms, pus had broken through the sphenoid sinus and involved the chiasm. This occurred five years after the attack of bilateral optic neuritis and blindness. Autopsy revealed the two optic nerves and the chiasm encircled with a mass of filamentary adhesions fixed to the base of the brain. This case is thus tied up with the cases of arachnoiditis. Operation was performed in 2 cases of what was considered optochiasmic arachnoiditis.

In 1 case (Cossa) the condition turned out to be encephalitis and in the other (Vail), multiple sclerosis. This last case deserves a more detailed report.

A white married woman of 27 was first seen on June 29, 1934, complaining of blurred vision, pain and tenderness of the left eye of two days' duration. Five years before a similar attack occurred in the right eye, which, in spite of an operation on the sphenoid sinus, at which nothing apparently was found, became entirely blind (no perception of light). Examination revealed atrophy of the optic nerve of the mixed type on the right and a normal disk on the left. The vision of the left eye was reduced to 20/40 and the visual field showed a small relative central scotoma. Since the possibility of early multiple sclerosis was considered, the patient was referred to a neurologist, Dr. H. D. McIntyre. According to his report there was weakness of the right side of the face. The remainder of the cranial nerves were normal. Deep reflexes were hyperactive on the two sides, those on the right being most pronounced. Abdominal reflexes were present but easily fatigued. A bilateral Hoffmann sign was present, which was more marked on the right. Ankle clonus was present on the right. Sensation was normal. Spinal puncture revealed normal pressure. The Wassermann reaction of the spinal fluid was negative, and the colloidal gold curve was normal. Analysis revealed a normal amount of globulin and 8 cells. The patient was given a course of typhoid vaccine intravenously, and four weeks later the vision in the left eye was 20/20+, with recognition of colors faintly in the fixation point. Two weeks later she returned, stating that her vision had begun to fail again. It was found to be 20/50, and two days later she was able only to count fingers at 2 feet (60 cm) in the nasal field. Craniotomy and exploration of the chiasmic area were performed by Dr. Nolan Carter. The right nerve was flat and yellowish, and the left was rounded, pink and edematous. An unusual amount of yellowish fluid was evacuated from the chiasmic cistern. No adhesions were found. Two weeks afterward, when the patient was discharged from the hospital, she could see light in the temporal field of the right eye (for the first time in five years) and was able to count fingers at 6 feet (182 cm) eccentrically with the left, which presumably had a large absolute central scotoma. The nerve head was somewhat edematous and pale. She has been followed since the operation at various intervals, and there have developed diminished motor power on the two sides and somewhat spastic extremities, especially on the left. The abdominal reflexes are absent. Her vision has remained unchanged, and both disks show atrophy of the mixed type. The neurologic diagnosis was multiple sclerosis.

In the present state of knowledge treatment should be liberal. All foci of infections should be attacked, especially the posterior sinuses, and the allergic state and the use of foreign proteins and salicylates should be investigated. If no improvement occurs after attention to these factors, surgical exploration of the chiasmic region should be performed. The operative mortality is low as a rule, and the recovery or the improvement of vision (in more than 50 per cent of the cases reported) is frequently assured, especially when the operation is undertaken soon enough. In many cases, particularly when the disks are atrophic, the recovery of vision is likely to be slow, so that one must not despair if

the vision remains poor for several months. It is possible, however, to say that if no vision is recovered within six months after operation the prognosis is hopeless.

CONCLUSIONS

Optochiasmic arachnoiditis exists as a clinical entity, presenting certain signs and symptoms which on careful study may yield a preoperative diagnosis. At any rate, the signs point to some involvement of the chiasmic region and warrant surgical exploration, at which time the diagnosis can be made. Case reports indicate that surgical intervention is justified on the ground of improved vision in most of the cases.

The posterior nasal sinuses may be the portal of entry for the toxin or virus, and recent evidence indicates that encephalitis, multiple sclerosis and the associated demyelination diseases as well as optochiasmic arachnoiditis may all be closely allied and may be different manifestations of the same pathogenic process, which could well be thrombi in the venous system of the nerve tissue. The many cases of retrobulbar optic neuritis in which dramatic improvement of vision occurs after treatment of the sinuses, whether by surgical or by medical hyperemic procedures, justify these steps.

It is too soon to say that signs of multiple sclerosis will not develop later in cases of optochiasmic arachnoiditis.

BIBLIOGRAPHY

- Arce, J. Optochiasmic Arachnoiditis (Syndrome of Balado), *Bull Acad de méd*, Buenos Aires **111** 106, 1934.
- Balado, M., and Satanowsky, P. Surgical Treatment of Atrophy of the Papilla, *Arch argent de neurol* **4** 71, 1929.
- Batson, O. V. Relationship of the Eye to the Paranasal Sinuses, *Arch Ophth* **16** 322 (Aug.) 1936.
- Bedell, A. J. Stereoscopic Fundus Photography, *Tr Sect Ophth*, A. M. A., 1935, p. 17.
- Beer, cited by White.
- Bollack, J., David, M., and Puech, P. Optochiasmic Arachnoiditis, Paris, Masson & Cie, 1937.
- Campbell, E. H. Relationship of Sinusitis to Optic and Retrobulbar Neuritis, *Arch Ophth* **16** 236 (Aug.) 1936.
- Cossa, cited by François.
- Craig, W. M., and Lillie, W. J. Chiasmal Syndrome Produced by Chronic Local Arachnoiditis, *Arch Ophth* **5** 558 (April) 1931.
- Cushing, W., and Eisenhardt, L. Meningiomas Arising from the Tuberculum Sellae, *Arch Ophth* **1** 168 (Feb.) 1929.
- Eagleton, W. P. Suppurative Meningitis of Otic and Nasal Origin, *Arch Otolaryng* **15** 885 (June) 1932.
- Fralick, F. B. Abstract from a course in retrobulbar optic neuritis given before the American Academy of Ophthalmology and Otolaryngology, 1936.
- François, J. Contribution to the Study of Optochiasmic Arachnoiditis, *J belge de neurol et de psychiat* **35** 188, 1935.

- Frost, A Papilledema, with Special Reference to Papilledema Associated with Sinus Disease, *Tr Am Ophth Soc* **33** 480, 1935
- Heuer, G J, and Vail, D T, Jr Chronic Cisternal Arachnoiditis Producing Symptoms of Involvement of the Optic Nerves and Chiasm, *Arch Ophth* **5** 334 (March) 1931
- Loeb, cited by Campbell
- Onodi, cited by Campbell
- Puech and Mahoudeau, cited by Bollack, David and Puech
- Putnam, T Etiologic Factors in Multiple Sclerosis, *Ann Int Med* **9** 854, 1936
- Rollet, cited by François
- Ruedemann, A Personal communication to the author
- Schaeffer, cited by Campbell
- Stark, cited by Campbell
- Vincent, C, David, M, and Puech, P In Regard to Seven Cases of Optochiasmatic Arachnoiditis, *Rev neurol* **1** 760, 1931
- White, L E Loss of Sight from Posterior Accessory Sinus Disease, with Report of Three Cases, *Boston M & S J* **176** 891, 1917
- Worms, G Oculo-Hypophyseal Syndrome Secondary to a Suppurated Sphenoidal Sinus, *Arch d'opht* **53** 207, 1936

RESEARCH STUDIES OF THE EYE

SOME TECHNICAL AND PRACTICAL NOTES

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In this article I do not claim to report any innovations but simply to call attention to some guiding principles of technic which have aided me greatly in histologic research in the field of ophthalmology

Since I am forced to work in a small private laboratory that lacks the substantial equipment of an endowed research institution and since I am able to devote to scientific research only such time as a professional practice permits, I have found it in some measure expedient to adapt my research technic to these conditions. Accordingly, these notes, although of no value to workers in large laboratories, may be of some use to men who, like myself, must carry on in a small laboratory

FIXING AND PRESERVATION OF SPECIMENS

Although solutions of formaldehyde are generally recognized as among the most unsatisfactory of fixatives and the disadvantages of their use as preservative fluids for anatomic specimens are known to all, nevertheless these solutions are still widely used by ophthalmologists

In 1925, while at Bologna, Italy, I had occasion to frequent the histologic laboratory of Professor Ruffini. After having examined the collection of histologic material which had been housed there for years, I asked the director's advice with respect to the most satisfactory methods of preserving ophthalmologic material for use in pathologico-anatomic as well as purely morphologic studies

Professor Ruffini showed me certain embryonal specimens which had been preserved for more than twenty years and which, so he told me, could still be put to admirable use in detailed cytologic research. He also provided me with notes on his technic of fixing and preserving, these, although unpublished at the time, appeared in print some years later

Ever since learning Professor Ruffini's technic, I have applied it in my own studies and in preserving my material. I find it superior to all others. First, in following meticulously the rules laid down by Professor Ruffini one obtains an excellent fixation which permits of the most varied staining and insures preservation of pathologicoanatomic specimens for years. Moreover, these specimens will retain the suppleness of fresh tissue and may be utilized for detailed histologic studies at any time

The sole disadvantage of the method is that it requires a good deal of time, especially if large specimens are to be fixed. Consequently, it becomes impracticable when there is urgent need for an early pathologicoanatomic report as an aid in diagnosis.

The fluid which Professor Ruffini recommended as a general fixative is designated "R 3" in his brochure. There are, as a matter of fact, two other liquids, R 1 and R 2, used, respectively, for fixing the eggs of batrachians and the larvae of anurous amphibians.¹

The formula for Professor Ruffini's fluid is as follows:

	Gm or Cc
Muller's fluid (2 Gm of potassium dichromate and 1 Gm of sodium sulfate in 100 cc of water)	50
Solution of chromic acid, 1 per cent	50
Glacial acetic acid	2
Mercury bichloride	1

The acetic acid can be added immediately, since it does not alter the fluid. On occasion, however, the fluid may turn brown and lose its efficacy. I attribute this phenomenon to the impurity of the reagents. If this discoloration does not take place within the first few days, there is no likelihood of its occurrence even at the end of several months.

This fixative is markedly hypertonic, and for this reason Professor Ruffini advised that in the preparation of delicate tissues the specimens should first be fixed for several hours in a dilute fixative and then placed in the fixative of full strength. As to ocular tissues, I have found it an excellent procedure to submit them first to a fixing fluid diluted to 50 per cent with distilled water. For small specimens of tissue, this preliminary process should last for from two to three hours. Whole eyeballs should remain in the weakened fixative for twenty-four hours, in fixing these organs it is well to make the injection into the vitreous by means of a platinum needle. The needle should penetrate along the equator in the superior or lateral segment. The site of perforation should be in the segment which will later be discarded, so that no mark will be perceptible in the cut sections. Enough fluid is injected—but the injection should not be made with too much force—to distend the eyeball that has collapsed after enucleation. After it has remained for from two to three hours in the diluted fluid, suspended by a thread

¹ F. Caramazza (*Rassegna ital d'ottal* **2** 1299 [Dec.] 1933), in his unwarranted criticism of my studies of filmy sediment on the crystalline lens capsule and on the iris (*Arch f Ophth* **119** 135, 1927), sought to display his familiarity with Ruffini's methods and stated that I referred to the fixative of Ruffini without specifying to which fluid I made allusion. Had he read Ruffini's brochure with any degree of attention he would have noted that only one formula is recommended for preservation of human material.

tioned to a tendon as a precaution against deformation, the eyeball will have become sufficiently firm to permit the removal of a segment from the walls in order to facilitate penetration of the fluid. An ordinary razor blade serves admirably for the removal of this segment. Subsequently, the eyeball is left suspended in the fluid proper for twenty-four hours.

After fixation in the diluted fluid, all specimens, large and small, are placed in the undiluted fluid for twenty-four hours. In the preparation of eyeballs it is well to remove a second segment opposite the first, this facilitates future treatment.

Coagulation without shrinkage of the vitreous is a phenomenon commonly observed after application of the fixative, this always occurs if the vitreous has been invaded by exudate.

The fixing process is followed by a lavage of twenty-four hours' duration under running water. After this, the specimens are regularly treated with Kaiserling solution II, the formula of which follows:

	Gm or Cc
Potassium acetate	50
Glycerin	100
Alcohol, 95 per cent solution	100
Distilled water	500

In this fluid the preparations not only resume their original suppleness but are rid of the chromic salts. As Professor Ruffini remarked, this loss is not the result of simple diffusion but of a veritable process of replacement similar to that which is observed in living tissue when, for example, ions of potassium are substituted for the ions of sodium. Here the chromium is replaced by potassium, a substitution which, if it is thoroughly carried out, makes it easier for one to attempt histologic staining which cannot be obtained on material fixed in fluid containing chromic salts.

Kaiserling solution, too, is strongly hypertonic. Professor Ruffini advised treating the preparations in a series of solutions of graduated concentration if one is to avoid too rapid changes in osmotic tension. He recommended placing specimens in each of the following dilutions for twenty-four hours: 1 part Kaiserling solution to 3 parts distilled water, 1 part Kaiserling solution to 1 part water, 2 parts Kaiserling solution to 1 part water, and undiluted Kaiserling solution. In order to eliminate the mercury bichloride, there should be added to the foregoing solutions a few drops of a concentrated solution of potassium iodide (10 Gm of potassium iodide to 10 Gm of water).

As I stated previously, tissues placed in Kaiserling solutions will be rid of the chromic salts. If the pieces of tissue are small, the fourth dilution will already be colorless. But in fixing whole eyeballs, par-

ticularly if the vitreous body, filled with exudate, has assumed a certain firmness, it is frequently necessary to change the fluid several times before the fluid within the tissue will remain colorless for twenty-four hours, that is to say, before one has obtained complete extraction of the chromic salts

This step tends to prolong the fixing procedure and may on occasion present a serious handicap in addition to entailing a great deal of expense in those countries in which alcohol and glycerin command high prices

After the specimens are free from the chromic salts, they are allowed to remain in the Kaiseiling solution. In this manner suppleness is preserved for many years with no modification of the histologic structure or tinctorial properties of the various tissues

For the process of rendering the specimens transparent after dehydration, I prefer to use benzene because of its great volatility. Professor Ruffini is opposed to the use of xylene and benzene, he expressed the belief that these substances render the tissues hard and friable. He recommended, however, that after the use of various solutions of alcohol a mixture of equal parts of cedar-wood oil and oil of bergamot be employed. I have never used this mixture for eyeballs for I personally prefer embedding in pyroxylin. On the other hand, I have placed smaller specimens of tissue in the mixture and readily observed its advantages, for with its aid I have been able to cut specimens of the human cornea into serial sections of from 6 to 8 microns

As I have already mentioned, one of the principal advantages of the technic just described consists of its adaptability to the most diverse methods of staining. For example, after following the outlined procedure one may obtain excellent stainings with the methods of Giemsa and of Bielschowsky

For ordinary staining with hematoxylin and eosin, I use Carazzi's hematoxylin, a staining solution that resembles the hemalum of Meyer. It offers two fundamental advantages. (a) It matures rapidly on account of its potassium iodate content (and this permits it to be utilized only a few hours after it has been made up). But in certain climates, that of São Paulo, for example, it is necessary to refrain from adding this salt, as it causes it to age rapidly and become useless. Without this salt, on the contrary, it matures in from three to four days and preserves its tinctorial properties for several months. (b) It never overstains the preparations, and no differentiation is necessary—two factors of definite importance to the worker who does not always have abundant time at his disposal. The sections remain in the staining solution for from ten to twenty minutes or more and are then washed in running water or placed in a large evaporating dish that contains tap water

The formula of Carazzi's hematoxylin is as follows

	Gm or Cc
Powdered hematoxylin	0.50
Potassium iodide (KIO_3)	0.01
Potassium alum	25.00
Glycerin	100.00
Distilled water	500.00

Dissolve at room temperature and stir frequently

For staining with eosin, I use a 1 per cent aqueous solution in which I allow the sections to stand for from twenty-four to forty-eight hours. I then wash the tissues in distilled water and differentiate them in alco-

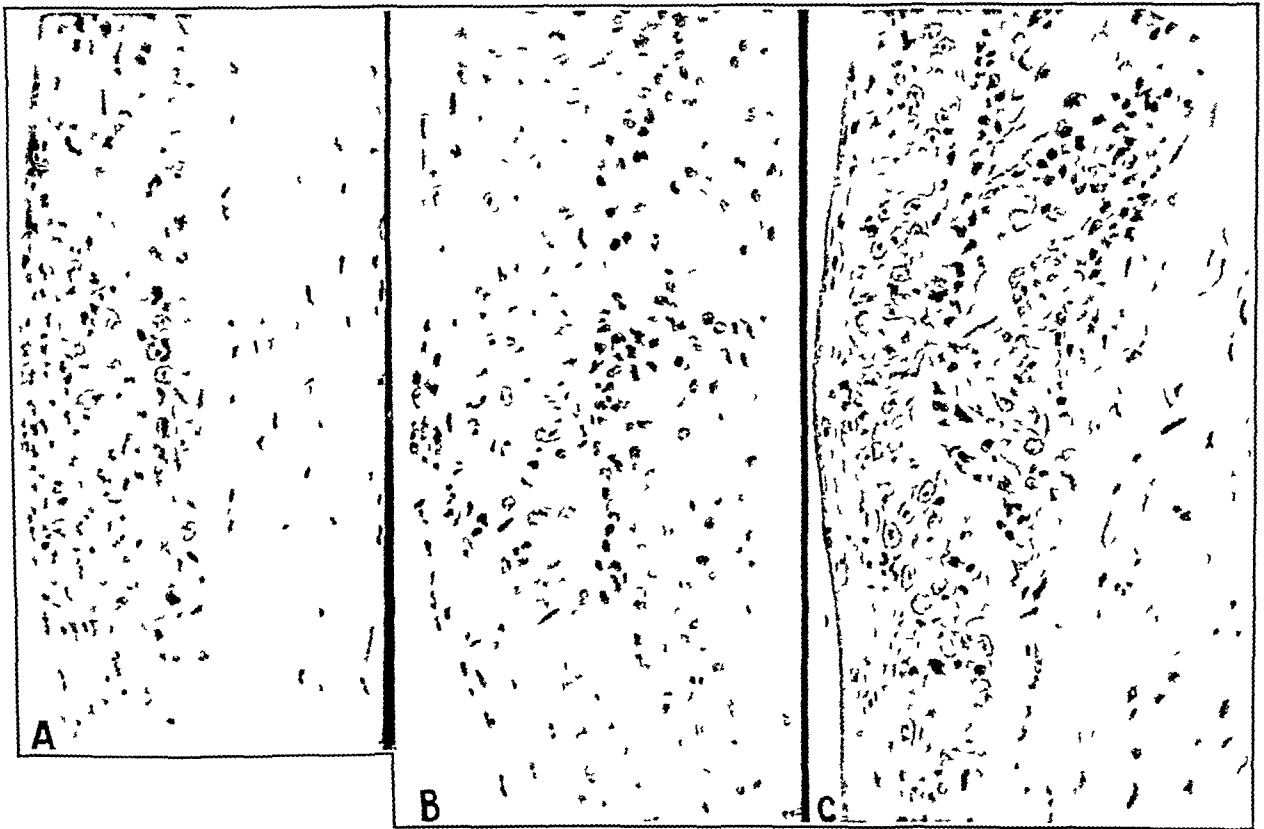


Fig 1—Corneal tissues fixed in Ruffini's fluid and stained with hematoxylin and eosin. *A*, trachomatous pustule of the cornea. The normal and altered epithelial cells and polymorphonuclears that have immigrated into the epithelium are well differentiated by the staining. *B*, extremity of a trachomatous corneal pannus. The structure of the nuclei is plainly distinguishable. *C*, extremity of a trachomatous corneal pannus. Edema of the epithelium and the different types of cells which go to form the extremity of the pannus and the vascular wall are plainly visible.

hol (rapid passage through 70 per cent alcohol and 95 per cent alcohol and twice through absolute alcohol). The preparation loses the excessive coloration without danger of complete decoloration (such as occurs in too rapid stainings). I obtain, in addition, a clear differentiation of the various protoplasmic and nucleolar parts (fig 1).

If the tissues have been fixed according to the methods described, silver impregnation by Bielschowsky's method (fig 2) will provide results inferior to those obtained after fixation in Flemming's solution. I have found that impregnation is easier and more thorough after double embedding in pyroxylin and in paraffin. However, in order to obtain satisfactory silver staining it is often necessary to keep the sections in a solution of silver hydrate for a long period. I employ Bielschowsky's staining fluid (or better still Achucario's formula) in undiluted form and

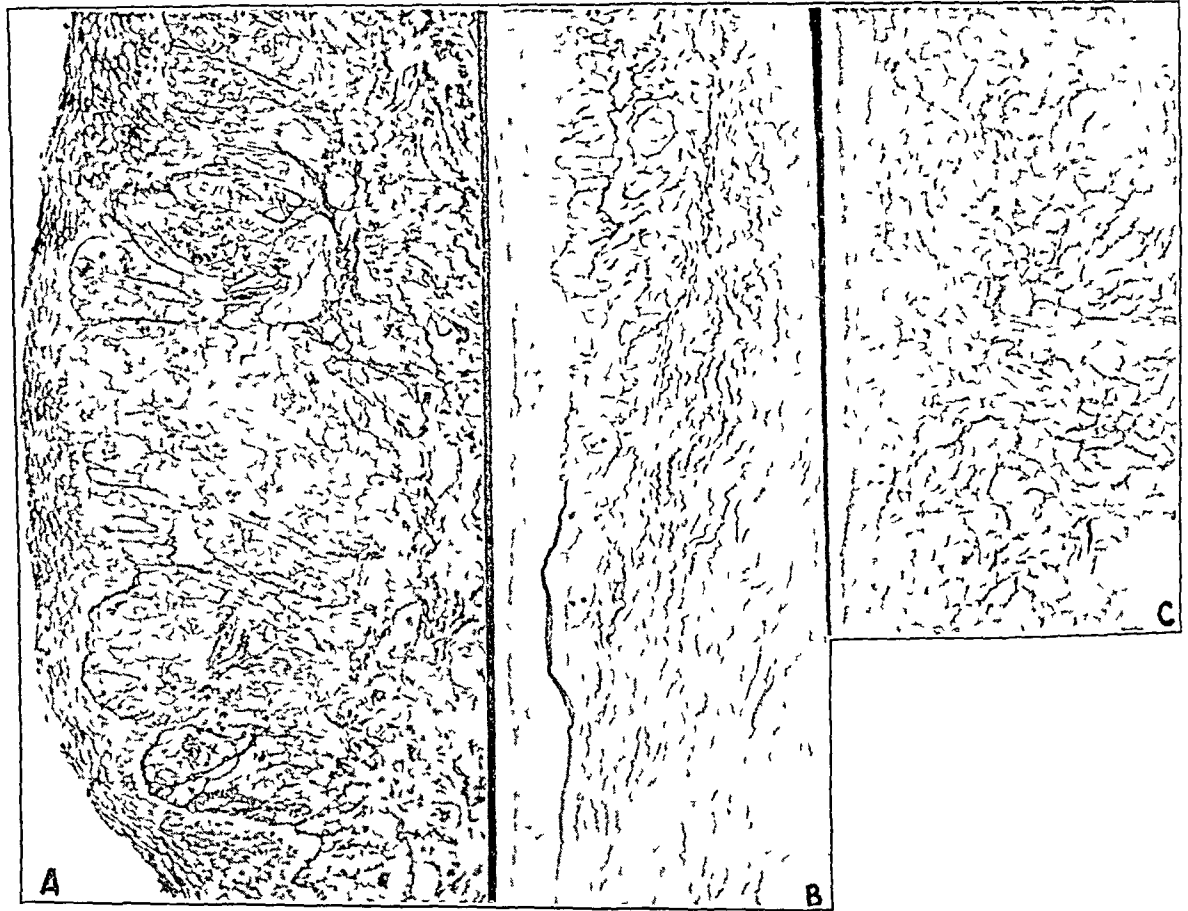


Fig 2—Corneal tissues fixed in Ruffini's fluid and stained by Bielschowsky's method. *A*, trachomatous tissue from the limbic region, connective and reticular tissue are present. *B*, extremity of a trachomatous corneal pannus, modifications of the corneal lamellae and of the lamina elastica anterior. *C*, trachomatous corneal pannus, modification of the infiltrated corneal lamellae.

leave the sections in it for one hour. I find it necessary now and then to double or even to treble the amount of time allowed for silver staining.

It is well in each instance to check the time required for impregnation by means of a small slide prepared expressly for this purpose, one

should keep in mind that granular reaction in the fibrils indicates that the time allotted has been inadequate. If the time required for impregnation is once determined for a certain type of tissue in a certain type of solution, the results will be absolutely constant.

Whenever I wish to examine the same tissue specimen under different stains, I make serial sections and then prepare four or more series in the following manner. I have before me four slides, on each of which I pour a small amount of distilled water and place the section obtained by the microtome directly on them. On the first slide I place the sections corresponding to the numbers 1, 5, 9, 13 and so on, on the second slide, sections numbered 2, 6, 10, 14 and so on, on the third

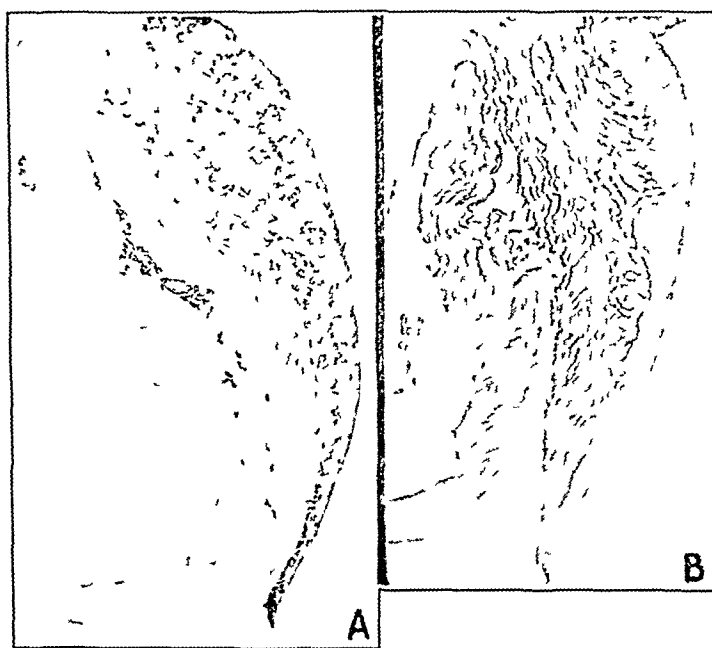


Fig 3—Trachoma of the lunula and an initial trachomatous nodule. The two sections, cut a few microns apart, were stained, respectively, with hematoxylin and eosin (*A*) and by means of silver impregnation (*B*).

slide, sections numbered 3, 7, 11, 15 and so on, and on the fourth slide sections numbered 4, 8, 12, 16 and so on. When the slides are full, I add several drops of water and place the slides in the incubator, where they remain for a number of minutes at 52 C, a temperature calculated to distend the sections both satisfactorily and quickly. I leave them in the incubator at 37 C for from twenty-four to forty-eight hours. This distribution of the series enables me to examine sections stained by four different processes, which are separated from one another by only a few microns. Thus, for all practical purposes it may be said that the identical section has been examined by different methods (fig 3). The advantages of such a technic are manifest.

REMOVAL OF CORNEAL TISSUE FOR BIOPSY

The removal of tissue for pathologic study is seldom applied to the cornea, no doubt because of the common feeling that it is dangerous to touch the cornea. Yet from my own experience I have found that nothing is more innocuous, even for the cornea itself, than skilful removal of tissue. Needless to say, one should not think of attempting to remove tissue from the pupillary field, as the remaining corneal facet would cause functional impairment.

In addition to the induction of complete anesthesia before the removal of tissue, there are two precautionary considerations: preliminary disinfection of the conjunctival sac and the use of well sharpened knives.

I am accustomed to begin disinfection of the conjunctival sac two or three days prior to removal of tissue. In the morning the parts are painted with a 2 per cent solution of silver nitrate, about ten minutes later the conjunctival sac is washed, the eyelids being inverted. When this is done, I introduce an ointment containing Besredka's antivenous into the conjunctival sac, and in the evening I instil a 4 per cent solution of mercuriochrome. Before removing any tissue I wash the conjunctival sac with a substantial quantity of physiologic solution of sodium chloride. With these precautions I have never encountered infections in the course of removal of more than 100 biopsy specimens, the majority of which were taken from trachomatous eyes.

For a cutting instrument, I use an iridectomy knife, it must be well sharpened, and I never use the same knife for removal of more than one or two specimens of tissue. The eyeball is immobilized by grasping it with a fixation forceps such as is used in cataract operations, this is done in a sector distal from that in which the tissue is to be removed. Tissue is removed from the central zone of the cornea toward the limbic zone. The tip of the lancet is inserted almost perpendicularly at the extremity of the zone to be removed, and when it has penetrated the superficial tissue layers it is turned so that its surface becomes virtually parallel to that of the cornea, with the lancet in this position, one advances directly toward the limbic region, forcing oneself to follow the curvature of the cornea. When the point of the instrument has penetrated beyond the limbic region, one of the borders of the piece to be removed is freed by a lateral movement, the lancet again penetrates beneath the piece of tissue and frees the other border by a second lateral movement. If the tissue still adheres to the side of the conjunctiva, it may be detached by means of iris forceps and scissors. In this manner sizeable specimens can be procured (fig. 4).

If the cornea is well infiltrated, removal of tissue is extremely easy, as the tissue offers no resistance. Conversely, in a cornea that is only slightly infiltrated one often encounters formidable resistance, hence the need of an excellent cutting instrument.

It goes without saying that the procedure described can be done only in approximately the anterior half of the cornea, since perforation would be impossible, and too deep an incision may produce ectasia. It is well before undertaking removal of tissue from the cornea to study the corneal thickness by means of the slit lamp. If the cornea has

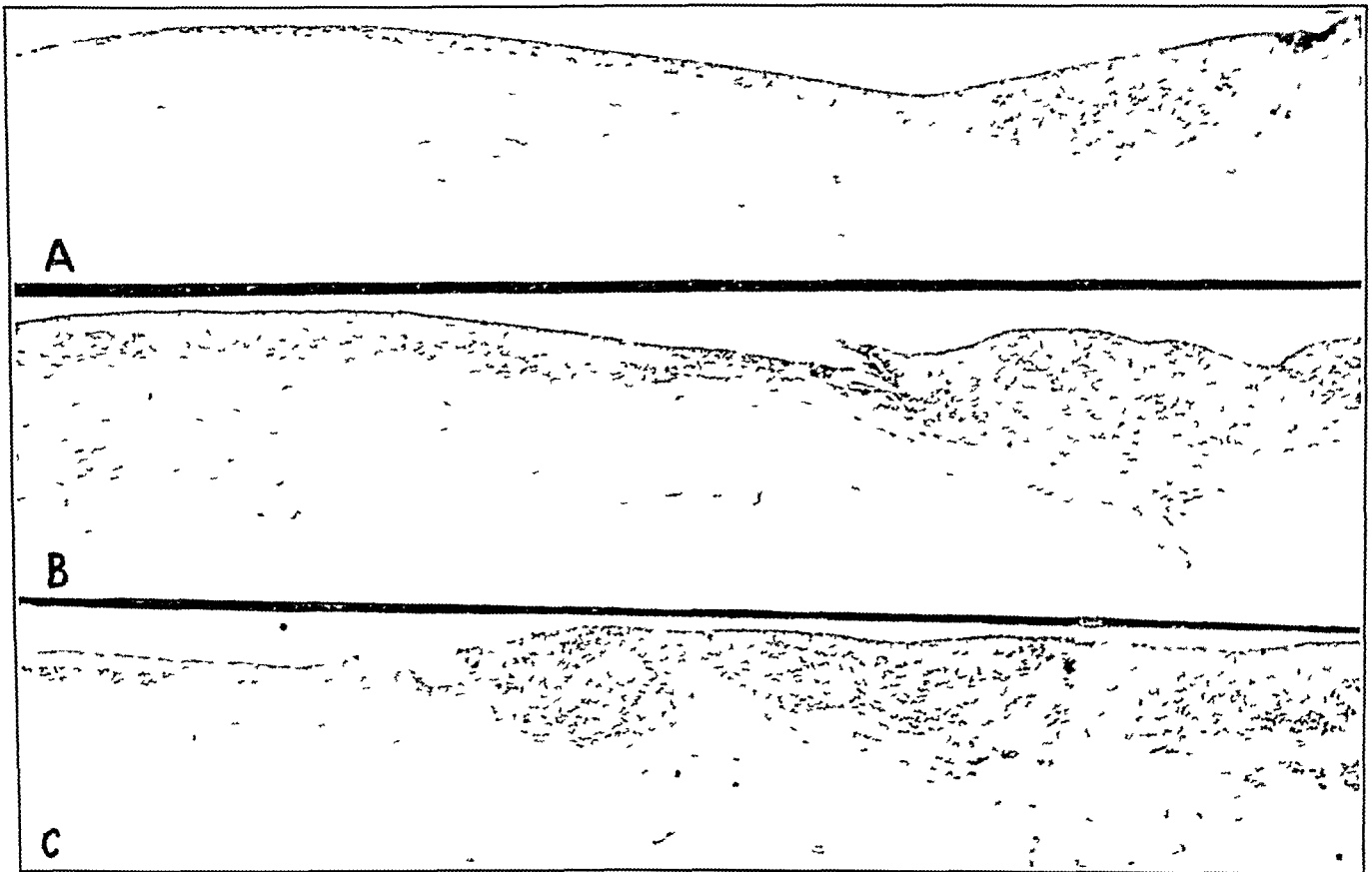


Fig 4—Biopsy specimen of the cornea showing *A*, an initial trachomatous corneal pannus and avascular trachomatous keratitis, *B*, a well developed trachomatous corneal pannus with papillary formation in the limbic region and discrete parenchymatous infiltration, *C*, trachoma of the lunula, a nodule of the limbic region and corneal pannus

already been rendered extremely thin by pathologic processes, removal of tissue will be difficult or even impossible.

I have removed corneal tissue for biopsy numerous times, and only twice has the tip of the lancet accidentally penetrated the anterior chamber, and then the corneas were thin. In both instances, as soon as the anterior chamber began to empty I withdrew the instrument without removing any tissue, and the incident was inconsequential.

When I have completed the removal of tissue, I instil a solution of mercurochrome into the conjunctival sac and introduce an ointment containing an antiviral, then I apply a dressing, which is renewed daily for three or four days. This length of time is sufficient for the cornea to be covered by epithelium, even if the loss of substance is considerable.

In connection with the removal of tissue for biopsy, a small false pterygium may be formed. Sometimes a corneal facet remains which is often more transparent than the adjacent zones but which, being situated at the periphery, is of no significance to corneal function.

PLACEMENT OF LANDMARKS ON CONJUNCTIVAL OR CORNEAL TISSUE

Whoever has devoted himself to the comparative study of the biomicroscopic and histologic aspects of tissue knows how difficult it may be to find again minute lesions, even on serial histologic sections especially if the pathologicoanatomic character of such lesions is unknown.

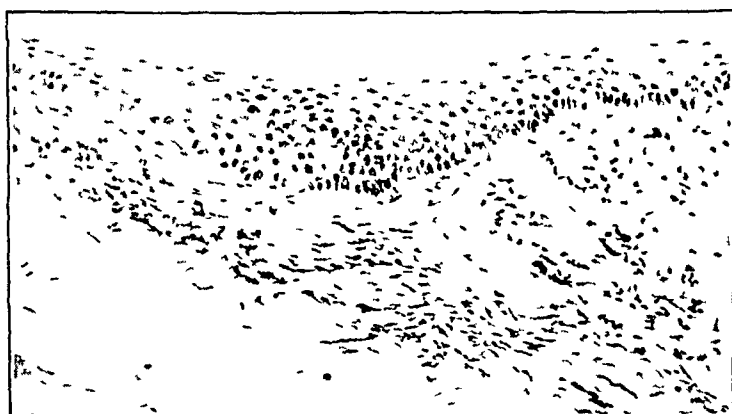


Fig 5—Section of corneal tissue demonstrating the value of tattooing. An abundance of India ink remains in the epithelial cells along the path of the needle (center of the fossette) and in the cells of the subjacent connective tissue.

I encountered this difficulty when I first attempted to study the parenchymatous vessels, which project forward into the cornea, and the structure of Herbert's extremity of pits. It then occurred to me to employ tattoo marks of India ink, which would serve as landmarks and which I could execute under the slit lamp. Although it would be superfluous to describe the technic of tattooing, which is so well known, I should like to outline certain technical points applicable to specific objectives.

In cases in which enucleation of the eyeball is urgent, tattooing can be done the night before the operation or even at the start of the operation. It is a useful procedure to mark two points, one on either side of the area of special interest. These points will be so situated that the line which unites them will indicate the precise course to follow in

sectioning the tissue with the microtome. A single prick with the tattooing needle suffices to leave behind enough india ink to identify in the specimens and the histologic sections the particular segments of tissue in which one may be interested.

If it is possible or advantageous to allow an interval between the time of tattooing and that of removal of the tissue (as is often the case when one has to deal with a small biopsy specimen), it is preferable to make three or four punctures with the tattooing needle, for, as every one knows, some of the ink will be lost during the first days. Figure 5 shows tissue which was tattooed under these conditions.

Tattooing can likewise be profitably employed for fixed specimens, as in the small lesion produced by the needle there remains a quantity of ink sufficient to serve as a guide when the piece is sectioned by the microtome. Furthermore, in histologic sections one sees only a few granules of ink running free in the edges of the slight solution of continuity. The method is serviceable, since often slight solutions of continuity suffice to put one on the trail of the points in which one is interested.

ADVANTAGES OF USE OF COAGULANTS IN OCULAR OPERATIONS

ESPECIALLY IN EXTRACTION OF CATARACT AND IN PLASTIC
OPERATIONS

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For several years I have been accustomed to use coagulants in surgical operations on the eyeball and its adnexa. The excellent results that I obtain with such agents and the fact that they are so little utilized, at least so far as I have been able to ascertain, prompted me to call the attention of my colleagues to a technic which, without warrant, has been generally neglected.

The objection may be raised that skilful and opportune administration of epinephrine hydrochloride or the use of thermocautery suffices to prevent or to inhibit the minute hemorrhages which may complicate an otherwise adroitly and expeditiously performed operation. These two measures represent completely disparate mechanisms.

Epinephrine, as every one knows, inhibits hemorrhage by the contractile effect exerted on the vascular walls and the concomitant constriction of the lumen. But the drug has no influence on atheromatous vascular walls, and this explains why the hemostatic effect appears so slight when the drug is administered to aged persons who are markedly arteriosclerotic. Moreover, as is well known, vasoconstriction is succeeded by a further effect, vasodilatation, which gives rise to secondary hemorrhages. Still one must not forget that this state of vasodilatation is anticipated and exacerbated by the administration of atropine and that this circumstance renders dangerous the instillation of the medicament directly after an operation in which epinephrine has been used.

Cauterization inhibits hemorrhage by the production of an eschar, which serves to occlude the vascular lumens, or by coalescence of the walls, the effect of a coaction. Yet there is no denying that this procedure, to which Denig¹ referred favorably in a recent article, is not always practicable, for example, if hemorrhage of vitic origin is present. Besides, even if cauterization is done with a loop heated at the lamp, the eschar produced, although extremely slight, still represents a source of inflammation which may retard the process of cicatrization.

¹ Denig, R. Die Iridotorsion im Vergleiche mit der Trepanation der Lagraneschen Operation und der Zyklodialyse, *Klin Monatsbl f Augenh* 99 1 (July) 1937

The action of a coagulant is altogether different from that of cauterization. With the use of such an agent a sufficiently tenacious coagulum rapidly forms, which occludes the severed vessels. In this way one merely accelerates and reinforces the physiologic mechanism which tends to arrest hemorrhage.

I have long used a proprietary coagulant from blood platelets (coagulen) for this purpose. Motivated by the usual feeling of fidelity to the methods which lead to favorable results, I have refrained from experimentation with other products, but it is likely that the same results would be achieved by means of similar substances or with the use of purified snake venom, which has recently been introduced into surgical practice as a hemostatic.

For operations on the eyeball, I prescribe the injection of 20 cc. of the coagulant into the subcutaneous tissue of the abdominal wall. Experimentation has shown me that a satisfactory effect is attained within two hours. It always seems to me that the action is more pronounced after a long interval. Accordingly, if I am to operate early in the morning I regularly administer the coagulant the evening before, but if an operation is scheduled for late in the day the patient receives the coagulant during the early hours of the morning.

For all operations in which the eyeball is not excised, I prefer the local application of the coagulant in its powdered form. At the proper moment a bit of powder is spread over the operative field. I then wait for the formation of a dense coagulum, which is removed before resumption of the surgical work.

It is not my intention to introduce any case histories, and I shall limit myself to a discussion of certain considerations and observations elicited by several years of practical experience.

Coagulants are useful in operations for the extraction of cataract, whether by the intracapsular or the extracapsular method.

As every ophthalmologist knows, when a conjunctival flap is made during keratotomy a considerable amount of blood will often penetrate the anterior chamber, in patients whose blood vessels are atheromatous and whose eyes are hypotonic, the surgeon may readily observe a small stream of extravasated blood penetrate and fill the anterior chamber, it may even happen that after he has succeeded in removing this initial extravasation of blood, a second, smaller stream will gather. Such incidents may be particularly annoying during operations on the iris. Hemorrhage sometimes continues from the iritic vessels even after the operation is completed, this is particularly likely to occur in cases of complicated cataract. But although hemorrhage of this sort may mar the smooth course of an iridectomy or make difficult the grasping of the capsule of the lens during an intracapsular extraction of cataract, it is most likely to cause serious consequences during extracapsular extraction.

In fact, whereas the blood deposited on the anterior surface of the iris is, as a general rule, absorbed with great ease, that which remains on the capsular surface is slow to disappear, and if it forms into masses, complications always arise. If the latter occurs, one observes over a period of several days minute coagula between the masses or on their surfaces. It is noted that the masses, which probably will be covered with a fibrinous sediment, are reabsorbed with difficulty. In general, if the coagulum is slightly larger, absorption no longer takes place, but instead the organization of the coagulum itself forms a synechia between the periphery of the iris and the remnants of capsular and crystalline substance. This marks the beginning of a secondary cataract.

But, if one has administered a preliminary injection of a coagulant and if after keratotomy one waits a minute or two, it will be observed that an extremely dense coagulum will form, which may have to be removed with the forceps. Blood that has extravasated into the anterior chamber or which appears during an operation on the iris will also coagulate rapidly. Usually this coagulum can be removed by lavage of the anterior chamber, this is done by the introduction of a David spoon and by directing to the part of the spoon remaining outside of the chamber a fine jet of physiologic solution of sodium chloride. More rarely, one has to extract the coagulum by means of the forceps. After the coagulum has been removed, no more blood is observed to extravasate into the anterior chamber, hemostasis remains complete.

The tenacity of the coagulum renders more solid the adhesion of the conjunctival flap and facilitates the formation of the anterior chamber. Another slight advantage to be obtained with the use of coagulants is that if one employs the Elschnig fixation forceps to grasp the episclera, the hemorrhage that is readily induced by this instrument in the region of fixation will be immediately arrested.

The use of coagulants offers the same advantages in operations for glaucoma as were mentioned in connection with the conjunctival flap employed in cataract operations, however, during the operation proper, the advantages are less important if one is accustomed to work under a fine jet of physiologic solution of sodium chloride, which will constantly keep the operative field free from blood.

After enucleation, a small amount of coagulant introduced into the orbital cavity, followed by compression of several minutes' duration through the eyelids and, finally, the application of a compressive dressing, obviates completely the extravasation of blood which is so often present, particularly in elderly patients, and which frequently involves the loose connective tissue of the other eye.

In various operations in the adnexa (such as an operation on the eyelids and the lacrimal sac or an operation for pterygium) the timely administration of a bit of powdered coagulant permits one to work in a

perfectly drained field and obviates the danger of minute secondary hemorrhages, which are often so troublesome

But the field in which the use of coagulants renders the most remarkable services is that of plastic operation. I was first moved to experiment along this line following an extremely untoward incident that complicated a technically successful plastic operation. In view of the good results that I was able to obtain with the use of a coagulant, I decided to apply such an agent regularly.

I extirpated the nasal half of the lower eyelid of a man about 75 years of age, and then reconstructed the lid by rotation of the pedicle flap transplanted from the cheek. In order to combat a particularly irksome hemorrhage, I also resorted to the instillation of epinephrine hydrochloride in the operative field. About two hours after completion of the operation the bandage was completely soaked with blood, and soon blood began to drip from it. Efforts to check the bleeding by injections of hemostatics failed completely, and it was necessary to remove the dressing. A huge hematoma was found under the flap. Digital compression brought about cessation of the hemorrhage. However, as one might have expected, the flap became necrotic, and it was only by further surgical intervention that the deleterious sequels of this accident were in some small measure mitigated.

This was the instance which, as I already mentioned, decided me to use a coagulant, and local applications of the latter from the first have proved extremely effective. However, in a case in which I had administered a preliminary injection of coagulant, necrosis of the flap took place. After meditating over the contrast between this bad result and the truly excellent results obtained with local applications, I arrived at the following (perhaps erroneous) interpretation. Manipulations of the cutaneous flap (compression with the fingers or forceps, stretching the tissue) tend to produce traumatic vascular lesions, the coagulum that forms within such lesions will be extremely dense owing to the action of the (previously injected) coagulant and accordingly absorption will be difficult and the restoration of circulation throughout the flap will be retarded. After this one unfortunate incident, I abandoned the injection of a coagulant in plastic operations. I am therefore unable to state with certainty whether the poor results were a coincidence or whether such results would be the rule.

One more advantage assured by the use of coagulants in plastic operations is that the resultant solidity of the coagulum promotes coalescence of the flap with the underlying tissue, and this doubtless facilitates the "taking" of the graft.

The foregoing observations clearly demonstrate the great importance of the use of coagulants in ocular operations.

ROTATION OF THE CHEEK IN OPHTHALMOLOGY

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The method of rotation of the cheek consists in loosening the still healthy portion of the cheek, combined with more or less skin of the neck, for the purpose of restoring the contour of the face after all sorts of mutilations. These may be either small or large and may comprise mutilations of the upper and the lower lip, the nose, the cheeks and the lower eyelids. Even complete noses can be reconstructed. In such cases the inner cover of the nose can be prepared beforehand by an "epithelial inlay" placed under the skin of the cheek which is used to form the nose.

This loosening of the skin is done by making a large incision around the cheek, the natural lines of the face always being followed, i. e., the lines between the nose and the cheek and the cheek and the edge of the eyelid. The incision continues as far as the temple and descends along the ear at the side of the neck.

In this way the cheek, more or less complete, is utilized to restore the face, and the skin of the neck, which it drags with it, partly covers the cheek, forming a large wound in the neck instead of the original one which was in the face. This wound in the neck can always be closed by sutures, owing to the great elasticity of the skin of the neck, and in cases of extensive repair the head is inclined toward the shoulder and held there for several weeks, until the wound is completely healed.

I introduced this method in 1915. It is a general method and can be applied to all sorts of mutilations of the face. For this reason it is of great practical value even for the ordinary surgeon who has not specialized in plastic surgery, for the stomatologist and for the ophthalmologist.

As the cheek is rich in vessels, necrosis is almost impossible, even though there is tension in the portion of the cheek which has to cover the gap. If the rotation is well applied, no tension will exist in the part covering the gap, but the tension of the sutures round the neck will increase as they approach closer to the neck. Numerous sutures must join the turned cheek to the surrounding skin. The result is that the great tension which is caused by the strong rotation is divided among such a great number of sutures that there is only a small amount of tension on each one. For that reason the stitches must be made with the finest needle and the thinnest silk that can be found. This is why the scars scarcely show. The alternate sutures are taken away at the end of a week, and those that are left remain as long as is necessary, according to the tension, and are taken away gradually.

The general scheme varies with the size and shape of the defect. Figure 1 *A* shows the technic used in the reconstruction of the lower eyelid, and figure 1 *B* that used in the restoration of the upper eyelid.

REPORT OF CASES

CASE 1—In 1937 I was called in by Professor Kondoleon, professor of surgery at the University Clinic in Athens, to see a girl 13 years old (fig 2 *A* and *B*), who had burnt her face when younger. The left cheek had one long scar running along the nasolabial fold, but the right cheek was almost completely destroyed,

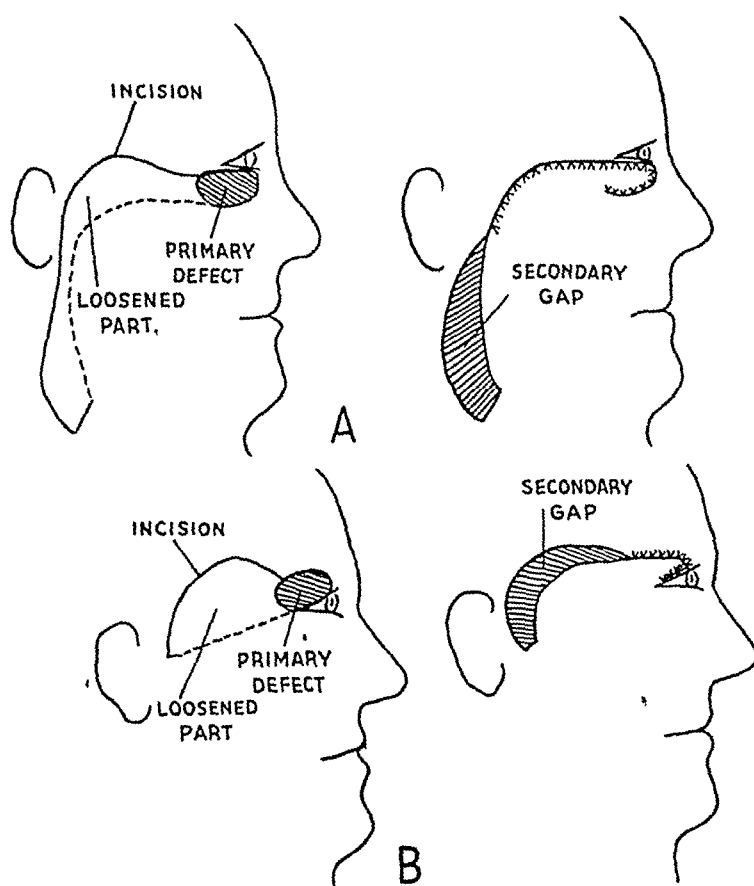


Fig 1—*A*, technic employed in the reconstruction of the lower eyelid, *B*, that employed in the reconstruction of the upper eyelid

there being one large scar. She had a large ectropion of the lower eyelid and of the upper lip. I decided to apply my method of rotation of the cheek to repair both ectropions simultaneously and also to reconstruct the cheek with normal skin. The portions of the conjunctiva and the mucous membrane of the lip which were dragged toward the center of the cheek were loosened by incisions from the underlying scarred muscle tissue until they were completely free. The edge of the eyelid was then shortened to its normal length by the excision of a portion of its outer edge. This was necessary because the scars of the cheek had gradually lengthened the edge.

Great care had to be exerted to take away the scarred muscle tissue with the conjunctiva, because this might cause a repetition of the ectropions. The rotation had to provide the muscles and skin of the new lower eyelid, just as it had to

furnish new muscle tissue and skin for the lip, after the mucous membrane of the lip had been freed in the same way as the conjunctiva

Figure 3 *A* shows the large defect in the cheek resulting from the operation and from the removal of all the scarred skin and muscle tissue from the right cheek. The dotted line in the sketch indicates the incision of the parts of the face

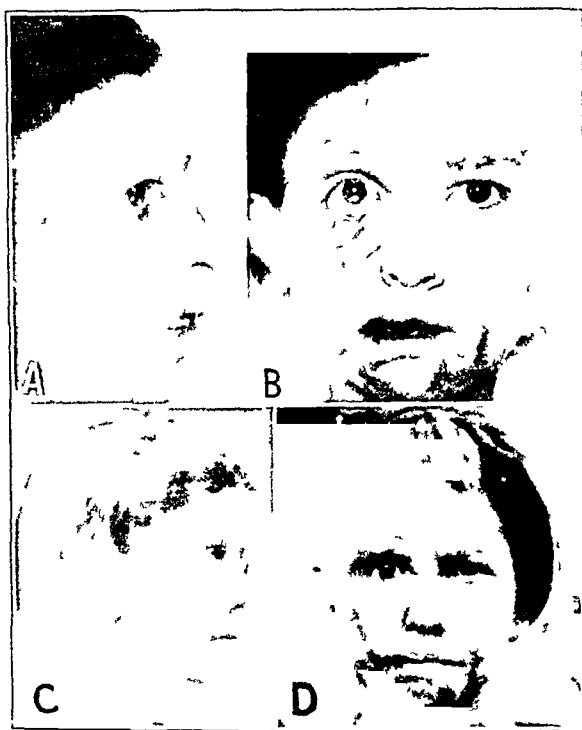


Fig 2 (case 1) — *A* and *B*, patient before operation, *C* and *D*, patient some weeks after rotation,

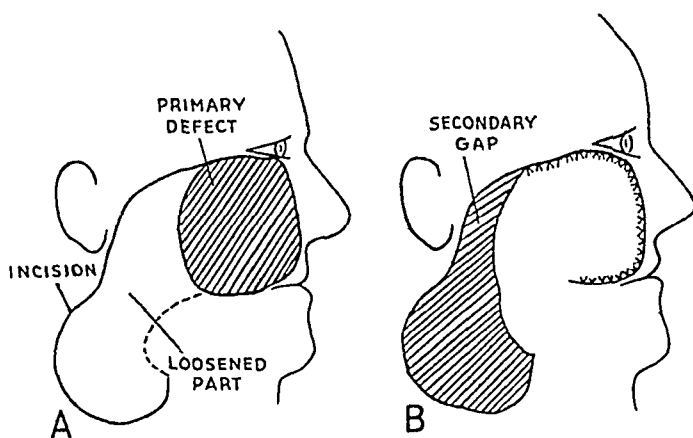


Fig 3—*A*, the first step of the operative technic employed in case 1, *B*, the second step

and neck to be rotated, when they had been sufficiently loosened and made mobile enough to cover the large defect in the cheek. This rotation caused a secondary gap in the neck, which could be sewn together. Figure 3 *B* shows the primary gap being closed and the resulting secondary gap which had to be sewn together.



Fig 4 (case 2) —A, photograph showing the inability of the patient to close his left eyelids, B, photograph showing method of rotation used, and C, photograph showing complete restoration of both eyelids

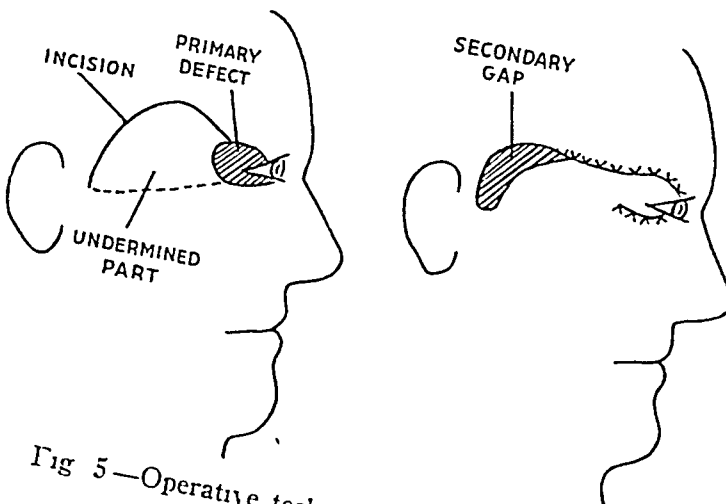


Fig 5—Operative technic employed in case 2

Figure 2 *C* and *D* show the girl some weeks after the rotation without any corrective operation. The ectropion of the eyelid and that of the lip were radically healed, scars could scarcely be seen on the right cheek. The expression of the face was almost normal except for the curve of the mouth, which was caused by the new function of the muscle tissue brought from some distance away. Gradually the expression of the face will become normal in the course of a few months when these muscles have adapted themselves to their new functions.

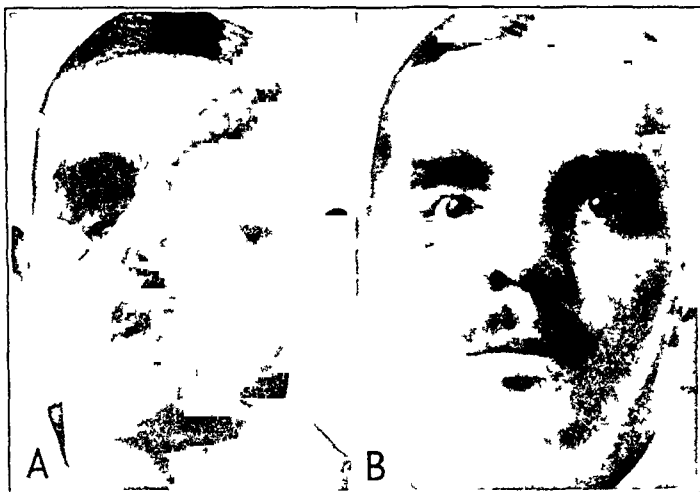


Fig 6 (case 3) —*A*, patient before operation, *B*, patient after operation

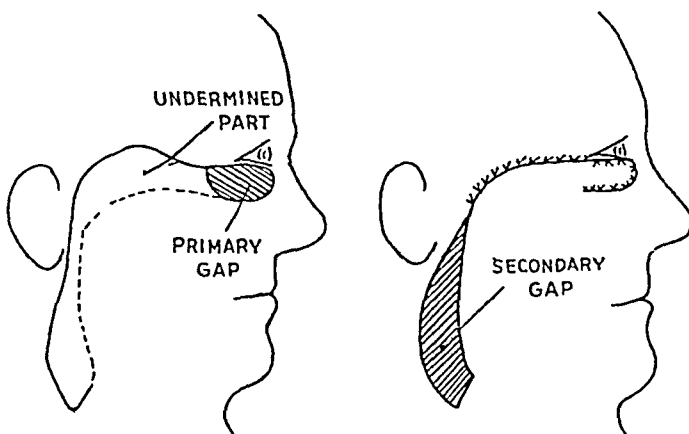


Fig 7—Operative technic employed in case 3

CASE 2—Patient W had both eyelids of his left eye destroyed. In figure 4 *A* the patient is shown trying to close his eyes, but as the greater part of the left eyelids was missing he could not make them meet more than is pictured. Rotation of the cheek repaired both eyelids. This is the highest point to which rotation of the cheek can attain. The method used is clearly seen in figure 4 *B* and in figure 5. Figure 4 *C* shows both eyelids completely restored. Slight corrections from the esthetic point of view are still needed.

CASE 3—Patient E needed an entire right lower lid, as his had been completely destroyed (fig 6 *A*). Figure 6 *B* and figure 7 show how rotation of the cheek was utilized for this purpose.

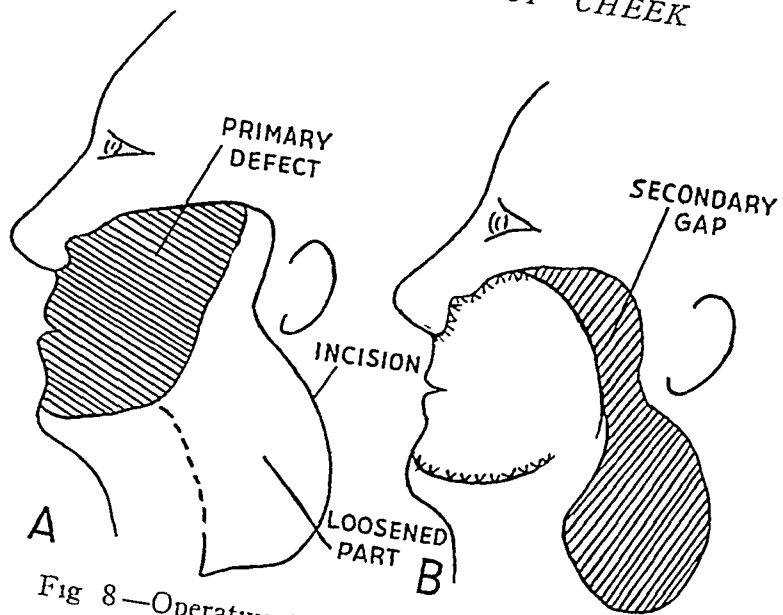


Fig 8—Operative technic employed in case 4



Fig 9 (case 4) —A and B, patient before operation, C and D, patient after operation

As the lower part of the socket was missing, an "epithelial inlay" was made to cover the inner side of the rotation flap in order to form the inner skin cover for the lower eyelid, so as to make it ready later to receive the artificial eye. In figure 6 *B* the artificial eye is already in place.

This "epithelial inlay," mentioned in the previous case, is a method I introduced over twenty years ago and is extensively used for plastic and structural surgical repair. It is also extremely valuable in the field of ophthalmology. The method consists in taking an exact model with stent (a material used by dentists) of wounds for which free skin grafts are to be used. When the stent becomes stiff, it is wrapped in a thin Thiersch graft, with the epidermis toward the mold. The mold then is replaced on the wound in exactly the same position as it was when taken and kept there under firm pressure for a week. This pressure prevents any secretion between the wound and the graft and in this way insures complete healing of the graft. With other methods these secretions loosen partly or completely the already attached graft. The thin grafts are taken from the inner side of the thigh, the resulting wound heals in a few days under a dry dressing.

I shall describe a case in which the gap is of the type that is the most difficult to repair, not that it is the largest, but it is so close to the neck that scarcely any part of the cheek is available and almost the entire rotation has to be made from the neck.

CASE 4—The patient, a French physician, was under the care of the well known ophthalmologist Morax, of Paris. The doctor was epileptic and had fallen into the fire, the left side of his face being entirely burned away. The eyelids were destroyed, and the cheek was replaced by a star-shaped scar. Morax had restored the eyelids, utilizing my method of biologic frontal flaps, i. e., pedicled flaps from the skin of the forehead, the pedicle not containing any skin but only veins, lymph vessels and nerves, all surrounding the frontal artery. These vessels remain protected by a layer of connective tissue. The advantage of this method is that the vessels of the elastic pedicle are never shut, even if the flap is turned at an angle of more than 180 degrees. Another advantage is that it is economical, as no skin tissue is wasted in the pedicle, the flap is mobile and there is no danger of necrosis.

After this operation Morax consulted me for the restoration of the left portion of the nose, the completely scarred cheek and the lower lip. I cut away the part shown in figure 8 and made an incision in the face and neck, as indicated in figure 8 *B*. The part surrounded by the cut was loosened and brought into the missing part of the face. The secondary gap was sewn together under great tension, and the head had to be inclined toward the shoulder for a week. Figure 9 *A* and *B* show the patient before operation, and figure 9 *C* and *D*, after operation. After such a large rotation of the cheek, corrective operation is usually necessary, but in this exceptional case the result was so satisfactory that the patient did not wish any further correction and is still delighted with the result.

BIOCHEMISTRY OF THE LENS

XII STUDIES ON GLUTATHIONE IN THE CRYSTALLINE LENS

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The possible functions of glutathione in the lens as well as in the rest of the body have been a matter of discussion since the discovery of this substance by Hopkins¹ Although much experimental work has been done on the glutathione in the lens, its actual isolation from this organ was only recently accomplished by two of us² That glutathione has an important function in the metabolism of the lens appears likely from the work of Adams,³ who showed that the oxygen uptake of a lens extract became nil when glutathione was dialyzed out, readdition of glutathione to the extract caused a resumption of its oxygen uptake It was indicated that this substance is a constituent of an auto-oxidation system in which the sulfhydryl can act as a hydrogen donor, being oxidized to the disulfide form, which in turn can accept hydrogen from a "thermostable residue" It was shown by Goldschmidt⁴ and Adams³ that the beta crystallin of the lens is capable of acting as this thermostable residue A further argument in favor of the importance of glutathione is the fact that in cataract there is little or no glutathione remaining in the lens That this diminution is not secondary to the occurrence of the opacity of the lens but is rather a part of the cataractous process itself is indicated by the observation of two of us⁵ that the glutathione in the lens of an animal on a diet high in galactose may be markedly diminished before the appearance of any opacity

From the Department of Physiological Chemistry and the Department of Ophthalmology, Northwestern University Medical School

1 Hopkins, F G Biochem J **15** 286, 1920

2 Bellows, J G, and Rosner, L Biochemistry of the Lens Preparation of Glutathione from Crystalline Lens, Arch Ophth **16** 1001 (Dec) 1936

3 Adams, D R Proc Roy Soc, London, s B **98** 244, 1925

4 Goldschmidt, M Arch f Ophth **113** 160, 1924

5 (a) Bellows, J G Biochemistry of the Lens The Influence of Vitamin C and Sulfhydryls on the Production of Galactose Cataract, Arch Ophth **16** 762 (Nov) 1936 (b) Bellows, J G, and Rosner, L Am J Ophth **20** 1109, 1937

On the other hand, some investigators think that the importance of glutathione in the metabolism of the lens has been overestimated Krause⁶ has pointed out that no specific essential function has yet been found for glutathione, but he has suggested that it may have a function in the synthesis of lens protein

In view of the interest centering about this compound, it seemed that further investigation into its variation in concentration under different physiologic conditions was indicated Potentiometric studies^{5b} on the cortex and nucleus of the lens showed a wide variation in potential between its cortical and its nuclear portion Evidence will be presented indicating that this difference is due to a change in the concentration of glutathione A more complete study of the concentration of glutathione in the two portions of the lens was therefore undertaken, particularly with relation to age and to the feeding of galactose

STUDIES ON THE CONCENTRATION OF GLUTATHIONE IN THE LENS WITH RESPECT TO ITS VARIATION WITH AGE

Rats from 1 day to 14 months old were used in our experiments The lenses were removed, and their cortical and nuclear portions separated, ground with metaphosphoric acid (2.5 per cent) and centrifuged to remove the precipitated proteins Aliquot portions were used to determine the amount of glutathione and of ascorbic acid present The total reducing power of the lens extract was determined by titration with potassium iodate in acid solution, the reducing power of the ascorbic acid, by titration with Tillman's reagent The difference between the total reducing power and the reduction by ascorbic acid was attributed to glutathione This assumption was shown to be valid by Evans⁷

As seen from table 1, the glutathione concentration of the lens in very young rats is low compared with that of older animals In these experiments the amount of glutathione rose rapidly from about 44 mg per hundred grams at the age of 1 day to about 200 mg three weeks later and reached a peak of 300 mg per hundred grams at the age of 4 months, after which there was a slow decline

In very young animals no differentiation could be made between the cortex and the nucleus of the lens However, in animals 3 weeks of age and over, in which a separation between the central and the peripheral portion of the lens was possible, it was found that the concentration of glutathione was higher in the cortex than in the nucleus This difference increased with age Our studies indicate that the con-

6 Krause, A. C. *The Biochemistry of the Eye*, Baltimore, Johns Hopkins Press, 1934

7 Evans, E. D. *Nature*, London **134** 181, 1934

centration of glutathione in the nucleus of the lens remains relatively constant with aging of the lens, while that of the cortex varies significantly with age

As to the ascorbic acid in the lens, it appears to be subject to a greater amount of fluctuation than does glutathione. Because of its great variability, no conclusion can be drawn at the present time

EFFECT OF GALACTOSE ON THE CONCENTRATION OF GLUTATHIONE IN THE LENS

Since we have previously shown the amount of glutathione to be diminished when galactose is fed, we undertook to determine the influ-

TABLE 1—*Variation in the Concentration of Glutathione and of Ascorbic Acid in the Lens with the Age of the Rat*

Age of Litter	Glutathione, Mg per 100 Gm			Ascorbic Acid, Mg per 100 Gm		
	Total	Cortex	Nucleus	Total	Cortex	Nucleus
1 day	44			9		
3 days	100			10		
10 days	62			15		
10 days	69			57		
17 days	94			14		
17 days	86			12		
21 days	131	153	106	32	32	31
22 days	125			11		
22 days	114	156	82	28	37	23
25 days	184			10		
26 days	200	236	141	11	13	10
2 months	179	371	105	13	14	10
2 months	173	371	82	14	14	13
2 months	172	371	82	13	14	12
4 months	278	497	107	15	19	12
4 months	320	516	133	19	20	18
7 months	207	341	107	12	10	14
7 months	216	339	107	10	10	10
11 months	233	434	61	13	14	12
11 months	193	316	100	13	15	11
14 months	203	285	87	5	5	5

ence of age on the rate and site of this loss, i e., whether from the nucleus or from the cortex

Litters of rats varying in age from 21 days to 14 months were used. The lens of one eye of each animal was removed, the lenses were pooled, and the glutathione and ascorbic acid contents were determined. The animals were then placed on a diet containing 50 per cent galactose for a given period. At the end of this time the glutathione and ascorbic acid contents were determined in the remaining lenses.

From the data in table 2, it is readily observed that the glutathione in the lens decreases in a short time when the animals are fed galactose. This decrease appears to be more marked in the cortical layer (of the lens) than in the nuclear layer. There seems to be no particular change in the rate of loss of glutathione with age. In the older lenses, because of their higher initial content a longer period is required for the glutathione to fall to a given level. A further observation from these

data is that as the level of glutathione drops when galactose is fed the lens retains the remaining glutathione more tenaciously. Thus, during the period covered by these experiments the lowest glutathione value reached was 38 mg per hundred grams (table 3)

To determine whether the glutathione in the lens would return to normal if the animals were replaced on a stock diet, the following experiment was performed. Two litters, one consisting of 6 and another of 8 rats, were used. The normal glutathione values were determined for 2 rats of each litter. The litters were then placed on a galactose diet for four days. The glutathione content in one eye of each remaining rat was determined, and the animals were then placed on a normal stock

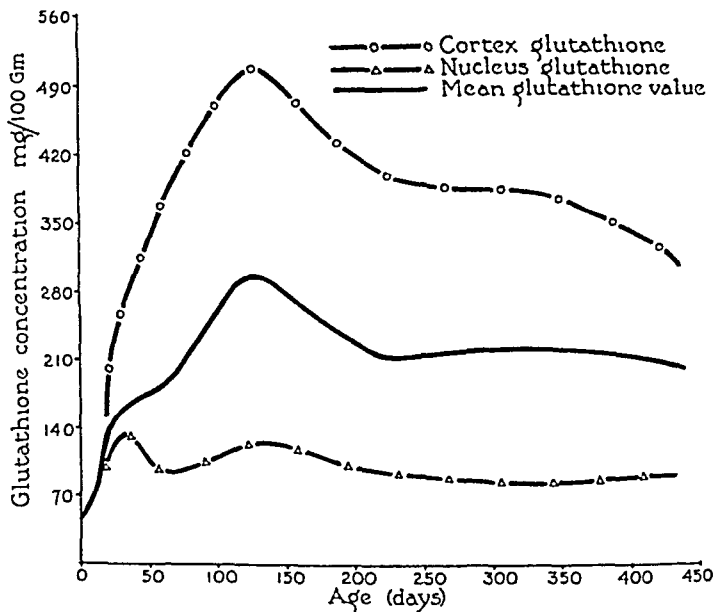


Chart 1—Relation between age and the concentration of glutathione in the lens

TABLE 2—Effect of Galactose Feeding on the Concentration of Glutathione and of Ascorbic Acid in the Lens

Age of Litter	Days on 50% Galactose	Glutathione, Mg per 100 Gm						Ascorbic Acid, Mg per 100 Gm					
		Before Feeding			After Feeding			Before Feeding			After Feeding		
		Total	Cortex	Nucleus	Total	Cortex	Nucleus	Total	Cortex	Nucleus	Total	Cortex	Nucleus
21 days	9	131	153	106	51	32	65	32	32	31	14	14	15
22 days	4	114	156	82	89	116	56	28	37	23	16	16	15
26 days	2	200	236	141	155	152	162	11	13	10	7	6	8
2 mo	2	179	371	105	94	123	69	11	14	10	9	7	10
2 mo	2	173	371	82	95	123	75	14	14	13	10	7	13
2 mo	2	172	371	82	95	123	70	13	14	11	12	7	16
4 mo	3	278	497	107	136	221	56	13	15	11	15	19	12
4 mo	14	320	516	133	47	49	45	19	20	18	10	10	10
7 mo	2	207	314	107	159	244	63	12	10	13	9	8	10
7 mo	2	216	339	107	155	215	82	10	10	10	10	10	10
11 mo	3	233	434	61	146	249	40	13	14	12	18	21	15
11 mo	7	193	316	100	76	128	28	13	15	11	11	9	13
14 mo	4	203	285	87	137	150	114	5	5	5	8	7	10
14 mo	10				51	65	35				7	7	6

diet. The glutathione values for the remaining eyes were determined eleven and seventeen days later, respectively. The results as shown in table 3 reveal that the concentration of glutathione returns to a value at least as high as that of normal animals of the same age. It is of interest to note that the usual marked difference between the values for the cortex and those for the nucleus is absent, the two portions of the lens containing almost similar amounts of glutathione.

A litter of animals with mature galactose cataract and practically no glutathione in the lens of one eye was placed on a normal stock diet for three weeks. At the end of this time one could see normal lens fibers in the periphery of the lens of the remaining eye of each rat by means of the ophthalmoscope. The lenses of these animals contained

TABLE 3—*Effect of Galactose Feeding Followed by a Normal Diet on the Concentration of Glutathione and Ascorbic Acid in the Lens*

Litter	Age, Days	Before Feeding Galactose		Four Days on Galactose		Seventeen Days on Normal Diet			
		Gluta- thione, Mg per 100 Gm	Vitamin C, Mg per 100 Gm	Gluta- thione, Mg per 100 Gm	Vitamin C, Mg per 100 Gm	Glutathione, Mg per 100 Gm		Vitamin C, Mg per 100 Gm	
						Cortex	Nucleus	Cortex	Nucleus
1	25	184	9.8	41	5.7	211	200	15.7	19.5
2	22	125	10.8	38	5.0				
After 11 days on normal diet						251	206	8.4	8.9

TABLE 4—*Concentration of Glutathione in Normal and Cataractous Lenses*

	Glutathione, Mg per 100 Gm	Vitamin C, Mg per 100 Gm
Central cataractous portion of lens	0*	0*
Normal peripheral portion of lens	231.0	6.0

* In this experiment concentrations of glutathione below 20 mg per hundred grams or of vitamin C below 1.5 mg per hundred grams were not measurable.

132 mg of glutathione per hundred grams and 3.5 mg of vitamin C per hundred grams. That glutathione and vitamin C are contained in the normal lens fibers laid down subsequent to the formation of the galactose cataract is shown by the determination of these substances in the cataractous and normal portions of the lenses of another litter of similarly treated rats (table 4).

This difference is strikingly brought out by the application of the nitroprusside test to a cross section of this type of lens. The normal peripheral fibers give a reddish purple hue, whereas the central cataractous portion remains unstained, indicating an absence of sulfhydryls.

THE RELATION BETWEEN GLUTATHIONE AND LENS POTENTIAL

In a previous report we presented data correlating lens potential with age and with the onset of cataract. Our interest was aroused concerning the question of what factors in the lens give rise to the lens potential.

and cause its variation under certain physiologic conditions. We therefore undertook to determine the potentials of certain constituents of the lens and to reproduce the potentials found in the lens.

Before entering into a discussion of this work, we should perhaps define the term potential. When a substance may exist in both an oxidized and a reduced form (such as Fe^{++} and Fe^{+++}), a solution containing this substance sets up an electrical potential at an inert electrode. This potential is a function of the relative amounts of the oxidized and the reduced form of the substance present, becoming more positive with an increase in the oxidized form and vice versa. This phenomenon is termed an oxidation-reduction potential and represents the tendency of the solution to deposit electrons on or to remove electrons from the electrode. Not all substances which exist in reduced and oxidized forms give such characteristic potentials. Glutathione, for

TABLE 5—*Relation of the Potential to the Concentration of Beta Crystallin, Glutathione and Ascorbic Acid in a Mixture at pH 7.30*

Substance	Concentration, Mg. per 100 Gm.	Approximate Ratio of Concentration to That Found in the Lens	Potential, Millivolts
Beta crystallin	200.0	1.85	156
Glutathione	17.4	1.7	-13*
Ascorbic acid	6.0	1.4	-11†

* The potential of glutathione plus beta crystallin

† The potential of glutathione and beta crystallin plus ascorbic acid

example, appears to give a potential dependent only on concentration of the reduced form. The significance of this phenomenon is still debatable.

In all of our work a gold electrode was used rather than a platinum electrode, as the former seemed to arrive at equilibrium with its surrounding medium more rapidly. It was found impossible to reproduce potentials when the system was in contact with air. A steady stream of purified nitrogen was therefore passed through the solution. This also served to keep the medium well mixed. All determinations were performed in solutions buffered with phosphates to pH 7.3.

Considering concentration and potential activity, it appeared that of the crystalloids only glutathione and ascorbic acid might significantly contribute to the potential. No work of this nature has previously been done with proteins, but it was reasoned that the main potential-giving group of proteins is probably the $-\text{SH}$ group. From this point of view, beta crystallin should be potentially quite active, alpha crystallin slightly active and albuminoid active hardly at all. This line of reasoning was proved valid by experiment. The potential of a solution of alpha crystallin was determined, and beta crystallin was then added, the potential changed to the normal potential of beta crystallin. The addition of

alpha crystallin to beta crystallin produced no change in the potential. The same was true for albuminoid.

An attempt was then made to determine whether ascorbic acid, from the point of view of its small concentration in relation to glutathione and beta crystallin, could contribute significantly to the lens potential.

Table 5 illustrates the significance of beta crystallin, glutathione and ascorbic acid so far as they contribute to the potential in the lens. In this experiment each of these substances was added in the order named. Ascorbic acid, even when present in a greater quantity than either of the other two constituents, as compared to its concentration in the lens, produced no significant change in the potential given by the mixture of

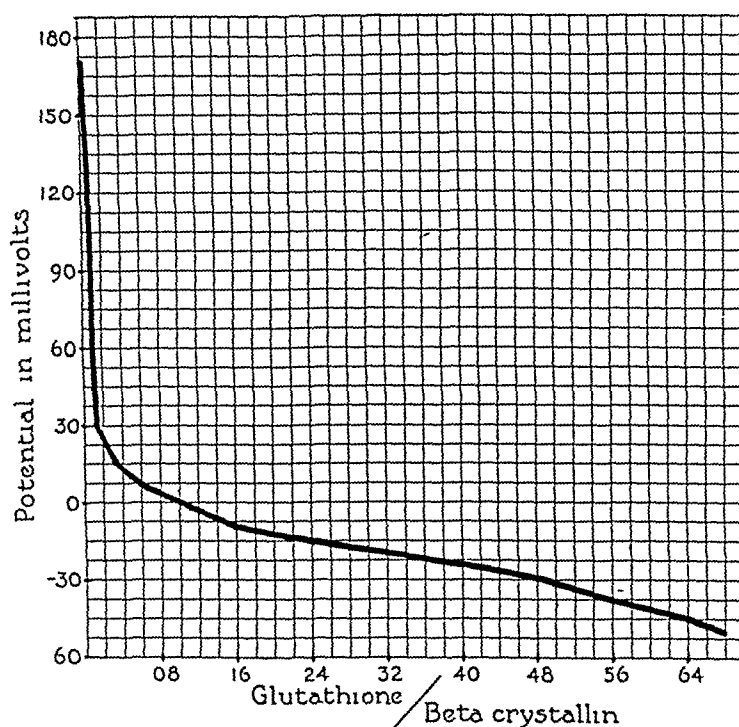


Chart 2—Effect of the glutathione-beta crystallin ratio on the potential

TABLE 6—Relation of the Potential to the Relative Concentrations of Glutathione and Beta Crystallin at pH 7.30

Concentration, Mg per 100 Gm		Glutathione- Beta Crystallin Ratio	Potential, Millivolts
Glutathione	Beta Crystallin		
197.4	300	0.66	-30
54.5	100	0.54	-36
47.4	200	0.24	-13
54.5	500	0.11	-3
30.0	500	0.06	-6
14.5	300	0.029	-15
3.4	320	0.010	-31
4.0	300	0.008	-42
2.0	300	0.004	-140
0.0	500		-170

beta crystallin and glutathione. Ascorbic acid was therefore ruled out as a factor contributing to the lens potential.

It is apparent, then, that the lens potential is determined by the relative concentrations of glutathione and beta crystallin in the lens and should be capable of duplication by *in vitro* mixtures of these two substances. A series of mixtures containing varying quantities of glutathione and beta crystallin were therefore prepared and their potentials determined.

Table 6 illustrates how the potential varies with variation of the ratio between concentrations of glutathione and beta crystallin. It may be noted that as the concentration of glutathione rises with respect to that of beta crystallin the potential falls. Chart 2 shows this relation strikingly. From this curve can, conversely, be estimated the ratio of glutathione to beta crystallin if the potential of the mixture is known.

COMMENT

Our findings indicate that the glutathione content of the crystalline lens at birth approximates that of blood. This observation is in agreement with the fact that in embryonal life the lens has a blood supply and in consequence has no more need for a special respiratory mechanism than has any other tissue. However, in postnatal life this blood supply is no longer available, necessitating a substitute mechanism. That glutathione takes part in this respiratory function was first indicated by Goldschmidt. Apparently in postnatal life some mechanism is set in motion (probably in the lens) that stimulates the production of glutathione. Thus there occurs a sharp rise in the glutathione content in the first few days of life to meet the increasing metabolic and growth demands of the lens. Because of the continuous requirements of the lens during its period of greatest physical development, the amount of glutathione continues to increase until the fourth month of life, after which it declines slowly.

It would seem that the concentration of glutathione in the nucleus shows no marked variation with age, the major change taking place in the cortical portion of the lens. This observation is in agreement with the fact that the nucleus of the lens maintains a sluggish metabolism as compared to the cortex. Where there is no defined nucleus, as in the very young animal, there is no large difference in the concentration of glutathione in the inner and the outer layers of the lens. This may be expected, since the lens fibers are all young with no great variation in age.

When animals are fed galactose, the glutathione in the lenses diminishes. We found that a longer time is necessary for the lenses of older animals to become depleted of this substance, but this appears

to be due to its initial higher value in the older lenses and not to a difference in rate of loss. The loss of glutathione in the cortex is rapid at the outset and becomes slower as feeding is continued. However, in the nucleus in which the concentration is initially low, glutathione is lost slowly. This difference in rate of loss can perhaps be explained as due either to the greater exposure of the cortex to external influences or to the difference in the metabolic activity of the two portions. From the latter point of view the respiratory activity of the cortex should diminish and approach that of the nucleus after several days of feeding with galactose. Measurement of the comparative oxygen uptake of the cortex and the nucleus before and after the feeding of galactose would be necessary to prove this point.

Although in young animals the appearance of cataract shortly follows the loss of glutathione, in older animals such a loss may occur long before the appearance of opacities. Any attempt at an explanation of this observation must involve discussion of the cause of glutathione depletion after the feeding of galactose. Such a loss may be explained on the basis of the phenomenon observed by two of us⁸ that the permeability of the capsule of the lens decreases in the presence of galactose. It is conceivable that this decreased permeability upsets the metabolism of the lens either by preventing passage of nutrients into the lens or by inhibiting the escape of waste products from the lens. This upset in the metabolism of the lens might interfere with the production of glutathione or increase its rate of breakdown. It has been shown by Friedenwald⁹ and by Gifford and his co-workers¹⁰ that with increasing age there occurs a decreased permeability of the capsule of the lens. Since this diminution occurs slowly, the lens may become adapted to this decreased permeability and be therefore not so sensitive to further decrease. Furthermore, the smaller metabolic activity of the older lens may make it more insensitive to external influences.

It appears from our data regarding the return of glutathione to the lenses of rats placed on a normal diet after a period on a galactose diet that the lens retains its ability to recover from incipient cataract. This recovery includes a return of normal transparency to the lens fibers as observed by the ophthalmoscope and a regaining of its normal concentration of glutathione. Even after the occurrence of mature cataract, the lens can still lay down normal lens fibers in the periphery. The

8 Bellows, J. G., and Rosner, L. *Biochemistry of the Lens*. XI. The Effect of Galactose on the Permeability of the Capsule of the Lens, *Arch. Ophth.* **20**: 80 (July) 1938.

9 Friedenwald, J. S. Permeability of the Lens Capsule to Water, Dextrose and Other Sugars, *Arch. Ophth.* **4**: 350 (Sept.) 1930.

10 Gifford, S. R., Lebensohn, J. E., and Puntenny, I. S. *Biochemistry of the Lens*. The Permeability of the Capsule of the Lens, *Arch. Ophth.* **8**: 414 (Sept.) 1932.

opaque portion, however, remains a dead tissue, being unable to recover its transparency of glutathione

We have previously presented studies dealing with variations in the potential of the cortex and nucleus of the lens under different physiologic conditions. The average potential in the cortex of the lens of young rats was found to be 28 millivolts, while that in the nucleus was 40 millivolts. In old rats, however, the cortex potential was 7 millivolts and the nucleus potential 132 millivolts. After a period of feeding the rats galactose the potential in the lenses of young rats rose to 40 and 150 millivolts in the cortex and nucleus, respectively, in the old rats the potential correspondingly rose to 42 and 153 millivolts. These results led us to attempt to find the origin of the lens potential and the factor or factors causing its variation under the physiologic circumstances mentioned. Thus, to glutathione and beta crystallin were eventually attached responsibility for the lens potential and its variations, and a curve was drawn showing the relation between the

TABLE 7—*Comparison of Observed and Theoretic Potential in the Cortex and Nucleus*

	Cortex		Nucleus	
	Observed Potential, Millivolts	Theoretic Potential, Millivolts	Observed Potential, Millivolts	Theoretic Potential, Millivolts
Young rats	28	24	40	90
Old rats	7	20	132	120

glutathione-beta crystallin ratio and the resulting potential. If it is assumed that the beta crystallin content of the lens remains constant at 17 per cent (Krause's figure, which we may accept as a working basis), theoretic values can be obtained from the glutathione-beta crystallin ratio by interpolating in chart 2. A comparison between potentials determined with in vitro mixtures of glutathione and beta crystallin and those actually found in lenses with corresponding relative amounts of the two substances is shown in table 7. The actual and theoretic values show a good correlation in all instances except those for the nucleus of young rats. It may be pointed out that the portion of the curve concerned with this glutathione-beta crystallin ratio undergoes a marked change in potential with a slight change in this ratio. Thus, only a small error produces large variations in potentials.

This sharp change in potential with small variations in the concentration of glutathione (beta crystallin remaining constant) might in fact be utilized as a more sensitive method for the estimation of glutathione than actual chemical determination when this substance is present in small quantities.

RETINITIS PROLIFERANS

CLINICAL AND HISTOLOGIC STUDIES

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CHICAGO

The term retinitis proliferans is purely descriptive and applies to a variety of pathologic conditions due to different causes. The expression retinitis designates a pathologic process in the retina, not necessarily of an inflammatory nature, and the adjective proliferans indicates the plastic character of the lesions.

Clinical and histologic studies of eyes during the earlier stages of proliferating retinitis reveal that there are two principles involved in the production of this condition, leading to two essentially different types of retinitis proliferans: type 1, in which the formation of connective tissue precedes the formation of new blood vessels, and type 2, in which there is a primary formation of new vessels with secondary production of a scaffolding of delicate connective tissue.

In type 1 exudation or hemorrhage from the retina into the vitreous, caused by inflammatory or traumatic alterations of the retinal vessels, is the primary event, and organization of the extravasation leads to the formation of the well known strands and membranes.

The group of conditions producing this type of proliferating retinitis includes (1) tuberculous retinal periphlebitis or recurrent hemorrhages into the vitreous in juvenile subjects, (2) neuroretinitis papulosa of the secondary stage of syphilis, (3) all injuries of the retinal vessels followed by hemorrhage and (4) secondary involvement of the inner retinal surface in severe cases of uveal inflammation, as recently described clinically by Arnold Knapp¹. In the last-mentioned condition exudate originating elsewhere in the eye is deposited on the inner retinal surface and there may produce alterations, which lead finally to partial organization of the deposit. These alterations will be discussed in a future publication.

In type 2 the main factor in the production of the plastic lesions is a slow circulatory impairment due to degenerative vascular disease with the formation of new compensating anastomotic channels, with or without hemorrhages at first. The retinitis is produced mainly by a disturbance of the venous circulation due to sclerotic changes of neighboring structures and preexisting unfavorable topographic anatomic relationship.

¹ Knapp A. Formation of Preretinal Connective Tissue in the Vitreous in Acute Choroiditis. Report of Three Cases. Arch Ophth 18:558 (Oct) 1937.

This type of proliferating retinitis is often found in association with (1) diabetes, (2) syphilis of the tertiary stage with predominating endarteritis or (3) arteriosclerosis, including forms in which there is occlusion of the central retinal vein.

To illustrate the two types of retinitis proliferans, several eyes are described in which typical examples of each type were present.

Clinical examination of a patient 43 years of age, with a healed neuroretinitis papulosa of the right eye and reduction of vision in the affected eye to perception of hand movements, revealed proliferating retinitis of type 1. The Wassermann reaction of the blood, which had never been tested before, was strongly positive. The results of other examinations were irrelevant, and the other eye was normal. The optic disk in the right fundus was ill defined, pale and partly covered with connective tissue, indicative of postneuritic atrophy of the optic nerve. Connective tissue also accompanied some of the large retinal veins, and fanlike structures spread over several old chorioretinitic lesions, connective tissue was found in all those places where the inflammatory process had been located, namely, at the site of the papular lesions, along the retinal veins and on the nerve head, thus indicating the characteristic trio of neuritis, periphlebitis and disseminated chorioretinitis, which was first described by A. Fuchs,² who also named this condition occurring in the secondary papular stage of syphilis, neuroretinitis papulosa.

The connective tissue forming these strands and membranes was dense and white and did not contain visible newly formed blood vessels (fig. 1).

For histologic demonstration of the first type of retinitis proliferans, two eyes with tuberculous retinal periphlebitis, one in the early and one in the late stage, appeared suitable. The first stage of this disease is well known clinically and histologically. The investigations of Axenfeld,³ Fleischer⁴ and Wolf⁵ are responsible for the knowledge of its tuberculous nature, however, the appearance is more that of non-specific, proliferative inflammatory changes on an allergic basis than of typical tuberculous granulation tissue. The disease manifests itself

2 Fuchs, A. Ueber einige seltene luetische Erkrankungen des Auges, *Ztschr f Augenh* **59** 213, 1926.

3 Axenfeld, T., and Stock, W. Ueber die Bedeutung der Tuberkulose in der Aetiologie der intraokularen Hamorrhagien, und der proliferierenden Veränderungen in der Netzhaut, besonders über Periphlebitis retinalis bei Tuberkulosen, *Klin Monatsbl f Augenh* **49** 28, 1911.

4 Fleischer, B. Die juvenile Periphlebitis retinae, mit ihren Folgeerscheinungen, eine echte Gefasstuberkulose der Netzhaut, *Klin Monatsbl f Augenh* **17** 769, 1914.

5 Wolf, H. Zur Angiopathia juvenilis mit Bemerkungen über den Bau und das Wesen der Limitans interna retinae, *Arch f Augenh* **89** 54, 1921.

first in the lymph spaces around the venous branches. Dense round cell infiltration distends the perivascular lymph spaces, thus forming nodules and spindle-shaped thickenings of the veins, which gradually encroach on and destroy the walls of the veins. The resulting intra-retinal and preretinal hemorrhages and exudates are partly absorbed and partly organized. When the hemorrhages are extensive, and particularly when they are recurrent, capillaries but rarely large blood vessels grow into the organizing extravasation.

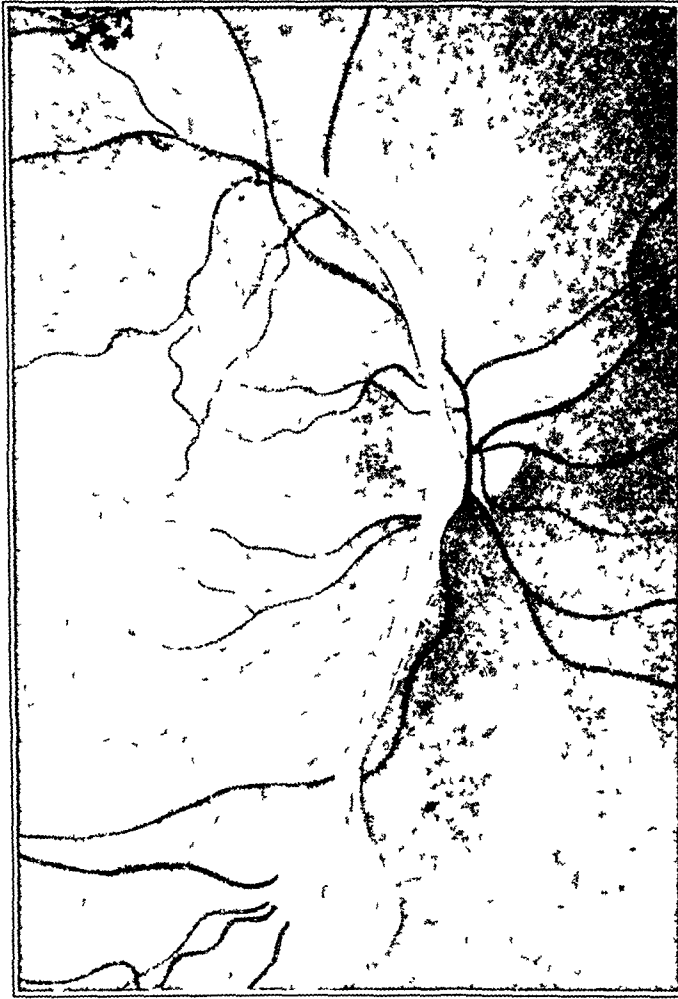


Fig 1—Retinitis proliferans of type 1 associated with neuroretinitis papulosa

In figure 2 such an advanced stage of recurring hemorrhages in the vitreous of a juvenile subject is shown. The retina and optic nerve are covered with a dense layer of connective tissue. This thick membrane is rich in cells and capillaries, and the older layers show stratification.

Clinical examination of a diabetic patient aged 46 revealed retinitis proliferans of type 2. The patient had been under observation in the ophthalmic clinic of Rush Medical College for a number of years, and the earliest stage of proliferating retinitis was observed in the

better eye When first seen, this patient had been on diabetic management for four years, and dietary measures sufficed to eliminate glycosuria The blood pressure was 134 systolic and 66 diastolic Vision in the right eye was first noted to fail three years prior to the first examination, while that in the left eye was first noted to fail three weeks prior to this examination, at which time the corrected vision in the right eye was 0.1 and that in the left eye was 0.4

In the right fundus there was a large fan-shaped membrane (fig. 3) of delicate semitransparent connective tissue, which contained a great number of large and small newly formed blood vessels in a brushlike

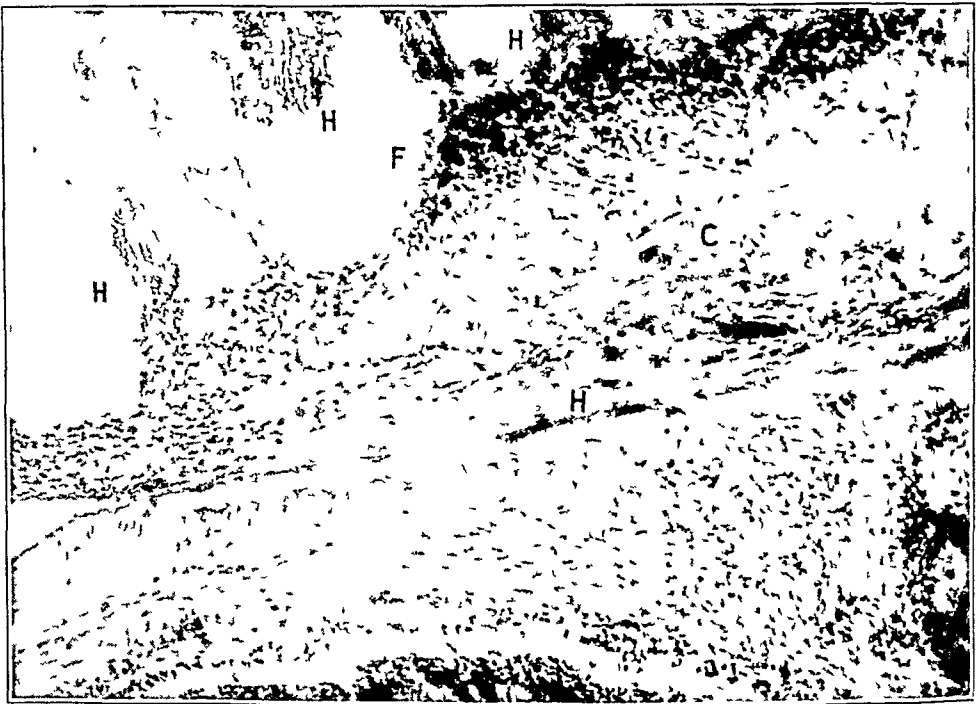


Fig. 2—Retinitis proliferans of type 1 associated with a late stage of tuberculous periphlebitis. *H* indicates free hemorrhages in the vitreous, *F*, fibroblasts, and *C*, capillaries

arrangement The membrane completely covered the optic disk, where it appeared to have its base Along the large retinal vessels and in the macular area there were numerous fatty degenerative retinal lesions and hemorrhages, mostly of the small capillary type

In the left fundus (fig. 4) a small brushlike arrangement of newly formed vessels, arising from the inferior nasal vein near the border of the optic disk, proceeded straight forward into the vitreous There was no noticeable connective tissue between these blood vessels at first There was only mild retinal angiosclerosis visible in each eye During the following six months of observation the brushlike arrangement of

newly formed vessels became larger (fig 5), and delicate grayish connective tissue became visible between the vessels. The proliferating retinitis in this eye began to resemble that of the right eye (fig 3).

This type of retinitis proliferans was also seen clinically in the left eye (fig 6) of a 64 year old woman, who came to the ophthalmic clinic of the Billings Memorial Hospital, complaining of failing vision in the left eye of four years' duration. The corrected vision in the right eye was 0.8 and that in the left eye was 0.6—3. There was moderate bilateral retinal angiosclerosis with obliterating endarteritis evident in some small branches in each eye. In the left fundus several

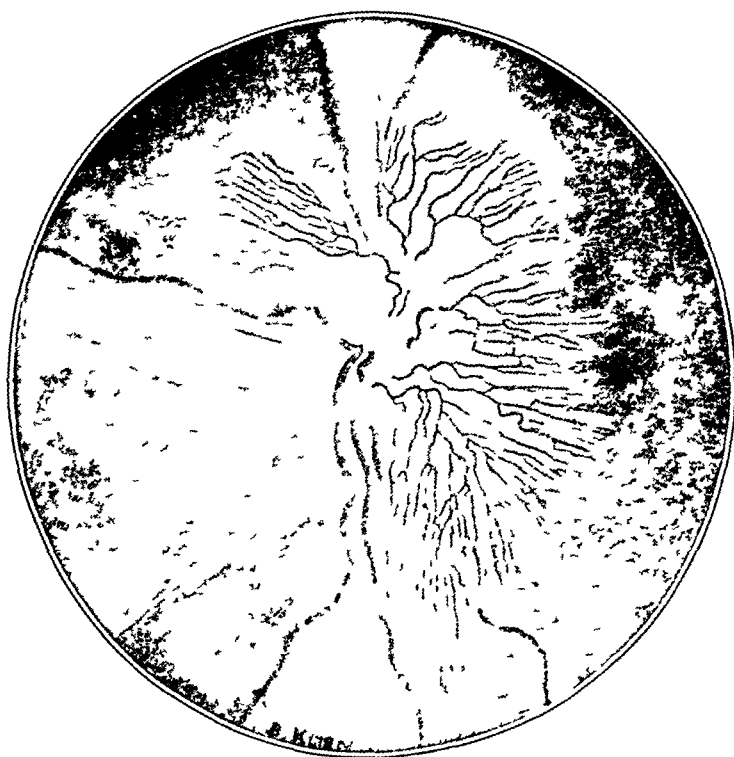


Fig 3—Advanced retinitis proliferans of type 2 associated with diabetes

brushlike arrangements of new vessels, surrounded by delicate connective tissue and ending with coil-like convolutions in the vitreous, were attached to the inferior nasal quadrant of the disk.

Medical examination revealed moderate arterial hypertension of the benign essential type and no other pathologic changes.

The predominating characteristic of the proliferating retinitis in these eyes was the great number of newly formed vessels, which were partly present in a brushlike arrangement, especially in the initial stage and originated from the nerve head. The connective tissue between had a delicate semitransparent appearance and thus a less white color than that in the eyes previously studied.

It is difficult to illustrate histologically the early stage of the second type of retinitis proliferans but it is possible to distinguish its char-

acteristics in more advanced conditions. The first eye showing this type of retinitis proliferans to be considered here was the left eye of a diabetic patient 63 years old, who had at the time of the enucleation a blood pressure of 210 systolic and 120 diastolic. The eye was enucleated ten weeks after obstruction of the central retinal vein and eight weeks after onset of the secondary glaucoma. Examination of the fundi a year and a half before revealed marked retinal angiosclerosis, numerous, mostly capillary hemorrhages scattered over both posterior polar regions and groups of fatty degenerative lesions around the left disk and macula. Both disks were normal.

Most striking was the histologic appearance of the retinal arteries. The central artery and its immediate branches were filled almost com-

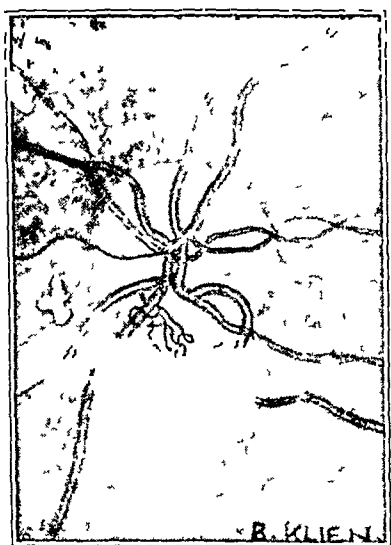


Figure 4

Fig 4—Very incipient retinitis proliferans of type 2 associated with diabetes



Figure 5

Fig 5—The same eye shown in figure 4 six months later

pletely with endothelial and subendothelial proliferation. This, together with some elastic lamellation and considerable thickening of the tunica media and the perivascular sheaths, transformed the central artery into a rigid tube, which compressed the central vein within the lamina cribrosa. There were large intraretinal hemorrhages along the course of the engorged veins. The lower part of the optic nerve and the adjoining retina were covered with a delicate membrane, which contained newly formed, thin-walled blood vessels in cross and longitudinal sections, some of them of large caliber (fig 7). The framework of the membrane around these vessels was extremely delicate, consisting of a few fibroblasts, wandering cells and a faintly staining, almost homogeneous transudate. These newly formed structures were on the inside of the internal limiting membrane.

The place where the internal limiting membrane is perforated by the newly formed blood vessels can usually be found in serial sections (fig 8). Only when the attempts at anastomosis originate from the nerve head the newly formed vessels do not have to perforate the internal limiting membrane and thus have a more straightforward course, projecting into the vitreous rather than creeping along the inner surface of the retina.

The considerable size of the new blood vessels and the marked degree of retinal angiosclerosis in this case, which had caused considerable arteriovenous compression even a year and a half prior to the

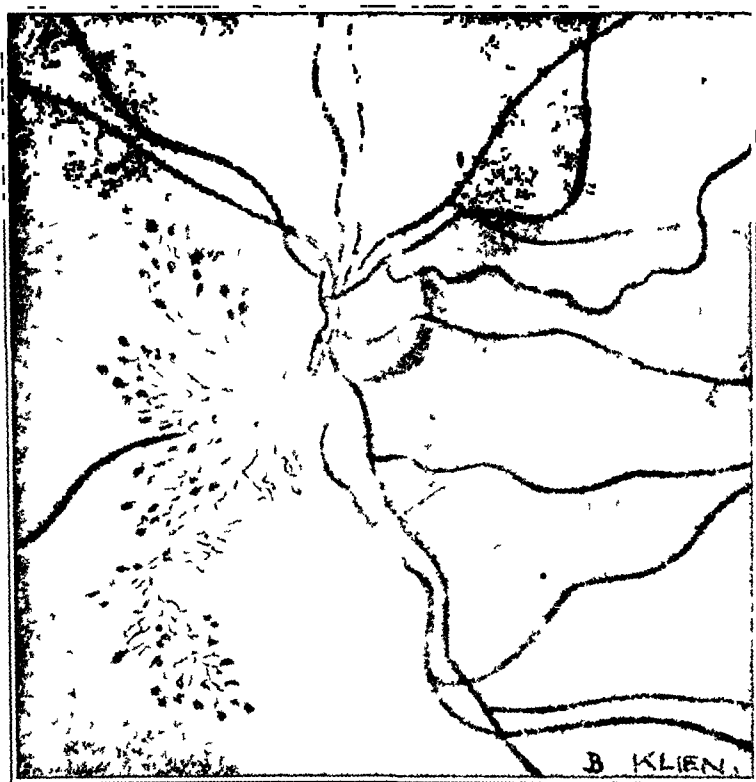


Fig 6—Retinitis proliferans of type 2 associated with arteriosclerosis

occlusion of the central vein, make it probable that the proliferating processes were well under way before the complete venous obstruction, which occurred only ten weeks prior to enucleation.

The second eye showing retinitis proliferans of type 2 was the left eye of a 68 year old woman which was enucleated on account of secondary glaucoma two years after occlusion of the central retinal vein.

The optic nerve was deeply excavated and there was a dense network of tortuous, partly preformed but dilated and partly newly formed veins in the retrolaminar and prelaminar regions. From some of the veins in the prelaminar region there arose branches, which could be followed far into the vitreous, where some of them split up into a brushlike arrangement of smaller vessels (fig 9), others took on a

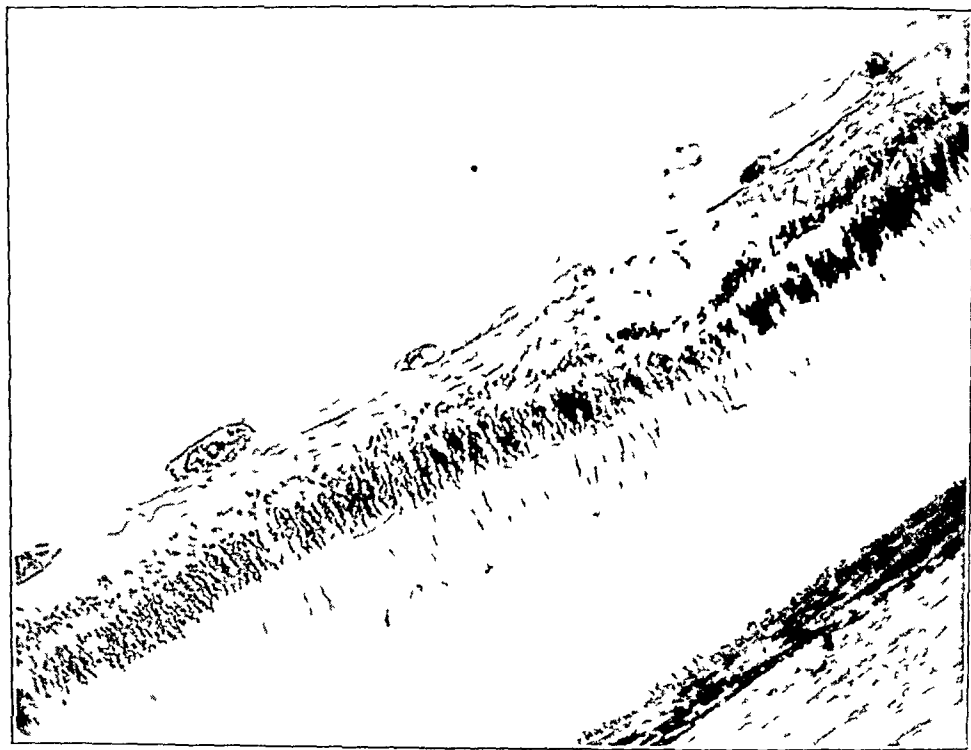


Fig 7—Earlier stage of retinitis proliferans of type 2. Large newly formed blood vessels and delicate connective tissue on the inside of the retina are present.

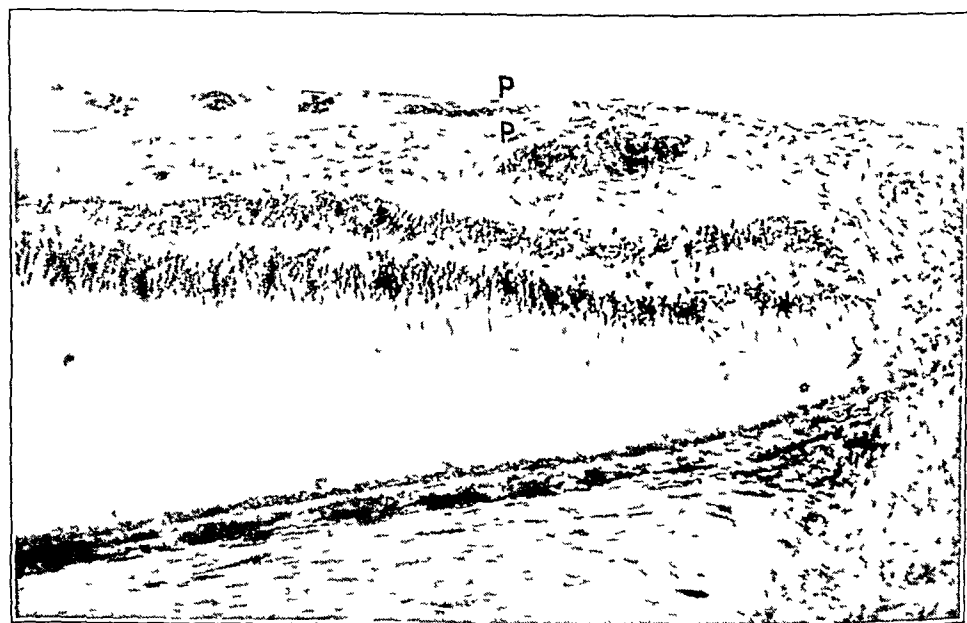


Fig 8—Section of the same eye as shown in figure 7. The internal limiting membrane is perforated by newly formed vessels. P indicates the point of perforation.

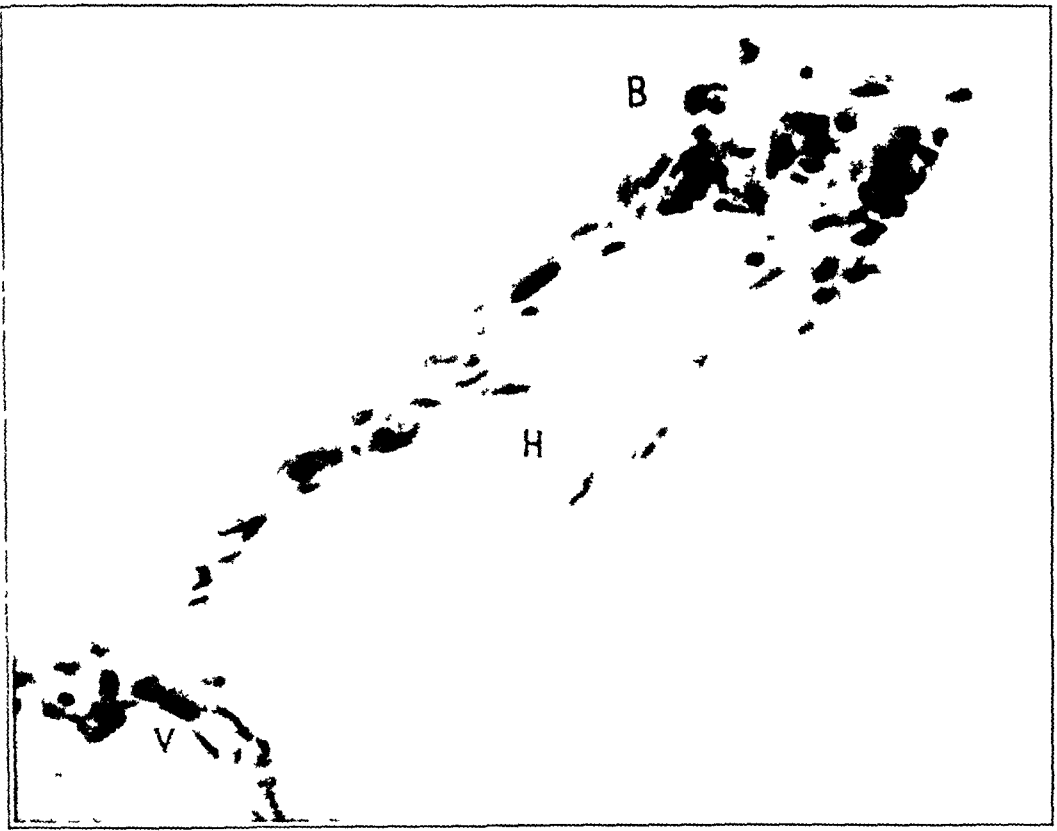


Fig 9—Late stage of retinitis proliferans of type 2 The blood vessel in the vitreous is split up into a brushlike arrangement of small vessels *V* indicates a vein in the nerve head, *H*, hyaline degeneration of connective tissue, and *B*, a brushlike arrangement of new capillaries

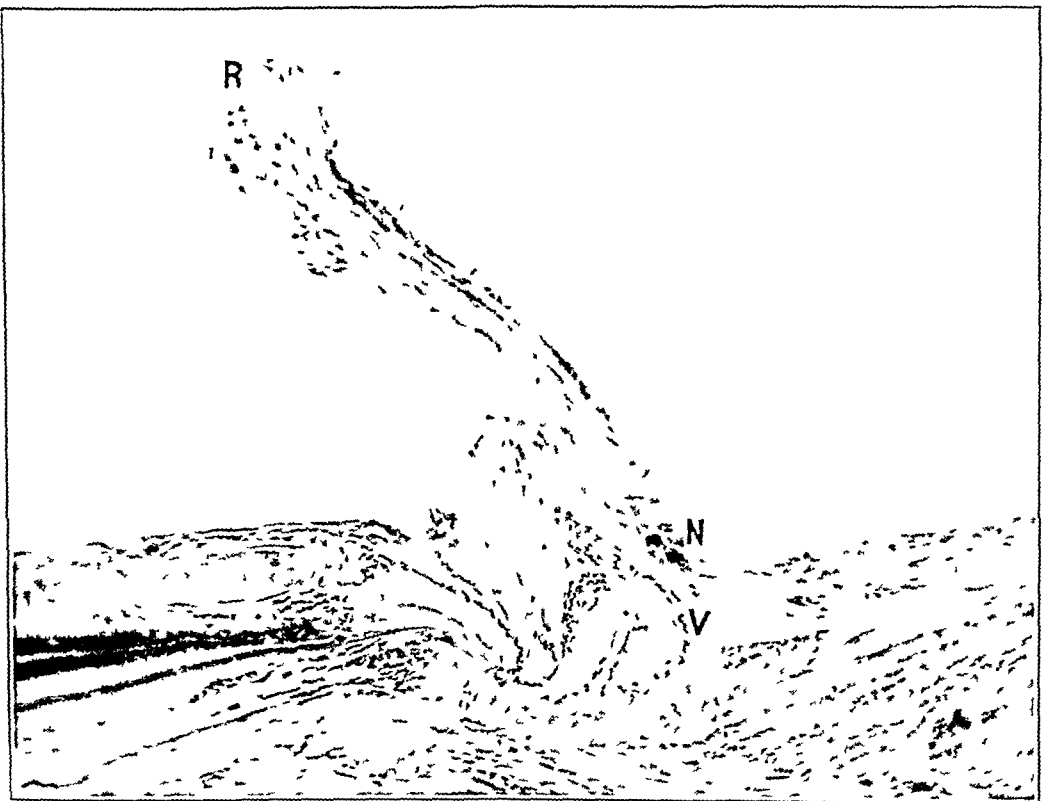


Fig 10—Late stage of retinitis proliferans of type 2 Large newly formed blood vessels are seen in the vitreous with a recurrent course *V* indicates a dilated and tortuous vein in the nerve head, *R*, a recurrent newly formed vein in the vitreous, and *N*, neuroglia

recurrent course, running backward again (fig 10) Free hemorrhages around some of these thin-walled vessels were in the process of organization

In this late stage the framework of the strands carrying the new vessels was dense, consisting of proliferated fibroblasts, neuroglia and a dense homogeneous material, which was shown to be hyaline by the Van Gieson stain But even in this late stage, the amount of connective tissue was negligible compared with the amount of newly formed blood vessels

SUMMARY AND CONCLUSION

On the basis of clinical and histologic studies it is possible to distinguish two types of retinitis proliferans and to deduce, in a not too advanced stage, from the location and appearance of the plastic lesions the nature of the primary disturbance

The lesions of the first type are characterized as follows

- 1 They may be located anywhere in the fundus
- 2 They consist of dense, opaque, white or grayish-white membranes
- 3 They contain no newly formed blood vessels, or only a limited number, which are rarely of large size and are seen only in the advanced stage of the disease
- 4 Histologically, the connective tissue is rich in cells and not infrequently has a stratified appearance
- 5 Inflammatory or traumatic alterations of the retinal blood vessels are the primary cause

The lesions of the second type are characterized as follows

- 1 They originate at or near the optic disk
- 2 There is a profusion of newly formed blood vessels, some of considerable size, which appear in advance of any noticeable amount of connective tissue They often show a bushlike arrangement, and the endings in the vitreous form either coil-like convolutions or loops, returning to the disk
- 3 The connective tissue between these vessels is delicate and semi-transparent
- 4 Histologically, numbers of new veins can be traced to branches of the central vein in or near the nerve head In the earlier stages there is a delicate network of fibroblasts within a homogeneous transudate between them Later, fibroblasts and neuroglia proliferate within these strands, and hemorrhages from the thin-walled new vessels become organized and give them a more substantial appearance
- 5 Degenerative vascular disease leading to circulatory impairment and venous anastomosis is the etiologic factor

STUDIES OF THE VISUAL FIELDS IN CASES OF VERIFIED TUMOR OF THE BRAIN

DANIEL KRAVITZ, M D

BROOKLYN

The value of studies of the visual fields as an aid in the diagnosis of obscure conditions of the eye and brain has long been known MacKenzie,¹ in his textbook on ophthalmology, published in 1854, called attention to the importance of hemianopic defects in the visual fields in the localization of diseases of the brain About this time the ophthalmoscope made its appearance, and ophthalmology was placed on a firm scientific basis

The importance of perimetry in the study of lesions that were not visible with the ophthalmoscope immediately became apparent Troncoso² stated that von Graefe stressed this fact in 1856 Wilbrand³ in 1881 reported a large number of cases and stressed the importance of examinations of the visual fields as an aid in localizing intracranial disorders He probably was the first to see the value of using small test objects in bringing out defects that had been missed by the cruder methods then in use In 1882 Berry⁴ foresaw the importance of studies of the visual field in the diagnosis and prognosis of diseases of intracranial origin

It was not, however, until Bjerrum and Ronne⁵ did their epoch-making work on glaucoma in the early part of the twentieth century that the modern method of perimetry came into being This method, applied by Cushing⁶ and his co-workers, Heuer and Walker,⁷ in analyzing defects due to chiasmal syndromes, rapidly advanced the

Read before the Brooklyn Neurological Society, Dec 22, 1937

1 MacKenzie, W A Practical Treatise on the Diseases of the Eye, ed 4, London, Longman [and others], 1854

2 Troncoso, M V Internal Diseases of the Eye, Philadelphia, F A Davis Company, 1937, p 81

3 Wilbrand, H Ueber Hemianopsie und ihr Verhältniss zur topischen Diagnose der Gehirnkrankheiten, Berlin, A Hirschwald, 1881, p 197

4 Berry, G W On Central Amblyopia, Ophth Hosp Rep, London **10** 44, 1880

5 Ronne, H Ueber das Gesichtsfeld beim Glaukom, Klin Monatsbl f Augenh **47** 12, 1909, numerous later publications

6 Cushing, H The Pituitary Body and Its Disorders, Philadelphia, J B Lippincott Company, 1912

7 Walker, C B Some New Instruments for Measuring Visual-Field Defects, Arch Ophth **42** 577, 1913

knowledge concerning defects in the visual fields in relation to tumor of the brain. Perimetric technic has been further refined by Evans,⁸ so that minute circulatory defects can be detected.

There have been a great number of papers and books on the value of perimetric study in the diagnosis of intracranial tumors. It would therefore seem that one should apologize for presenting a subject so thoroughly discussed. However, as will be shown even by the small series of cases presented here, through neglect tumors of the brain are still allowed to progress until the tumor becomes inoperable or blindness supervenes.

In this paper are reports of 23 cases of verified tumors of the brain. All but 2 of the patients were seen by me preoperatively. One was seen prior to a second operation and the other after removal of the tumor. The reports of both of these cases are included because of

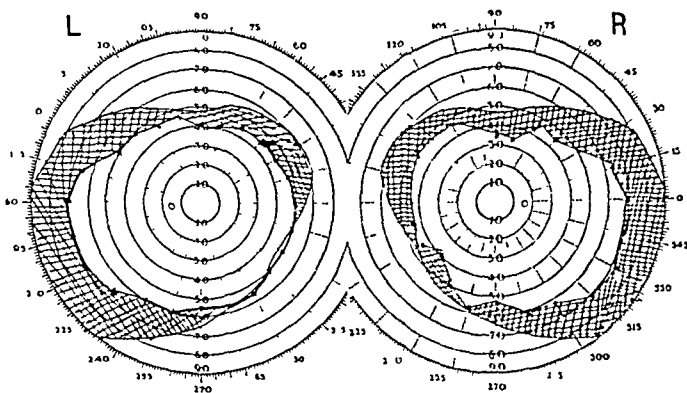


Fig 1 (case 1) —Visual fields of F S, taken on May 7, 1932, with a 2 mm white test object at 330 mm

their great clinical interest. An attempt will be made to compare the value of visual examinations of the fields with the other methods used in the diagnosis of tumor of the brain.

GROUP 1 TUMORS OF FRONTAL LOBE

CASE 1—F S, a man aged 34, was seen on May 2, 1932, with a history of severe pains in the forehead and vomiting for the past seven weeks. Five weeks before he went to an otolaryngologist, who advised removal of a deviated septum. As relief was not obtained by this procedure tonsillectomy was advised.

Vision was 20/30 in the right eye and 20/25 in the left eye. Improvement was not obtained with glasses. There was no nystagmus or ocular palsy. The right disk was swollen about 2.5 D, and there were many hemorrhages around and for some distance away from the disk. The left disk showed only 1 D of swelling and appeared somewhat atrophic. The visual fields on several examinations showed an increasing concentric contraction, without localizing signs. There were no personality changes.

In view of these findings, it was felt that the patient had a tumor of the frontal lobe, and he was referred to the Brooklyn Hospital, where the following observations were made by Dr E J Browder. The patient had slight difficulty in walking, with a tendency to stumble, although he never fell. There were no character changes or forced grasping. On May 11, while at the hospital, he had an attack of mental confusion and was found with his feet on the window sill, as if to climb out. Later, he remembered nothing of this episode. This was thought to be characteristic of a tumor of the frontal lobe.

On June 19 a bone flap was elevated in the right frontal region and a cyst, the size of a golf ball, was found in the right frontal lobe. It was evacuated, and the walls were cauterized. Examination proved it to be a glioma.

This case is interesting in that there was atrophy of the optic nerve on the side opposite to the site of the tumor—a paradoxical Foster Kennedy syndrome.

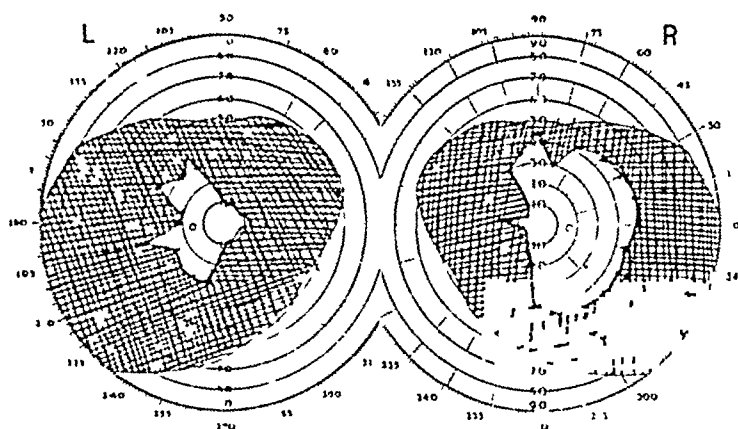


Fig 2 (case 2)—Visual fields of C M, taken on April 4, 1935, with a 10 mm white test object at 250 mm for the left eye and a 2 mm white test object at 250 mm for the right eye.

CASE 2—C M, a woman aged 29, was admitted to the Brooklyn Hospital on April 1, 1935, with a history of intermittent headaches of ten years' duration. For the past six months her vision had become progressively more blurred, and her sense of smell had been lost. She had a constant buzzing in the left ear for the past six months.

Röntgenographic examination on April 2 showed an area of lessened density of the frontal bone which was almost centrally placed a little above the orbits. The sella turcica was not well defined. It was definite that it was enlarged. The floor was depressed, probably actually eroded, and the posterior clinoid processes were eroded. In view of these findings it was thought that a tumor of the sella turcica or of the area around it and a tumor of the frontal bone were present. The growth in the frontal bone was probably metastatic.

Ocular examination on April 4 disclosed no ocular palsy and no nystagmus. The pupils reacted to light and in accommodation. There was 5 or 6 D of swelling of both disks. There were a few hemorrhages around the left disk but none around the right. Vision in the left eye was so poor that a 10 mm test object was the smallest that could be used. This showed a complete loss of the nasal field with retention of a small island in the temporal field. The right visual field

could be plotted with a 2 mm test object and showed a marked concentric contraction with a loss of the nasal field. There was no evidence of secondary atrophy of either disk.

On the basis of the results of the ocular examination, it was thought that the condition was tumor of the left side of the brain, near the chiasm. For the degree of intracranial pressure and the headaches complained of by the patient, she was markedly euphoric and jocular. There was a distinct forward bulge in the middle of the forehead.

The neurologist found no signs of localizing value and was of the opinion that the condition was a tumor of the frontal lobe.

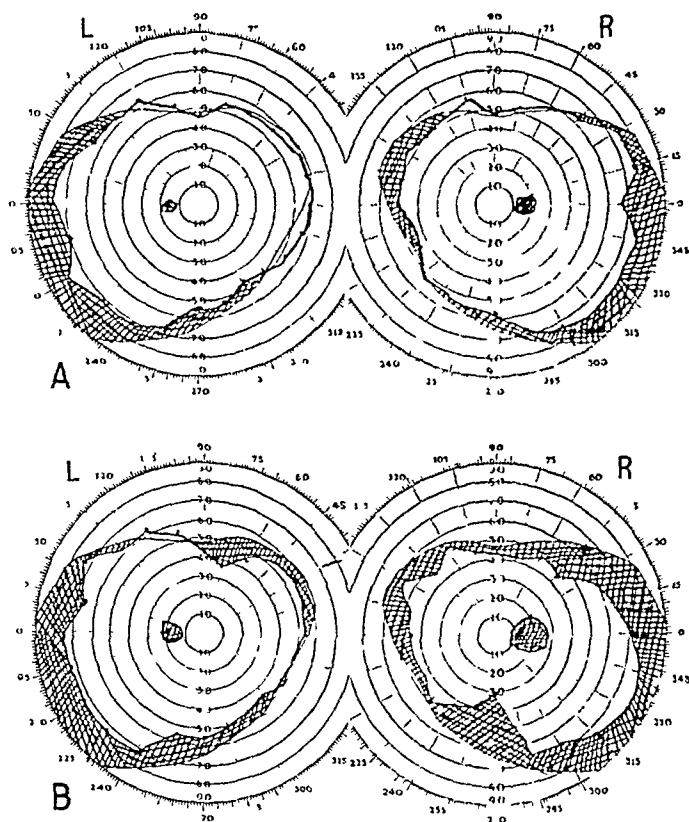


Fig 3 (case 3)—*A*, visual fields of M E, taken on March 9, 1933, with a 1 mm white test object at 250 mm. *B*, visual fields of the same patient, taken on March 16, with a 1 mm white test object at 250 mm. Vision was 13/15 in each eye.

On April 11 Dr E J Browder turned down a bone flap in the right frontal region and disclosed a meningioma, the size of a small orange, occupying the frontal aspect of both anterior cranial fossae. This well defined meningioma of the midline was removed.

The loss of both nasal fields is interesting and can be explained by a flattening of the chiasm against both internal carotid arteries.

CASE 3—M E, a woman aged 36, was admitted to the Brooklyn Hospital on Feb 27, 1933, with a history of blurred vision and headaches for the past seven or

eight months. She has been perfectly well previously. Her illness began with a severe headache, which woke her early in the morning and lasted all day. Since then the headaches had recurred every two or three weeks. They were diffuse all over the head. Her vision had been continuously blurred since the onset. She never had diplopia. Two months before admission to the hospital she began to be annoyed by bad odors and a bad taste in the mouth. At times she would talk incoherently for a few minutes. During these attacks she was conscious but could not recall what she said. She had no dreams, hallucinations or delusions.

Neurologic examination on the day of admission revealed no apparent abnormality in the motor system. Coordination and muscle strength were good. Sensory examination gave negative results, and the cranial nerves were normal except for a questionable weakness of the right facial nerve.

In view of the history of uncinata attacks, asymmetry of the lower right side of the face, an increased knee jerk on the right and ankle jerk on the left and a history of loss of consciousness, it was thought that the patient probably had a tumor of the left temporosphenoid region.

Ocular examination on March 9 disclosed vision of 13/13 in each eye. No ocular palsy nystagmus was present. Both disks were blurred and edematous for about 1 to 1.5 D. There were no hemorrhages or exudates. The visual fields were uniformly contracted, with a slightly greater involvement of the right nasal field. The blindspot in the right eye was much larger than in the left eye.

The results of the ocular examination, the contracted fields, with more marked involvement of the right nasal field and greater enlargement of the blindspot in the right eye, were suggestive of a tumor of the right frontal lobe.

Another survey of the eyes, on March 16, showed the picture of the fundi to be unchanged. The right visual field was further contracted, and there was a beginning defect in the lower nasal quadrant. The blindspot in the right eye showed further enlargement. The left visual field showed only a slightly increased concentric contraction.

The beginning sector-like defect observed at this examination, with the greater increased concentric contraction and enlarging blindspot on the right, was suggestive of a lesion of the right frontal lobe rather than of a lesion on the left side.

On March 22 air was injected into each ventricle. The roentgenogram showed no findings of localizing value. In view of this negative report and the uncertain neurologic signs, the patient was discharged for further observation.

On April 3, 1934, she was admitted to the Neurological Institute of New York with the following symptoms: absence of the upper abdominal reflexes, weakness of plantar flexion on each side, weakness of the muscles of the left eye, complete anosmia, pallor of the right disk and high papilledema of the left eye, bilateral paresis of the abducens nerve and facetiousness and irresponsibility.

The right visual field showed perception of light in the lower temporal and nasal quadrants only. The left visual field showed supramedial and inferolateral quadrantanopia.

A roentgenogram of the skull showed distinct evidence of an intracranial pathologic process, probably a tumor. The optic foramina were normal.

The preoperative diagnosis was meningioma of the olfactory groove.

On April 13 a right transfrontal osteoplastic flap was turned down, and a meningioma was found in the right anterior and middle fossa, which was subtotally removed. More tissue was removed on April 30 and May 19.

The postoperative diagnosis was meningioma of the right sphenoid ridge.

Of interest were the symptoms usually associated with tumor of the temporosphenoid region and the negative encephalographic picture. From a diagnostic point of view, one must evaluate the importance of an enlarging blindspot in the absence of a greater degree of choked disk on the same side. This is the first stage of the Foster Kennedy syndrome. It was possible in this case to follow the formation of the syndrome from the beginning.

CASE 4—E. R., a man aged 29, was seen on May 7, 1935, with a history that in March 1934 he began to have convulsions accompanied by immediate loss of consciousness and biting of the tongue. There was no aura or incontinence. The patient was mentally clear after the attacks, most of which occurred during the night. After April 1934 he had severe headaches but he never vomited. Neurologic examination gave negative results, except for a doubtful weakness of the lower right side of the face and a more active ankle jerk on the right than on the left. Recently he had been having personality changes and he appeared euphoric. He was unusually sleepy. The neurologic diagnosis was tumor of the frontal lobe.

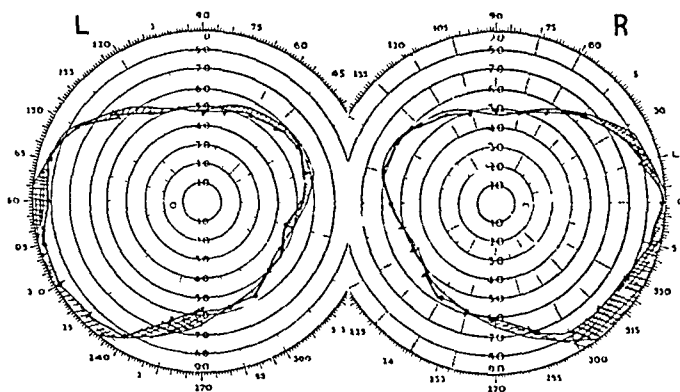


Fig. 4 (case 4)—Visual fields of E. R., taken on May 7, 1935, with a 2 mm. white test object at 250 mm.

Ocular examination disclosed vision of 20/25 in each eye. There was no nystagmus or ocular palsy. The pupils were equal and regular and reacted to light and in accommodation. The right disk was well outlined. There was an increase in the size and number of the capillaries, which gave the disk a violaceous hue. Both the arteries and the veins seemed increased in size, and there was a small flame-shaped hemorrhage near the nasal side of the disk. The left disk was somewhat more obscure, but there was no measurable edema. There were many flame-shaped hemorrhages near the disk. The visual fields were full.

The patient went to the New York Post-Graduate Medical School and Hospital, where roentgenographic examination revealed thinning of the bone over the premotor area, more marked on the left. The sella turcica was flattened from above down, and the posterior clinoid processes were eroded. There were three calcified plaques just to the right of the midline.

A diagnosis of tumor of the left premotor area was made.

Examination of the visual fields repeated at the hospital revealed no defects. On May 13 ventriculography was attempted, without success. Two days later the neurologist noted a definite weakness of the right side of the face and an exagger-

ation of the achilles tendon reflex on the right. There was partial aphasia. The patient repeated words and syllables. Examination of the fundi showed about 5 or 6 D of swelling of both disks, and vision had depreciated to the perception of large objects. On May 16 Dr Scarf made a ventriculogram, which showed marked distortion of the ventricles by a tumor occupying the premotor areas.

A diagnosis of parasagittal meningioma, more to the left, was made.

Craniotomy was done on the left side, and a parasagittal meningioma was disclosed which apparently arose from the falx. Subtotal removal was done. The tumor measured from 4 to 5 cm long and from 1 to 2 cm wide. Dr Scarf declared it to be the largest meningioma he had ever seen.

It is amazing to see the tremendous size a slow-growing tumor may reach before giving rise to serious symptoms, but how rapidly it progresses once it has started.

CASE 5—F F, a man aged 47, was referred to the ophthalmic clinic of the Brooklyn Hospital because of dizziness for the past year. He had been under

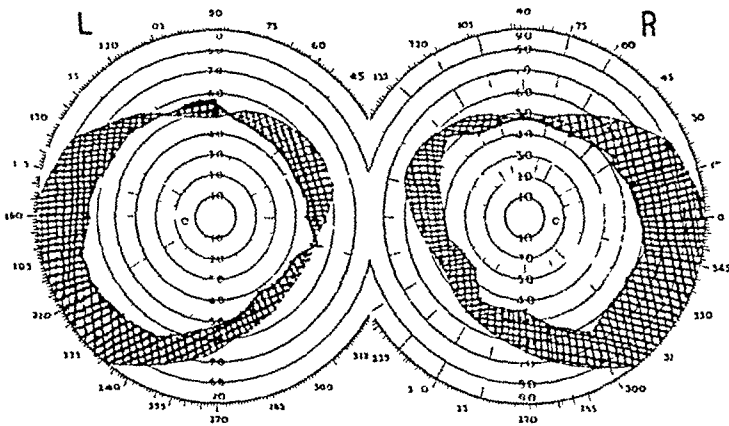


Fig 5 (case 5)—Visual fields of F F, taken on March 2, 1938, with a 2 mm white test object. Vision in each eye was 16/30.

treatment at a medical clinic for three months for hypertension and obesity. His blood pressure was 158 systolic and 112 diastolic.

About a year before admission to the hospital he was discharged from the post-office, where he was a laborer, for disobedience to his superiors. About this time paralysis of the left side of the body developed. For the past year there had been a progressive deterioration in his mentality as well as of his character. In 1917 he was struck on the head by a heavy package and was knocked unconscious for a short while. In 1935 he was struck by an automobile and was again knocked unconscious. About six years before admission to the hospital he had a seizure while combing his hair. This was characterized by a ten minute period of staring and speechlessness. There were no convulsive movements, but he kept repeating "la, la, la, la." He was able to understand spoken words and nodded his head in response. These attacks had recurred about eight to ten times a year, the last taking place about five days before admission to the hospital. This time the head tilted to the right, and he vomited a little.

His complaint on admission was that he became dizzy when he stooped and that he frequently had a pain in the left side of his head. When he stretched his arms upward, he had pain back of each ear.

Vision was 16/30 in each eye. There was no ocular palsy or nystagmus. There was about 3 D of choking of the right disk, with a small hemorrhage on the disk. The veins were dilated, and the arteries were narrow. The left disk was swollen about 2 D, and no hemorrhages were visible. There was marked secondary atrophy of both disks.

The visual fields were difficult to take and were not completely reliable, but apparently they showed nothing more than a concentric contraction, more marked in the right field.

The impression on the basis of these findings was that the patient had a tumor of either the frontotemporal or the frontoparietal region. Because of the paresis on the left side, the growth was thought to be located on the right. The patient was admitted to the Neurological Institute of the Brooklyn Hospital on March 8, 1938. The salient points of the neurologic examination were as follows: (1) complete anosmia, (2) slight facial paresis on the left side, (3) motor weakness of the left arm and leg, (4) ataxia of the left hand with the finger to nose test and (5) standing on a wide base.

The unanimous opinion of the members of the neurologic staff as well as of those of the neurosurgical staff was that the condition was a tumor of the frontal lobe on the right side. The tumor was probably a meningioma arising from the falx.

On March 14 a ventriculogram showed marked displacement of the ventricular system to the right, with the left lateral ventricle being pushed downward and medially. A lateral view showed the left lateral ventricle to be occluded in the midparietal region.

In view of the ventriculographic picture, it was thought that there was a tumor in the left frontal region.

Operation by Dr. E. J. Browder disclosed a tremendously large meningioma (from 6 to 7 cm at its greatest diameter) embedded in the left premotor area. It was attached to the falx, from which it derived a considerable vascular supply.

The visual fields, rightfully, showed no localizing defects and so could be considered correct. However, paresis on the same side as the tumor certainly was misleading, and if it had not been for the usual carefulness shown by Dr. Browder in making a ventriculogram, in the face of definite localizing signs, much unnecessary surgical intervention would have been carried out.

CASE 6—W. L., a man aged 24, came to the dispensary of the Brooklyn Hospital on Dec. 16, 1937, with a history of recurrent convulsive attacks for the past three months. These attacks were preceded by a slight feeling of numbness in the hands, which spread over the entire body, so that he grabbed onto the nearest object for support. Then a typical jacksonian convulsion developed, which started with the left hand and spread up that arm, the mouth was drawn to the left, followed by the turning of the head and eyes to the left. Generalized clonic convulsions developed during which consciousness was lost. Over the period of three months he had three major and four minor attacks, the longest lasting thirty minutes.

For the past year his mother had noticed a carelessness in his dress and some mental slowness. Every month or two he had attacks of severe frontal headaches, but for the past few months the headaches had been mainly in the occipital region.

There had also been episodes of momentary blindness when he arose from a sitting position. For the past four or five weeks he had been voiding involuntarily. He never vomited.

The patient was uncooperative, so that all ocular examinations were conducted with great difficulty. Vision was 20/40+ in the right eye and 20/25+ in the left eye. The pupils were equal and regular and reacted to light and in accommodation. There was no ocular palsy or nystagmus. The disks were swollen slightly more than 4 D. The visual fields showed a concentric contraction, that in the left being most marked, and there was a tendency toward a lower right quadrant-anopia.

In view of the results of the ocular examination, it was thought the patient had a tumor of the left frontoparietal region. He was admitted to the Neurological Institute of the Brooklyn Hospital on Jan. 3, 1938, where the following data were recorded. The patient was untidy and unkempt and was unable to concentrate on any one subject. He answered questions sluggishly. There was impairment of judgment, and he frequently made mistakes in reading.

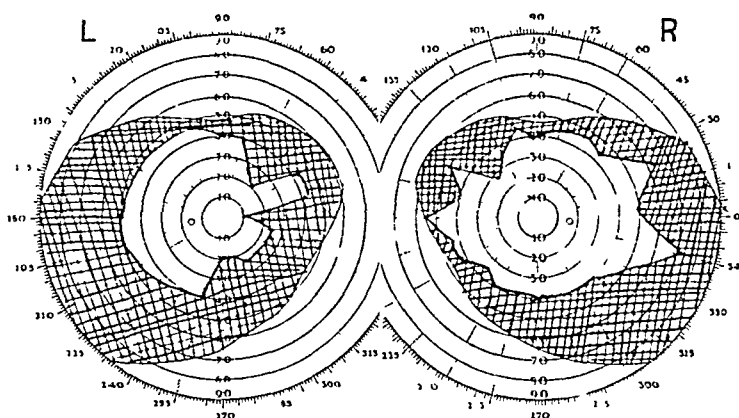


Fig. 6 (case 6)—Visual fields of W. L., taken on Dec. 16, 1937, with a 2 mm. white test object at 250 mm. Vision was 20/50+ in the left eye and 20/40+ in the right eye.

Neurologic examination showed (1) bilateral impairment of the sense of smell, (2) fine tremors of the extremities and lips, (3) deep reflexes which were slightly more active on the left and (4) good coordination. There was no stereopsis.

On the basis of the results of the neurologic examination, it was thought that there was a tumor of the right frontoparietal region, affecting secondarily, by pressure, the circulation on the left side.

A flat roentgenogram taken on January 4 showed fine mottled calcareous shadows in the right parietal area just above the temporal region. The sella turcica was apparently enlarged.

On the basis of the roentgenographic picture, it was thought that a tumor of the right parietal area was present.

The ventriculographic diagnosis was tumor in the right parietofrontal region.

On January 7 Dr. E. J. Browder turned down a bone flap in the right frontoparietal region and uncovered a highly vascularized type of malignant tumor (glioma) occupying the right premotor area and extending forward and possibly deep to involve the corpus callosum.

In 1911 Foster Kennedy⁹ stressed the localizing value of the syndrome in which there is unilateral loss of vision with secondary atrophy of the optic nerve and swelling of the opposite disk. In 10 of 14 cases of tumors of the basal portion of the frontal lobe Lillie¹⁰ found a central scotoma to be present. The scotoma was always on the side of the tumor. In 3 of the remaining cases homolateral blindness or near blindness was present. In no one of the 6 cases here presented was there a central scotoma. A Foster Kennedy syndrome was present in 1 and a paradoxical syndrome was present in another. A feature of interest, and I believe of some importance from a diagnostic standpoint, is the rapid deterioration of vision, once the patient has complained of blurring. The presence of a Foster Kennedy syndrome is an important sign in the diagnosis of a tumor of the frontal lobe, but when it is not present, a progressive concentric contraction, choked disks and an enlarging blindspot on one side should make one think of such a tumor.

GROUP 2 TUMORS IN THE REGION OF THE CHIASM AND THE MIDBRAIN

CASE 7—J. S., a man aged 42, was admitted to the Brooklyn Hospital on March 24, 1934, with a history of daily headaches and failing vision in the left eye since May 1933. The right eye had been crossed since childhood and had never had vision. The first symptom was a sudden blurring of vision before the left eye, so that the patient ran his automobile into a trolley car. Three months later he was admitted to a hospital in New York. A summary of the report made at this hospital follows:

The patient was admitted to the hospital on Oct. 18, 1933, with a history of blurred vision for the past three months. He had been a heavy drinker of alcohol and beer. A week before admittance he had twitching movements of his eyes, with pain. There was no loss of consciousness or vomiting. There were a right convergent strabismus and loss of sight. The left nerve head was pale. There was tenderness to pressure along the nerves of both lower extremities. The patient was disoriented, there were confabulation and memory defects. The visual fields were good, and the blindspots were not enlarged. A diagnosis was made of alcoholic intoxication with peripheral and optic neuritis.

Ocular examination on April 12, 1934, showed vision of 5/200 in each eye. The pupils were equal and regular and reacted to light and in accommodation. There was a right convergent strabismus. There was complete loss of color perception in the left eye, but color perception in the right eye, the convergent one, was perfect. Examination with a 2 mm white test object showed almost complete loss of the nasal field in the left eye, with sparing of the macula and in addition some concentric contraction of the temporal field. The right eye showed a loss of about one half of the temporal field, with some contraction of the nasal field.

⁹ Kennedy, F. Retrobulbar Neuritis as an Exact Diagnostic Sign of Certain Tumors and Abscesses in the Frontal Lobes, *Am J M Sc* **142** 355, 1911.

¹⁰ Lillie, W. I. Ocular Phenomena Produced by Basal Lesions of the Frontal Lobe, *J A M A* **89** 2099 (Dec 17) 1927.

In view of the results of the ocular examination, it was thought that the patient had a suprachiasmal tumor which pressed on the left optic nerve tract.

Laboratory examinations gave negative results. Roentgenographic examination of the skull and optic canals also gave negative results. Several studies of the visual fields, the last on May 3, showed substantially the same findings. Notes made by Dr. E. J. Browder on May 1 contained the following data: There was apparently an organic lesion. The character of the defects of the visual fields pointed to the parasellar zone on the left side. The condition was most probably a meningioma of the left sphenoid ridge just lateral to the tuberculum sellae.

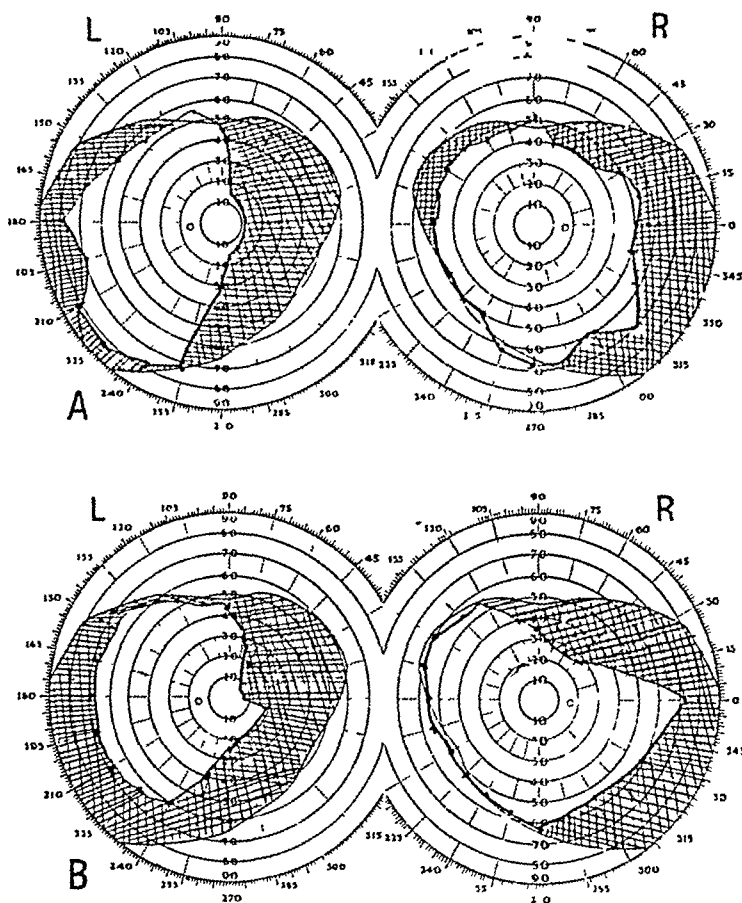


Fig 7 (case 7)—*A*, visual fields of J. S., taken on April 12, 1934, with a 2 mm white test object at 250 mm. Vision was 5/200 in each eye. *B*, visual fields of the same patient, taken on May 3, with a 2 mm white test object at 250 mm. Vision was 5/200 in each eye.

On May 8 Dr. Browder turned down a bone flap in the left frontal region and exposed the sphenoid arch. This was followed medially where the tumor was expected to be, but none was found. When the left optic nerve was visualized, however, he could see a bulbous swelling of the nerve trunk just before it entered the left optic foramen. It was clear to him that the tumor was located within the nerve itself. Several fair-sized blood vessels could be seen coursing over the upper surface of this tumor. An incision was made into the mesial part of the tumor, and several small fragments were taken for histologic study. Dr. Browder decided to get the histologic report before further resection was done, because it would be necessary to sacrifice a portion of the chiasm. As the histologic

study of the fragments revealed them to be edematous nerve tissue with no suggestion of tumor tissue present, nothing further was done

When the patient was last seen on Sept 30, 1934, he was completely blind. In spite of the negative histologic report, Dr Browder felt that we were dealing with a tumor of the optic nerve of unknown origin. The defect in the temporal portion of the right visual field pointed to an extension upward of the tissue into the chiasm involving the crossed fibers of the right optic nerve. Against an inflammatory cause and in favor of a tumor was the absence of atrophy of the optic nerve in the right eye, with the presence of good color perception, which was absent in the left eye.

CASE 8—M. W., a girl aged 17, was seen on Oct 3, 1931, with a history that about two years before she had lost considerable weight. About a year later a fever developed. Her condition was diagnosed as glomerulonephritis, with uremia. At that time she had headache, nausea and vomiting. These symptoms cleared, and soon after she began to gain a good deal of weight, and her menses

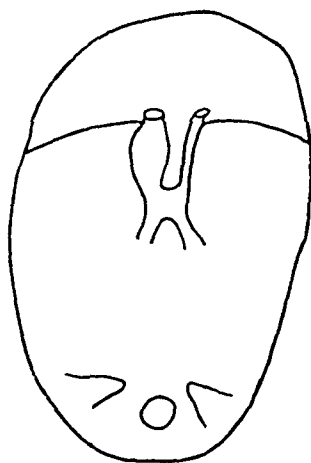


Fig 8—Drawing of operative findings by Dr E. J. Browder

became scanty and irregular. She also began to become dull mentally. For the past several months she had been under the care of a "gland specialist," who had been giving her injections. She had no headaches, blurring of vision or diplopia.

Vision was 20/20 in each eye. The margins of both disks were somewhat blurred, and the temporal portions were slightly pale. The blood vessels were normal. There was some loss of both temporal fields, particularly of the lower part of the right field, and both blindspots were somewhat enlarged.

A diagnosis of suprasellar cyst was made, and the patient was asked to return in a month for further study. She returned two months later, December 5, saying that she was feeling stronger and more ambitious, but that she was still gaining weight and that her menses were still scanty.

Vision was 20/20 in the right eye and 20/100 in the left eye. The right disk showed slight pallor in the temporal portion. The margins of the left disk were definitely edematous, though not measurable. The pallor in the temporal portion was more marked. Examination of the visual fields showed a cecocentral scotoma and a marked defect in the lower part of the left temporal field. The right visual field showed little change.

The patient went to a prominent neurologist in New York, who in addition elicited a history of diabetes insipidus. A flat roentgenogram showed definitely that there was no tumor of the pituitary gland. The sella turcica appeared half the normal size for a girl that age and seemed flattened from above.

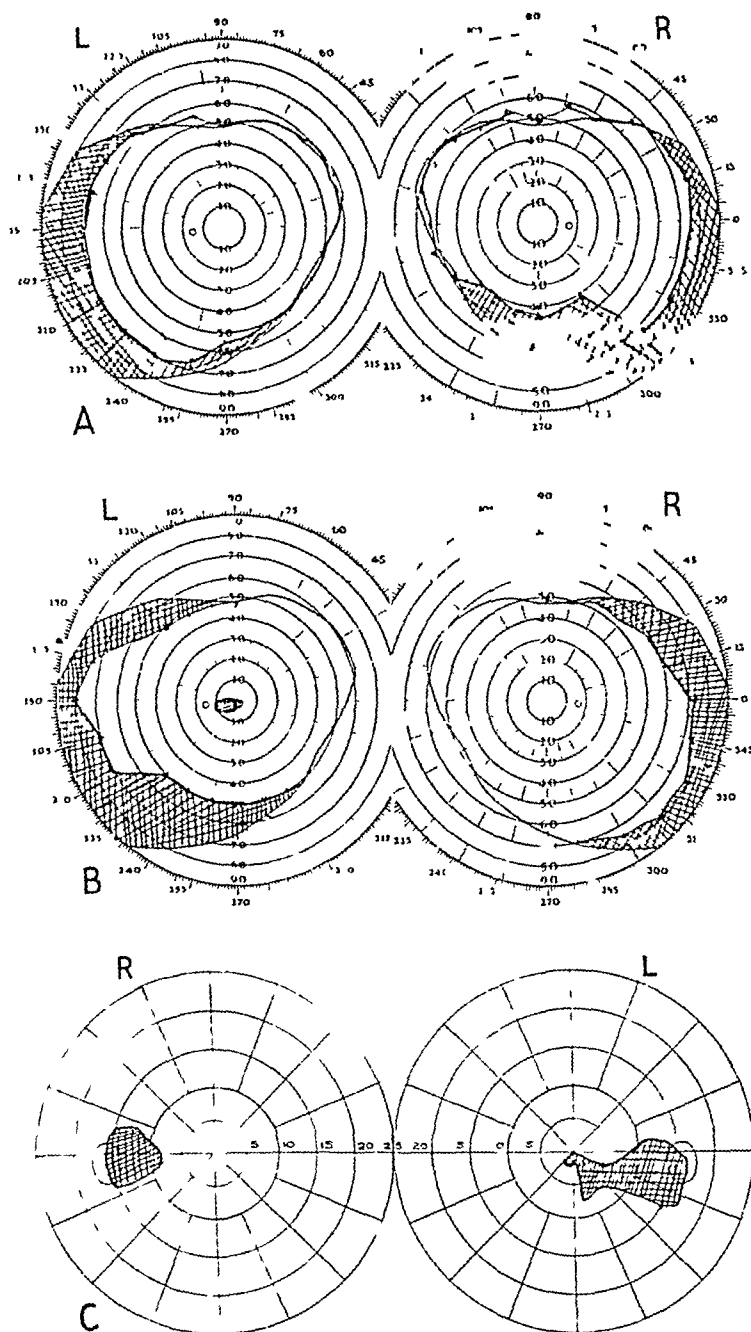


Fig 9 (case 8) —*A*, visual fields of M W, taken on Oct 3, 1931, with a 1 mm white test object at 250 mm. *B*, visual fields of the same patient, taken on December 5, with a 1 mm white test object at 250 mm. *C*, visual fields, taken on December 5, with a 2 mm white test object at 1,000 mm.

It was the neurologist's impression that the patient might have a suprasellar cyst but that the weight of evidence was against such a diagnosis. As he stated his impression, "Her amenorrhea, her obesity and diabetes insipidus could be accounted

for by a lesion on the floor of the third ventricle in approximation of the infundibulum" He referred the patient to an ophthalmologist, who found the same defects in the visual fields previously described, but it was his opinion that because the defect was ventral the lesion was not on top but under the nerve

This interpretation of the visual fields is not in accord with the present conception of the pathways of the nerve fibers from the retina to the brain Henschen¹¹ and others have conclusively shown that the nerve fibers from the retina, with the exception of the macular fibers, keep the same relative position from beginning to end, i. e., fibers from the upper part of the retina, which supply vision in the lower visual fields, are situated in the upper part of the nerve tract and radiation, so that pressure from above will cause loss of the lower visual fields and pressure from the temporal side will cause loss of nasal fields

However, the neurologist and the ophthalmologist referred the patient to a neurosurgeon, expressing their uncertainty as to the diagnosis He agreed with them, and referred her back for further observation She was observed monthly until her vision was completely lost in both eyes, when she went to the Johns Hopkins Hospital, where she was operated on, July 12, 1932

The following report was taken from Dr Dandy's notes When the left lateral ventricle was tapped, fluid spurted out under high pressure About 20 cc of fluid was removed, and an equal amount of air was injected The right lateral ventricle did not fill The third ventricle filled only in part and showed a filling defect due to a tumor There was no dislocation of the lateral ventricles The patient was kept in the hospital for two weeks because of a fever, the temperature ranging from 102 to 103.5 F The leukocyte count was around 10,000 No infection was found to account for the fever, and it was finally concluded that it was central in origin The patient had been extremely drowsy throughout her stay in the hospital, sleeping most of the time, but she could be roused Pressure could not account for her drowsiness, because the ventricular dilatation was too slight, so it had to be explained solely on the basis of the part of the brain involved by the infiltrating lesion

A preoperative diagnosis of an infiltrating lesion of the floor of the third ventricle was made

Operation revealed the third ventricle to be full of gliomatous tumor tissue The tumor tissue, being on the floor of this ventricle, had directly involved the visual fibers

In view of the course of the disease, it is interesting to speculate on the cause of the fever in 1929 Did the patient have glomerulonephritis, of which she gave no later symptoms or objective findings, or did she have hyperthermia of central origin with diabetes insipidus?

Also of interest are the marked symptoms of pituitary dysfunction, in spite of the fact that roentgenograms showed no direct pressure on the pituitary gland by the absence of an enlarged sella turcica

CASE 9—C A, a man aged 27, was seen on March 8, 1937, with a history that about twelve years before he noticed a beginning loss of libido He went to an internist, who treated him with glandular preparations Shortly after, he complained of visual disturbances, and glasses were prescribed, with relief from his

11 (a) Henschen, S. E. Zur Anatomie der Sehbahn und des Sehzentrums, Arch f Ophth **117** 403, 1926 (b) Ronne, H. Pathologisch-anatomische Untersuchungen uber alkoholische Intoxikationsamblyopie, *ibid* **77** 1, 1910

symptoms. For some time he continued to receive glandular therapy. Every now and then he would have a severe headache. Vision began to deteriorate, but so slowly that he was not conscious of its loss until it was well advanced. In the meantime he had changed doctors several times but continued to receive glandular treatment.

In November 1936, while in Chicago, his headaches became severe and persistent and were accompanied by projectile vomiting. He went to an ophthalmologist who referred him to Dr. Loyal Davis. Studies of the visual fields showed a defect in the upper temporal portion of the right field and a tendency toward a concentric contraction in the left field.

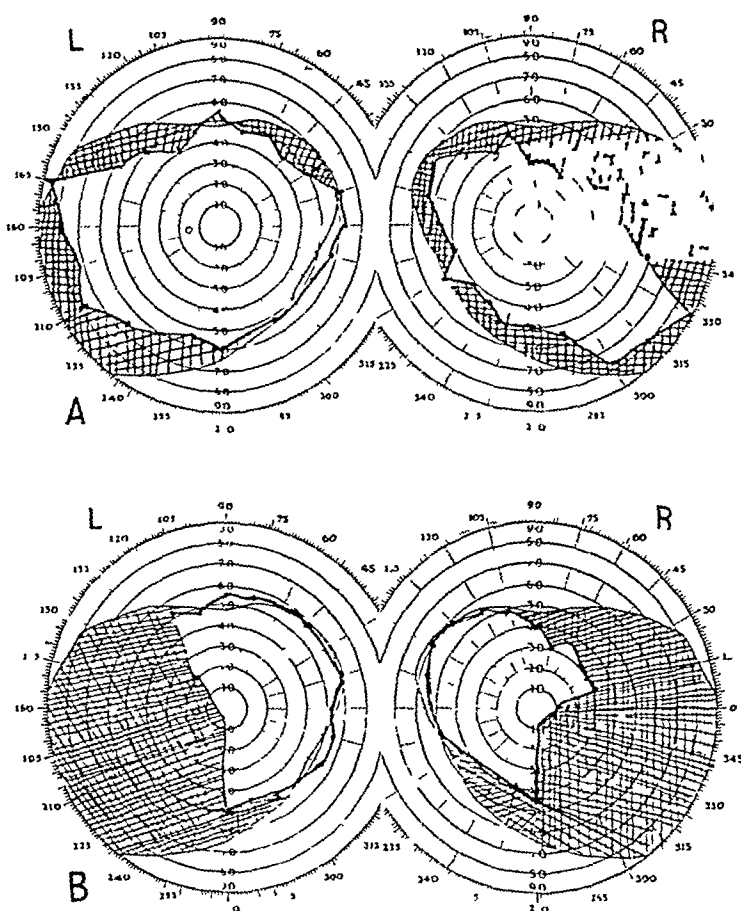


FIG 10 (case 9)—A, visual fields of C A, taken on Nov 10, 1936, with a 4 mm white test object. B, visual fields of the same patient, taken on April 20, 1937, with a 2 mm white test object at 330 mm. Vision was 20/70 in each eye.

On November 16 Dr. Loyal Davis performed a right transfrontal osteoplastic craniotomy and found a cyst extending anteriorly to the chiasm rather than posteriorly. When it was opened a large amount of yellow fluid containing fatty deposits was evacuated. Immediately below the chiasm and pressing upward into its structure was a large globular irregular mass of cholesterol. It extended into the tissue of the chiasm. Part of the wall of the cyst and as much of the cholesterol as possible were removed without severing the chiasm. The chiasm was so involved by this mass that Dr. Davis was certain the visual fields obtained were absolutely incorrect (obtained by clinical clerks). The damage was in the center, and one would expect the visual fields to show bitemporal hemianopia.

After operation the patient's vision improved. Physical examination showed the patient to be short and slight of build, with no hair on the face. He had small delicate hands and feet with the voice of a preadolescent boy. He had complete loss of sexual power.

Ocular examination on April 20, 1937, showed vision of 20/70 in each eye, which improved with correction. Both disks showed advanced primary atrophy. The visual fields showed practically complete bitemporal hemianopia, which corresponded to what Dr. Davis thought should have been present at the first examination.

CASE 10—L. T., a woman aged 50, entered the Brooklyn Hospital on Oct. 5, 1936, with a history that thirteen years before she had noticed a tingling of her hands and feet. One year later she began to grow larger in all dimensions until four years before admission to the hospital. During this period she gained about 100 pounds (45 Kg.), and her hands and feet grew so large that she had to wear men's sizes. Two years before admission she began to have pains in her legs. One and a half years before a tonsillectomy was performed in a

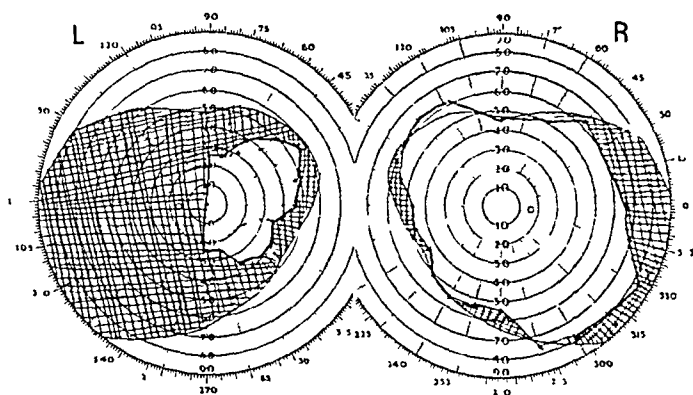


Fig. 11 (case 10)—Visual fields of L. T., taken on Oct. 8, 1936. Vision in the left eye was limited to perception of fingers at 1 foot. The visual field with a 15 mm. white test object at 250 mm. is indicated by the dotted line, that with a 40 mm. white test object is indicated by the heavy black line. The right eye was tested with a 2 mm. white test object at 250 mm. Vision was 13/13.

hospital in Brooklyn. About a year before she began to have polyuria and polydipsia. About this time she received roentgen treatment to the region of the pituitary gland, which was followed by loss of vision in the left eye. Her periods started at the age of 13 and were normal until her menopause at 44.

Physical examination revealed a person of the typical acromegalic type. Dr. Browder made a diagnosis of acidophilic adenoma.

Ocular examination on October 8 showed vision in the right eye of 13/13, vision in the left eye was limited to perception of fingers at 1 foot (30 cm.). The left pupil was slightly larger than the right, but both reacted to light and in accommodation. The margins of the right disk were blurred, although no measurable edema was present. There was no pallor of the temporal side. The veins were not unusually full, and there were no hemorrhages or exudates. The margins of the left disk were blurred. The entire disk was pale, the temporal side being more so. With a 2 mm. test object there was a tendency toward a concentric contraction of the right visual field, with a greater loss in the upper

temporal portion The smallest test object that could be used for the left eye was a 40 mm target With this test object there was complete loss of the temporal portion of the field, with a marked contraction of the nasal side

On the basis of the results of the ocular examination, it was thought that a tumor of the pituitary gland was pressing on the left optic nerve, with beginning involvement of the right crossed fibers

On October 19 Dr Browder, through a transfrontal approach, removed an acidophilic adenoma, a knuckle of which had broken through the dorsum sellae

CASE 11—D F, a man aged 42, came to the dispensary of the Brooklyn Hospital on Sept 26, 1934, with a history that a year previously a herniorrhaphy had been performed (right inguinal) with spinal anesthesia Since then he had severe headaches in the frontal and parietal regions Five months ago before coming to the dispensary he noticed that his right upper lid drooped and that vision in the right eye was lost He had never vomited

Ocular examination on September 27 showed ptosis of the right upper lid which, while the patient was under observation, varied from slight to complete

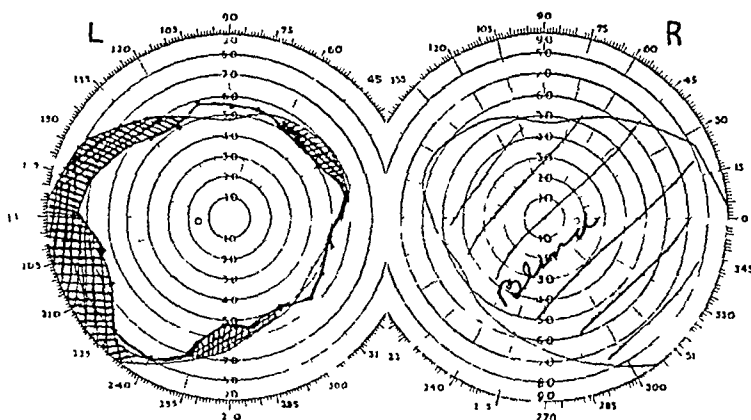


Fig 12 (case 11) —Visual fields of D F, taken with a 2 mm white test object at 250 mm

The eye was practically fixed in the midline, having but slight movement in any direction The right pupil was widely dilated and reacted to consensual but not to direct light The left pupil reacted well both to direct and to consensual light Vision in the right eye was limited to uncertain perception of light and that in the left eye equaled 20/20 The right disk showed moderate primary atrophy The left visual field was normal There was tenderness on percussion over the right orbit

On the basis of the results of the ocular examination, it was thought that there was a tumor in the apex of the right orbit, the only point against this diagnosis being the absence of exophthalmos

The roentgenographic report was negative, except for mention that the posterior clinoid processes were not visible

The patient was admitted to the hospital On October 5 Dr Browder made the following observations The patient vomited for the first time the previous day There was pain over the right side of the head Nothing, however, was heard on auscultation The eye was held in external rotation (this was evidence of the varying state of the ocular paralysis) There was no loss of sense of smell

It was thought that the condition was either a tumor in the apex of the orbit or one involving the sphenoid fissure. The possibility of it being a meningioma of the right tuberculum sellae or of the medial aspect of the right sphenoid ridge was recognized. However, the first impression, in view of the normal left visual field, was that the condition was a metastatic lesion in the situations previously mentioned. Exploration was advised.

Ocular examination on October 11 showed no change over the previous findings, except that the atrophy of the right disk was more pronounced.

Another roentgenogram made on October 8 was normal.

On October 13 neurologic examination revealed the following symptoms: (1) a diminution of pain and temperature sense on the right, (2) a diminution of tactile discrimination and vibratory sensation on the right, (3) normal stereognosis and (4) increased spinal fluid pressure.

The patient refused to have an operation performed and was discharged from the hospital.

On November 13 he was admitted to the Neurological Institute New York, when the following findings were recorded. After the patient's discharge from the Brooklyn Hospital vision in the left eye began to fail and became progressively worse. The deep reflexes on the right were hyperactive, and the Babinski sign on the right was positive. There was marked atrophy of the optic nerve on the right, and the left visual field was greatly restricted. The serologic reactions and the chemical composition of the blood were normal.

The first roentgenographic report stated that the condition was an intracranial expanding lesion and that aneurysm was to be considered, but definitely not a tumor of the pituitary gland.

A second roentgenogram strongly suggested that an aneurysm of the right internal carotid artery or of the posterior communicating artery was present.

According to the encephalographic report an expanding lesion, slightly posterior to the chiasm was present, which was probably a tumor, an adenoma of the pituitary gland, and not an aneurysm.

On December 1 a right transfrontal craniotomy exposed a tumor in the suprasellar region, which was partially removed. The pathologic diagnosis was that of chordoma.

The postoperative diagnosis was chordoma of the middle fossa.

CASE 12—W. C., a Negress aged 32, came to the neurologic clinic of the New York Post-Graduate Medical School and Hospital on Oct. 16, 1937, with a history that since December 1936 her vision had been failing. In April 1937 her menses stopped, and she began to have occasional frontal headaches. Occasionally she vomited, especially in the morning. She had continuous thirst and an excessive craving for sweets. She had been going to doctors and clinics, but her vision rapidly became worse.

Neurologic examination disclosed a well developed Negress without any physical signs of a deficiency in the pituitary secretion, such as adiposity or dull mentality. The fundi were normal, and neurologic examination gave negative results.

The opinion was that the condition was probably a suprasellar cyst.

Ocular examination on October 22 showed vision in the right eye to be limited to perception of hand movements on the nasal side. Vision in the left eye was limited to perception of fingers at 2 feet (60 cm.). The visual fields were unreliable but suggested left homonymous hemianopia. The pupils were equal and regular and reacted slowly to light and well in accommodation. The fundi were normal.

In view of the normal fundi and the history of such long-standing blindness, a lesion of the posterior cortex was suggested

A week later the patient was again examined in the ophthalmic clinic by another observer, and at this time she was completely blind and the fundi were still normal

Flat roentgenograms on October 23 were completely normal

A month later the patient went to the Neurological Institute of New York, where Dr John Scarf found the right eye to be completely blind and the other eye to have a temporal hemianopia to strong light

At this time the disks showed evidence of primary atrophy The plain roentgenograms were normal An encephalogram revealed evidence of a suprasellar cyst

On November 27 a large suprasellar cyst was exposed, and about 30 cc of xanthochromic fluid was evacuated The following day the patient could count fingers with either eye at 5 feet (152 cm)

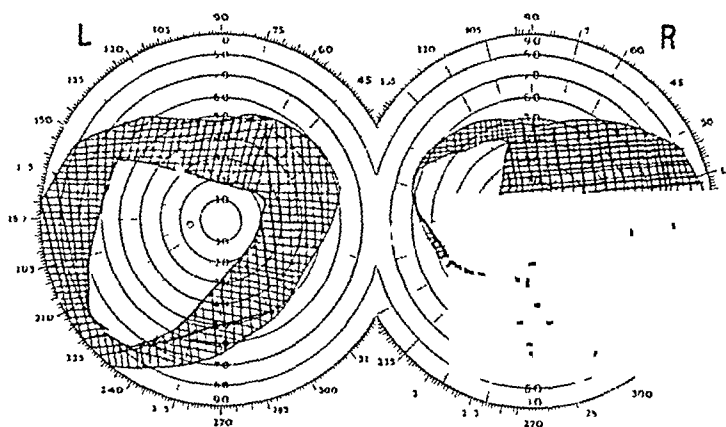


Fig 13 (case 12) —Visual fields of W C, taken on Oct 22, 1937 Vision in the right eye was limited to perception of hand movements on the nasal side, that in the left eye was limited to perception of fingers at 2 feet

A great deal of the loss of vision must be explained on a hysterical basis Though the patient was so blind that she had to be led around, the fundi remained normal for a while and even later showed only early atrophy of the optic nerve After operation, Dr Scarf expressed astonishment that the next day the patient was able to count fingers at 5 feet with either eye

CASE 13—J G, a Negro aged 34, was admitted to the Neurological Institute of the Brooklyn Hospital on Feb 24, 1938, with a history that until 1929 he was of the average normal height of the rest of his family At this time he wore size 9 shoes Then he began to grow in size, his features began to change, and his hands and feet began to grow progressively larger Until 1933 there were no other symptoms At this time vision in the right eye began to fail, the hair on the face and pubis became more scant, and his sexual power was impaired He also became lethargic, so that in time he was unable to do any work At the time of admission to the hospital he wore size 12½ shoes, and there was complete loss of sexual power He was also several inches taller than in 1929

Examination showed a very tall Negro, who appeared ill and toxic. He had the typical features of a person with acromegalia. Mentally, he was dull.

A diagnosis of eosinophilic adenoma of the pituitary gland was made. A flat roentgenogram taken on March 9 showed the bones of the skull to be rather thin and the sella turcica to be immense. The anterior and posterior clinoid processes were partially eroded.

The impression on the basis of the roentgenographic picture was that the condition was a tumor of the pituitary gland.

Ocular examination on March 16 showed advanced atrophy of the right disk. There was imperfect perception of light. The left disk was of good color. There was loss of the temporal field, with sparing of the macula.

A diagnosis of tumor in the region of the sella turcica was made.

On March 18 Dr. E. J. Browder removed a large cystic degenerated tumor of the pituitary gland through a flap in the right frontal region.

CASE 14—A. D., a woman aged 29, came to the neurologic clinic of the New York Post-Graduate Medical School and Hospital on Nov. 17, 1937, with a

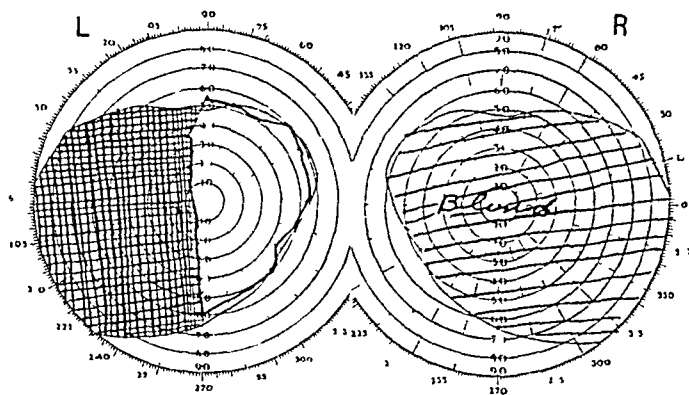


Fig. 14 (case 13)—Visual fields of J. G., taken on March 16, 1938, that for the left eye being plotted for a 2 mm test object at 330 mm.

history that since July 1937 her vision had been getting progressively worse. For the past five months she had been having periodic headaches which arose from the frontal region and radiated to the back of the head. They usually came about once a month and lasted from one to two days. The headaches were severe. She also vomited frequently. Her menstrual periods stopped about seven years previously, and she had not had one since. She had not lost or gained any weight, and there was no history of polyuria or polydipsia. At 9 years of age a nail pierced the right eye, and since then she had never been able to see with that eye. An ophthalmologist had made a diagnosis of bilateral central scotoma.

Flat roentgenograms taken on Jan. 11, 1938, showed the following picture. The sella turcica was much distended. The posterior clinoid processes were hazy, and the sphenoid sinuses were encroached on. All these findings were thought to point to the presence of an expanding neoplasm in the region of the sella turcica.

Ocular examination on January 12 showed vision in the right eye to be limited to perception of hand movements. It was not improved with correction. There was a scar in the upper right quadrant of the cornea. The pupil was drawn to

the scar by an adherent iris. The disk showed moderate primary atrophy. Vision in the left eye was 5/200, it was not improved with correction. The disk showed moderate primary atrophy. Examination of the visual fields revealed a bitemporal macular defect and a defect in the lower temporal quadrant of the left eye. The macular defect in the right eye involved the entire central area. This, I felt, was caused by the long-standing amblyopia.

It was thought that the bitemporal macular defects were due to pressure on the crossed macular tracts in the chiasm and that the defect in the inferior quadrant indicated pressure from above.

A diagnosis of a suprasellar cyst was made.

On January 28 Dr. Mac Lean turned down an osteoplastic flap in the right temporofrontal region. In the region of the sella turcica there was a cyst which encroached on the chiasm posteriorly and superiorly. The cyst was needled and 8 cc of thick yellow brown fluid was aspirated. The cyst was then incised, and tumor tissue was curetted out.

A postoperative diagnosis of tumor of the pituitary gland with cystic degeneration was made.

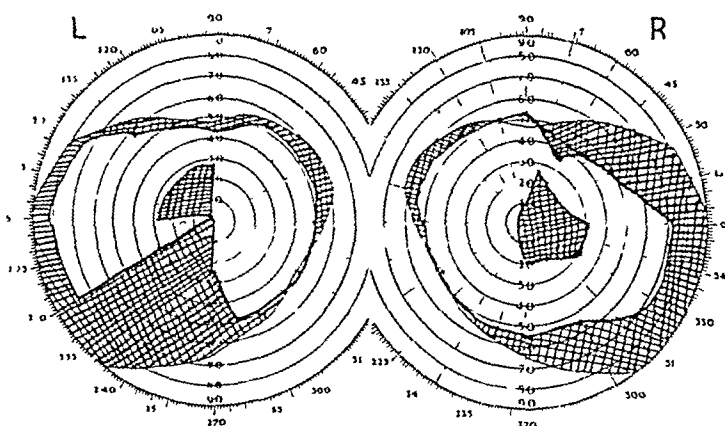


Fig. 15 (case 14) —Visual fields of A. D., taken on Jan. 12, 1938, with a 5 mm white test object at 250 mm. Vision was 5/200 in the left eye; that in the right eye was limited to perception of hand movements.

The second group of cases can only emphasize what has so often been emphasized by Cushing, Walker and others,¹² that lesions in the region of the chiasm, owing to the close proximity of so many structures, can give rise to puzzling changes in the visual fields as well as to unusual physical signs.

It hardly seems possible that persons with such outstanding signs of acromegaly as those considered in cases 10 and 13 would be allowed to go for so many years without diagnosis and therapy. The patient in

12 (a) Walker, C. B., and Cushing, H. Chiasmal Lesions with Especial Reference to Homonymous Hemianopsia with Hypophyseal Tumor, *Arch. Ophth.* **47**: 119, 1918. (b) Horrax, G., and Putnam, T. J. Distortions of the Visual Fields in Cases of Brain Tumor, *Brain* **55**: 499, 1932. (c) Cushing, H., and Heuer, G. J. The Distortions of the Visual Fields in Cases of Brain Tumor, *Bull. Johns Hopkins Hosp.* **22**: 190, 1911.

case 9 should have been operated on long before such complete loss of pituitary function developed, nor should the patient in case 12 have been allowed to become completely blind.

Of great interest is case 8, in which, in the presence of a tumor of the third ventricle, there were all the symptoms, physical and endocrinologic, as well as changes in the visual fields, suggestive of a suprasellar cyst. Outside of the hyperthermia, there were none of the symptoms usually associated with tumor of the third ventricle, such as loss of upward gaze, altitudinal anopsia and high grade papilledema.

In no other group of cases can studies of the visual fields, and especially follow-up studies, be so fruitful of good results. Any patient showing evidence of pituitary dysfunction and changes in the visual fields which can be attributed to a lesion in the region of the chiasm and

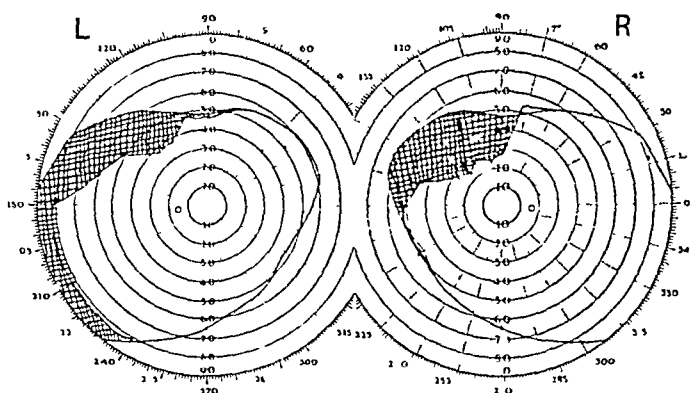


Fig 16 (case 15)—Visual fields of I S, taken on Oct 13, 1929, with a 1 mm white test object. Vision was 20/15 in each eye.

progressive loss of vision is entitled to an exploratory operation instead of waiting for blindness to supervene.

GROUP 3 TUMORS POSTERIOR TO THE CHIASM

CASE 15—I S, a man aged 26, was seen on Oct 11, 1929, with a complaint of double vision. For the past year he had been having occasional attacks of unconsciousness. These attacks lasted for a few seconds and were accompanied by either visual or auditory aura. The auditory aura consisted of beautiful angelic voices, while the visual aura were made up of angles or corners of rooms, never of persons. The aura were always the same. He never had hallucinations of taste or smell, nor did he ever have convulsions. About one year before the present examination, at the commencement of his complaint, the patient had profuse rhinorrhea and a severe headache and vomiting. His condition had been diagnosed as petit mal several times.

On September 13 he saw double for the first time. The diplopia persisted. In October 1929 he again had a severe attack of rhinorrhea and headache but no vomiting. The attack lasted ten minutes and was accompanied by blurred vision. Physical and neurologic examination gave completely negative results.

Ocular examination showed vision of 20/15 in each eye. There was no ocular palsy and no nystagmus. There was a slight, barely perceptible convergence of the right eye when the patient looked at a distance. The pupils were equal and regular and reacted to light and in accommodation. Examination of the fundi showed about 15 D of swelling of the right disk and about 25 D of swelling of the left disk. There were no hemorrhages or exudates. The patient had homonymous diplopia, which increased for distance and disappeared at the reading distance. The diplopia did not increase to the right or left, a true divergence paralysis.

Perimetric study revealed hemianopia in the upper left quadrant, and a diagnosis of a tumor deep in the right temporal lobe was made.

On November 5 Dr. Dandy removed a large deep-seated glioma, and with it practically the entire right temporal lobe. The first symptom to disappear was the diplopia. Dr. Dandy stated in a letter that the only localizing sign the patient ever had was the defect in the visual field.

CASE 16—C. F., a man aged 39, entered the Brooklyn Hospital on March 5, 1935, with a complaint of severe headaches for the past year. Two weeks before

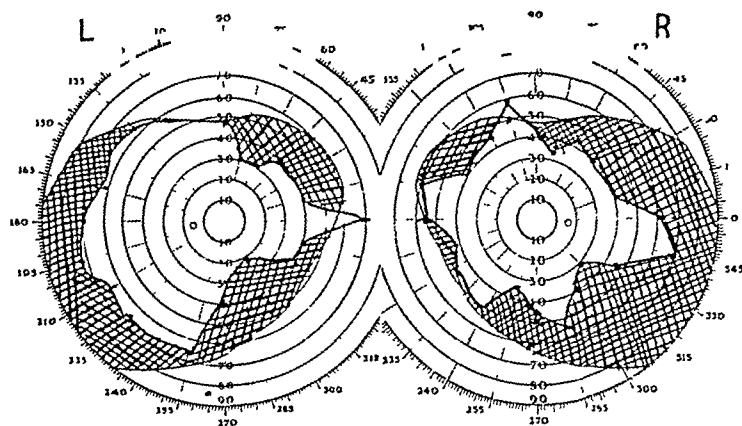


Fig. 17 (case 16)—Visual fields of C. F., taken on March 6, 1935, with a 2 mm white test object at 250 mm.

admission the headaches became more severe, and he vomited a great deal. On the day of admission he became stuporous. Examination of the fundi showed the right disk to be swollen about 25 D and the left, about 15 D. The disks showed evidences of secondary atrophy. There were a few hemorrhages around the disks. The next day the patient was fairly alert, irritable and uncooperative, but satisfactory visual fields were obtained. These showed an irregular concentric contraction and right hemianopia, the lower portions of the fields being particularly involved.

A diagnosis of tumor of the left frontoparietal region was made. On March 7 examination by a neurologist revealed the following data. The patient was clear mentally and was complaining of severe frontal headaches, mostly behind the eyes. His mental processes were slow, but not strikingly so. He had noticed a queer numbish feeling in the right upper extremity for the past week, and there was a drifting of the right upper extremity. There were no gross stereognostic changes. There was bilateral ankle clonus but no Babinski sign.

In view of these findings, it was thought that the patient had a tumor of the inferior parietal lobe.

On March 10 Dr Browder turned down a bone flap in the left parietal region (somewhat posterior), and a soft tumor in the lower parieto-occipital region was scooped out. It proved to be a spongioblastoma.

CASE 17—J. M., a man aged 23, came to the dispensary of the Brooklyn Eye and Ear Hospital on Sept 2, 1932, with a history of headaches for the past year and diplopia for the past week. The headaches came on about two or three times a week. A month previously he noticed numbness and weakness of his left side, and he began to vomit. He entered a local hospital, where he was kept for ten days and was discharged without a diagnosis. A week before he came to the dispensary diplopia developed.

Ocular examination showed vision of 20/25—in each eye. The right eye was slightly turned in. There was homonymous diplopia, which increased for distance and disappeared at the reading distance. The diplopia did not increase to the right or left, a true divergence paralysis. The right disk showed about 25 D of swelling and the left, somewhat less. The visual fields showed hemianopia in the upper left quadrants. A roentgenogram of the skull on October

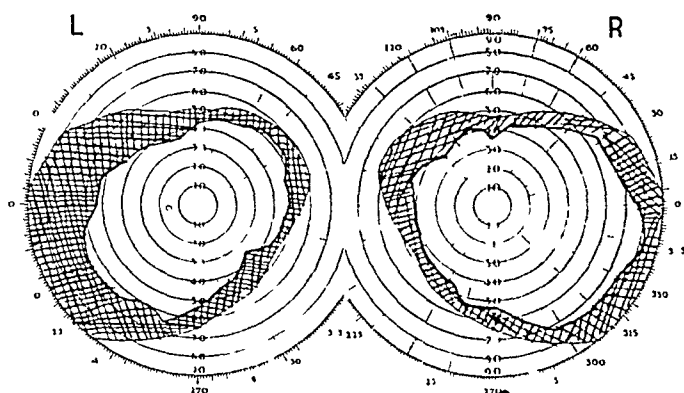


Fig. 18 (case 17)—Visual fields of J. M., taken on Sept 5, 1932, with a 2 mm white test object at 250 mm.

6, at the Brooklyn Eye and Ear Hospital, did not show any evidence of an intracranial abnormality.

A diagnosis of tumor of the right temporal lobe was made.

The patient was admitted to Dr Browder's service at the Long Island College Hospital on October 14. Physical examination revealed choking of the disks and active knee and ankle jerks but no localizing signs. Perimetric studies at this hospital showed general contraction of the visual fields, which was more noticeable on the left. Ventriculographic examination by Dr Mendelson showed tumor of the brain, probably of the right frontal lobe.

On October 16 a bone flap was turned down on the right side, and a tumor of the right ventricle was exposed. It arose from the medial wall about opposite the junction of the posterior and the temporal horn of the right ventricle. It was fairly well circumscribed and attached to the choroid plexus.

A diagnosis of ependymoma of the right ventricle was made.

CASE 18—M. B., a woman aged 46, entered the Brooklyn Hospital on Sept 23, 1933 with a history that three weeks previously severe pains suddenly developed across the top of the head. Later that day the pains became localized to the

right frontal area. Since then the headaches had been constant and knifelike. Vomiting began simultaneously with the headaches and was so severe that she could not retain even water. She remained in bed, because even raising her head made her dizzy. A local physician diagnosed the condition as sinusitis, and much medication was prescribed. From November 1932 to June 1933 she had frequent nose bleeds, but none since then. The house surgeon at the hospital made the following observations: Vision was poor. There was a suggestion with a gross test of left homonymous hemianopia. There was bilateral ptosis, more on the right than on the left. The right pupil was smaller than the left and both reacted sluggishly to light and in accommodation. There were bilateral facial paresis and a slight drift of the right arm. There was bilateral papilledema.

On the basis of the results of the ocular examination, it was thought that the condition was an abscess of the right hemisphere of the brain or a tumor of the brain involving the midbrain and hypothalamus.

The same day neurologic examination showed the following symptoms: (1) bilateral ptosis, greater on the right than on the left, (2) a defect of upward gaze, more marked on the right, (3) vertical nystagmus when the patient

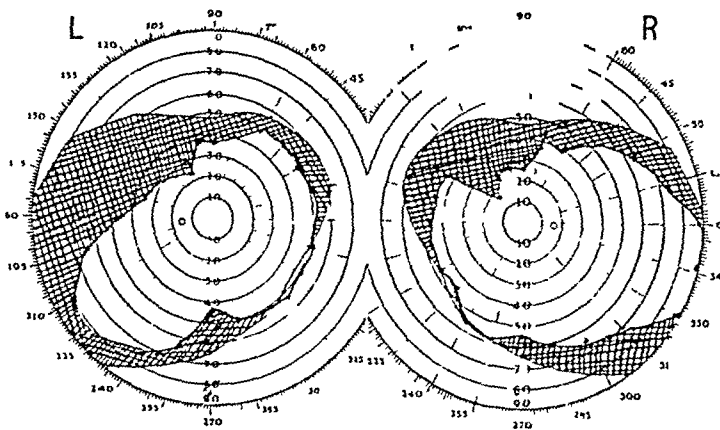


Fig 19 (case 18) —Visual fields of M. B., taken on Jan 9, 1934, with a 2 mm white test object at 250 mm.

attempted to look up, (4) early papilledema, more marked on the right, and (5) exquisite tenderness of the right frontoparietal region to tapping and deep pressure.

The state of the reflexes was not considered of localizing value. Syphilis, because of involvement of the tegmental plate, was ruled out as were also tumor of the brain and encephalitis.

On September 27 Dr. Browder made the following observations: Because of the acute onset and rapid progression of symptoms to a marked state of stupor within a period of three weeks, the amount of choked disks (which had increased tremendously), the absence of upward gaze, the bilateral ptosis and the stiff neck, the condition was probably a posterointraventricular tumor or a lesion of the quadrigeminal plate.

On October 1 a ventriculogram showed a shift to the left indicating a tumor in the right frontotemporal area.

That day Dr. E. J. Browder turned down a bone flap on the right side, and a tumor 5 cm in diameter was outlined in the posterior part of the temporal convolution about 1.25 cm under the cortex.

The pathologic diagnosis was glioblastoma multiforme.

The patient was readmitted to the hospital on Jan 6, 1934, because of recurrent headaches and vomiting. She was referred for studies of the visual fields, which revealed left homonymous hemianopia. On January 17 the bone flap was reelevated by Dr Browder, and a recurrent tumor occupying the region of the temporal fossa was identified. The entire temporal lobe of the brain with the tumor was removed.

I did not examine this patient until before the second operation, but the report of the case is included because of its many points of interest. Although all neurologic signs pointed toward a tumor of the midbrain, even the superficial examination of the visual fields made by the house surgeon revealed a left homonymous hemianopia. If this patient had been properly examined by the perimeter, particularly before the vision became too poor, a more exact diagnosis would probably have been made by the neurologists. This case emphasizes again the importance of early and accurate examinations of the visual fields.

In contrast to case 8, the classic signs of a tumor of the third ventricle were present in case 18, but the tumor was in the posterior part of the temporal lobe. The left homonymous hemianopia was the only correct localizing sign, but in the presence of neurologic signs which seemed so definite, little value was placed on it. This can only reemphasize the value of early and correctly plotted visual fields.

Perhaps the loss of upward gaze must be reevaluated as a symptom of tumors of the third ventricle. Of 7 cases of tumor of the third ventricle reported by Globus,¹³ loss of upward gaze was present in only 1. It seems to be more constantly a sign of tumors of the pineal body, which as a rule give rise to higher intracranial pressure. It may therefore be a sign of remote pressure effects, possibly a generalized pressure on the posterior longitudinal fasciculus.

CASE 19—E. A., a woman aged 50, was admitted to the Brooklyn Hospital on Feb 4, 1933. She had never been ill until two months before, when she was awakened at night with a severe headache. The headache had been constant since then.

Three weeks before admission to the Brooklyn Hospital she was admitted to a local hospital, where six teeth were removed, with amelioration of the symptoms. A few days later the headaches recurred, more severe than before. For the past one and a half weeks her vision had blurred and the left eye turned in. Vision rapidly became worse, so that she had only preception of light.

On admission to the hospital the intern noted right hemianopia. The next day the neurologist made the following note: "Because of the poor condition of the patient and the poor vision, localization is not possible. In view of the intern's findings, a left temporal lobe tumor is a likely diagnosis."

Ophthalmologic examination on the following day showed the following facts. The patient was uncooperative and restless. She was able to count fingers at 6 inches (15 cm). There was palsy of the left external rectus muscle. Both disks

13 Globus, J. H., and Silverstone, S. M. The Diagnostic Value of Defects in the Visual Fields and Other Ocular Disturbances Associated with Supratentorial Tumors of the Brain, *Arch Ophth* 14 325 (Sept) 1935.

were swollen about 5 or 6 D, and there were numerous hemorrhages scattered about the fundi. Gross tests of the visual fields, taken under great difficulty, revealed definite left hemianopia. This was confirmed the next day. Fifteen minutes after this examination the patient lapsed into unconsciousness, from which she did not emerge until after the operation.

Solely on the basis of the findings in the visual fields, Dr Browder on February 7 turned down an osteoplastic flap in the right temporoparietal region and removed a solid tumor from the right inferior parietal area. This proved to be a fibrillary astrocytoma.

CASE 20—E. H., a woman aged 34, was admitted to the Brooklyn Hospital on Jan 10, 1938, complaining of attacks of severe pain in the face and head.

Her illness started in March 1937, with what she stated was a "nervous breakdown." This was characterized by severe headaches, so that she had to give up her work as a school teacher. In April 1937 she had a facial paralysis on the left side, from which she recovered. At that time she had mild attacks of pain in both cheeks. Since November these had been constant and more severe, coming on

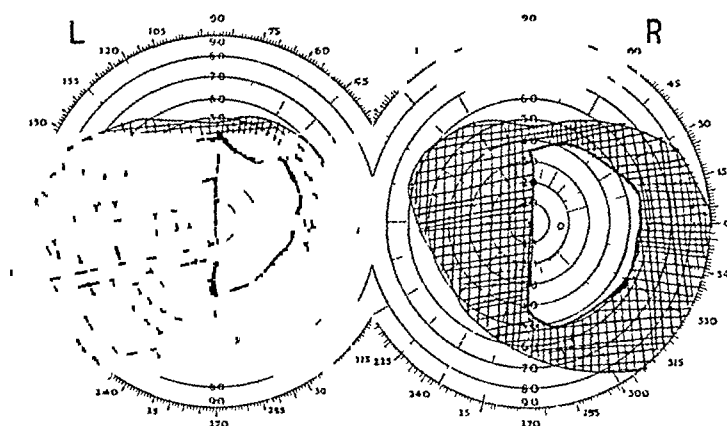


Fig 20 (case 19)—Visual fields of E. A., taken on Feb 5, 1933, with the gross finger test.

about a dozen times a day. The pain was sharp. There had been no visual or auditory disturbances. Around December 25 there was a period of vomiting, which lasted for two days. Since the occurrence of the facial paralysis, speech had always been somewhat thick.

In 1936 a physician made a diagnosis of neuritic atrophy of both disks and referred the patient to a hospital in New York, where roentgenograms of the skull were taken. The report, dated May 5, 1936, stated that there were areas of calcification in the left occipital lobe, with moderate accentuation of markings in the lateral frontal area. No interpretation of the findings was made, and nothing further was done.

The attacks of pain in the face were always brought on by cold weather, particularly drafts. The patient was observed during several of these attacks. The facial muscles became rigid, especially those of the lips, which caused the face to become "puckered." The upper lip especially was stiff, and speech became "tongue-tied," or like "baby talk."

Apprehension was impaired, in that the patient was unable to explain the nature of her husband's work, and although a teacher of languages, she was

unable to parse simple verbs. She also found great difficulty in explaining things in detail.

Neurologic examination revealed the following symptoms: (1) paralysis of the right side of the face of the supranuclear type, (2) a peculiar dysarthric speech, (3) slightly hyperactive deep reflexes, (4) completely normal sensory findings and (5) no word blindness of any kind.

A diagnosis of tumor of the left occipital lobe was made.

A flat roentgenogram taken January 11 showed a calcified mass in the left occipital lobe. The sella turcica was enlarged, and the posterior clinoid processes were eroded. There was wavy unevenness of density in the frontal area.

On the basis of the roentgenographic picture, it was thought that the condition was a tumor of the left occipital lobe. Ocular examination on January 14 showed vision of 13/30+ in the right eye and 13/70+ in the left eye. The pupils were equal and regular and reacted to light and in accommodation. There was no nystagmus or ocular palsy. Examination of the fundi showed the disks to be swollen about 3.5 D. There were no hemorrhages or exudates. The visual fields showed right homonymous hemianopia, which in part cut through the maculas.

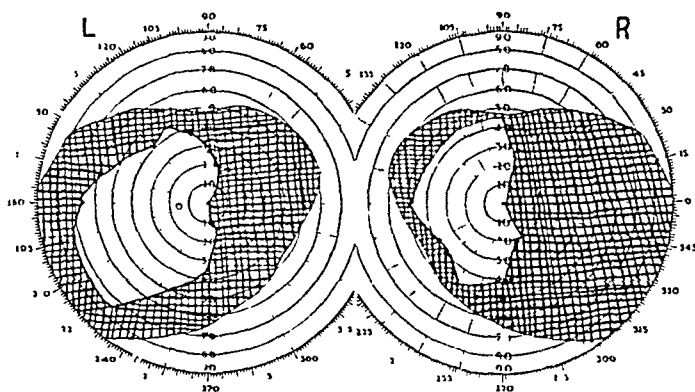


Fig. 21 (case 20)—Visual fields of E. H., taken on Jan. 14, 1938, with a 2 mm white test object at 330 mm. Vision in the left eye was 13/70+ and the right eye, 13/30+.

On the basis of the results of the ocular examination, it was thought that the condition was a tumor of the left occipital lobe.

On January 17 Dr. E. J. Browder turned down an osteoplastic flap in the left occipital region. An encapsulated cystic tumor was found, and from 8 to 10 cc of yellow fluid was removed. The tumor mass was a whitish yellow, similar in color, but of firmer consistency, to the brain. Blunt dissection showed the tumor to have pushed the parietal region in front of it rather than to have invaded it. The entire left occipital lobe was removed.

Pathologic examination proved the growth to be an astrocytoma.

GROUP 4 TUMORS OF POSTERIOR FOSSA

CASE 21—J. A., a boy aged 14, was seen on Feb. 24, 1938, with a history that about five years previously he had been struck on the head with an oil drum. The parents stated that he was unconscious for a little while but was not taken to a hospital. He was perfectly well until about a year before the present trouble, when he began to complain of headaches. Little attention was paid to his complaints, as

they were not severe. During the previous summer he had fallen and was unconscious for a while. Three months before he had a severe spell of vomiting, which lasted for three weeks. At that time he had a cold with fever. A physician was called, and he found pus and sugar in the urine. Another physician could not corroborate these findings. For the past few months he had been acting peculiarly. He had gained about 17 pounds (17 Kg) in the past two months. Recently he had been having sensations of falling backward while walking.

Ocular examination showed that vision in the right eye was limited to perception of fingers at 2 feet. Vision in the left eye equaled 20/70. There was no nystagmus or ocular palsy. Both pupils were dilated but reacted to light and in accommodation. There was about 6 D of swelling of the right disk and 5 D of swelling of the left disk. The arteries were extremely small, and the veins were dilated, but no hemorrhages were visible. Both disks showed evidences of advanced secondary atrophy. The right visual field showed an extreme concentric contraction to a large test object (40 mm white), while the left showed a concentric contraction to a 10 mm test object.

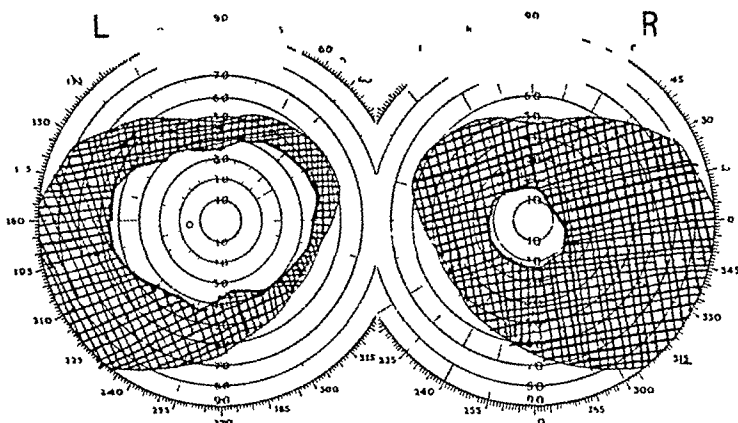


Fig. 22 (case 21) —Visual fields of J. A., taken on Feb. 24, 1938, with a 10 mm white test object for the left eye and a 40 mm white test object for the right eye. Vision in the left eye was 20/70, that in the right eye was limited to perception of fingers at 2 feet.

A diagnosis of tumor in the posterior fossa was made, and the patient was referred to the Brooklyn Hospital, where he was admitted on February 25. A summary of the neurologic findings follows. Roughly, odors were identified. There was a transient and slight paresis of the right seventh nerve of the supranuclear type. There was a drift of both extremities, but the right tended to drift more. There was a slight terminal ataxia in the right finger to nose test. There was a right Babinski sign, and the patient had a tendency to go to the right on walking. He stood equally poorly on either foot. Sensory examination gave negative results. Examination of the left visual field by the intern showed a loss in the nasal portion. The right visual field was not plotted.

On the basis of the results of the ocular examination, it was thought that the condition was a tumor deep in the left parieto-occipital region. This diagnosis was concurred in by the visiting neurologists.

A flat roentgenogram of the skull taken on February 26 revealed the following picture. The bones of the skull seemed thinner than normal. They showed a wavy unevenness of density, and all suture lines were wide. The sella turcica was

not clear. There were two tiny metallic-like foreign bodies, which appeared to be within the skull in the posterior parietal area. On the basis of the roentgenographic picture, it was thought that there was increased intracranial pressure and foreign bodies in the skull.

A ventriculogram taken on March 2 showed air in both lateral ventricles and in the third ventricle. They were markedly dilated but centrally placed. No air was seen in the fourth ventricle, and the iter was not definitely outlined.

The impression was that there was a mass in the fourth ventricle.

That same day, through a cross bow incision in the posterior fossa, a tumor which completely filled the fourth ventricle was exposed. On removal, it was found to be adherent to the medulla in the region of the nuclei of Goll and of Burdach.

CASE 22—F S V, a woman aged 35, was seen on Oct 12, 1935, with a history of tinnitus and deafness on the left side for four years. For the past few years there has also been numbness of the left side of the mouth. Later a neuralgic tic of the right fifth nerve developed. For the past year she had

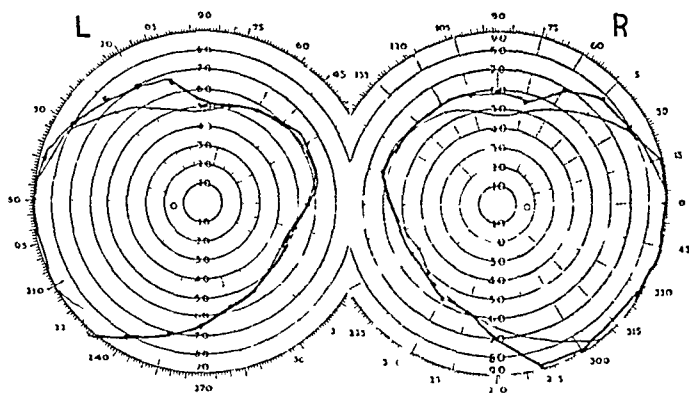


Fig 23 (case 22)—Visual fields of F S V, taken on Oct 12, 1935, with a 2 mm white test object at 330 mm

been having headaches, which for the past four months were fairly constant. Dr E J Biowder had made a diagnosis of a tumor of the left cerebellopontile angle.

Ocular examination showed vision of 20/20 in each eye. There was no ocular palsy and no nystagmus. Both disks were swollen about 1.5 D. The veins were distended, but there were no hemorrhages. The visual fields were normal.

The impression was that the visual fields had no localizing value.

On October 16 Dr Charles Ellsberg, of the Neurological Institute of New York, removed a large meningioma from the left cerebellopontile angle.

CASE 23—J V, a man aged 43, entered the Brooklyn Hospital on Oct 28, 1933, with a history of headaches, vomiting and fainting spells for the past three weeks. These began three weeks previously with sudden, severe temporal and occipital headaches and vomiting.

On November 7 a neurologic examination showed the following facts. There was nystagmus only to the right. No personality changes, no loss of memory, no forced grasping and no convulsive seizures or pareses were noted. There

was no astereognosis or loss of two point discrimination and no uncinate seizures. It was advised that nothing be done until more localizing signs developed.

Ocular examination the same day revealed a history of transient attacks of diplopia. The right disk was swollen about 2 D. The margins of the left disk were slightly blurred. The arteries were thin, and the veins were slightly dilated. No hemorrhages or exudates were present. The right visual field shows a concentric contraction with a defect in the upper temporal quadrant. The left visual field showed a slight concentric contraction.

On the basis of the results of the ocular examination, it was thought that the condition was a tumor deep in the left temporal lobe.

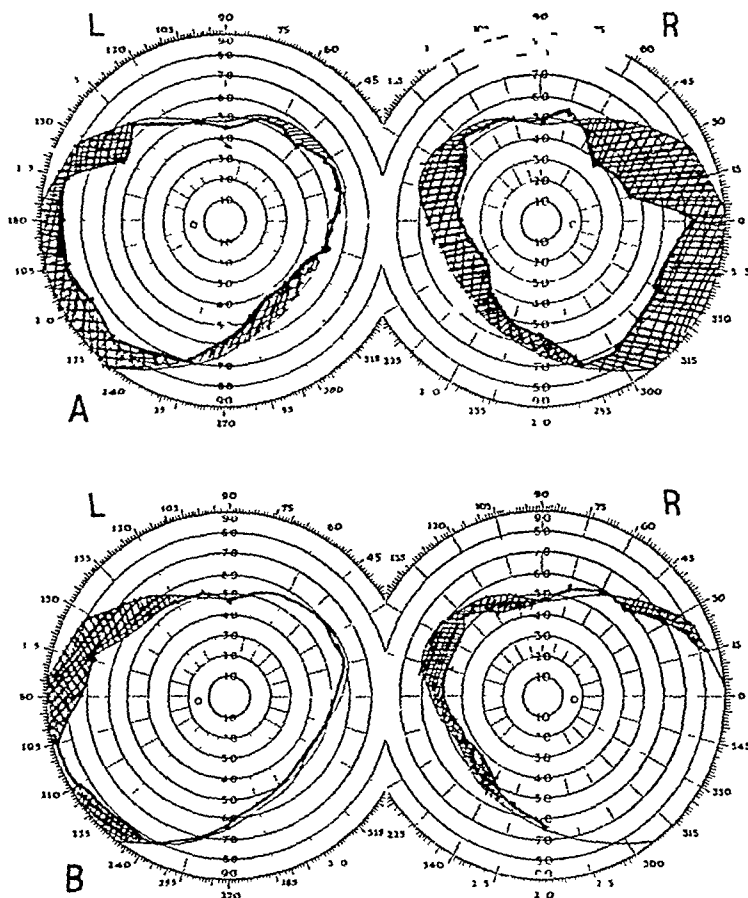


Fig 24 (case 23) —A, visual fields of J V, taken on Nov 7, 1933, with a 2 mm white test object at 250 mm. Vision was 13/13 in each eye. B, visual fields of the same patient, taken on November 16, with a 2 mm white test object at 250 mm. Vision was 13/13 in each eye.

The next day the edema of the right disk had receded, though the margins were still blurred. The left disk showed no change, and there was definite improvement in the visual fields. Because of the improvement, an inflammatory lesion was considered, though a tumor of the brain was not overlooked.

On November 15 the patient became much worse, the nystagmus to the right became more marked and the papilledema increased. Dr Merwarth therefore suggested the advisability of exploration with the possibility of a tumor of the vermis or the subtentorial region being present.

On November 16 ocular examination showed vision of 13/13 in each eye. The right disk was elevated about 4 or 5 D, and there were several hemorrhages around the disk. The left disk showed only a slight increase in blurring. The visual fields showed no localizing defects.

On November 18 a ventriculogram made by Dr Browder showed an obstruction at the base of the brain posteriorly, and that day a tumor of the vermis was removed. An extension of the tumor had become adherent to the brain stem and could not be removed.

CONCLUSION

In 1935 Globus¹³ analyzed 171 cases of verified tumor of the brain. He concluded that the number of cases in which perimetric study was of diagnostic assistance was surprisingly small, but that when characteristic defects were present they were of immense importance. That the conclusion about the relative small percentage of cases in which visual fields were of diagnostic value was not based on solid premises is shown by an analysis of his series.

Of the 171 cases, no visual fields were taken in 56, or 33 per cent, and visual fields were taken only with the gross finger test in 42, or 24 per cent. But even in the latter group the visual fields showed defects of localizing value in 16 instances. In other words, in 58 per cent of his series of cases either no examination or only a superficial examination of the visual fields was made.

Further analysis shows that in 105 of the cases the tumors were found in the temporal, parietal, occipital and chiasmal regions, situations in which one would expect tumors to give rise to significant localizing signs in the visual fields. In 29 of these cases, or almost 28 per cent, no visual fields were taken, and in 30, or slightly over 28 per cent, visual fields were taken only with the gross finger test. In 29, or 63 per cent, of the remaining 46 cases, the visual fields showed signs of localizing value, and even in 15, or 50 per cent, of the 30 cases in which the visual fields were taken with the gross finger test the fields showed defects of localizing value.

Foster Kennedy, in discussing Globus' paper, expressed the view that the low number of instances in which satisfactory fields could be taken was due to the fact that the studies were undertaken late in the clinical course. This is borne out by the series of cases here presented.

In 5 of the 6 cases of tumor of the frontal lobe, the visual fields, correctly, showed no localizing defects. In the other case (6) the visual fields, incorrectly, showed localizing signs. This patient had good vision but his mental condition was subnormal and he cooperated poorly.

Tumors of the frontal lobe are also characterized by a paucity of neurologic signs. In 2 of the 6 cases here presented, neurologic examination showed correctly localizing signs, in 2 it showed no findings of localizing value, and in the remaining 2 it showed incorrectly localizing signs, resulting in wrong diagnoses.

Flat roentgenograms were made in 4 of the cases. In 2, the roentgenograms showed defects of localizing value, in 1, no localizing defects and in the other (case 2), defects which resulted in a wrong interpretation.

Encephalograms were made in 4 cases. In 1 case (3) no evidence of tumor was found.

In but 1 of the 8 cases in group 2 (case 11) the visual fields were of no localizing value. The growth in this case was a rare chordoma. It is interesting that the Greens,¹⁴ of San Francisco, described a similar case which practically paralleled the case here reported, even unto the mistakes in diagnoses made by the ophthalmologist, the neurologist and the roentgenologist. In case 12 a wrong interpretation was made. Here again both the vision and the cooperation of the patient were extremely poor. The defects in the visual fields in the other 6 cases were of definite localizing value.

Flat roentgenograms showed no localizing defects in all 4 cases of suprachiasmal lesions but showed definite defects in the other cases.

Correct interpretations were made on the basis of the encephalographic picture in all 6 cases in which encephalography was carried out.

In the third group of 6 cases the visual fields showed, correctly, defects of localizing value. The results of the neurologic examination were of no localizing value in 3 of the cases and gave correct information in the other 3.

Flat roentgenograms showed defects of correct localizing value in only 1 case, while encephalographic examination gave correct information in the 3 cases in which it was carried out.

In the last group of 3 cases, the visual fields, correctly, showed no localizing defects. Neurologic diagnosis was wrong in 1 case (21). Encephalographic examination gave correct evidence in all 3 cases.

In summary, the visual fields in the 14 cases in groups 2 and 3, which would be expected to present localizing defects, showed signs of definite localizing value in 12, or 86 per cent. From a diagnostic standpoint, a correct negative field may be as important to the neurosurgeon as a positive field, as witness cases 3 and 21. Of the 9 cases of tumor in the frontal and the posterior fossa, an incorrect visual field was found in only 1, so that the visual fields in 20 of the 23 cases here presented were correct.

Neurologic examinations, usually made by several observers, gave correct information in 16 cases. In 4 cases the diagnoses were wrong and in the other 3 neurologic signs were of no localizing value. Flat roentgenograms were taken in 13 cases. In 6, the roentgenographic

¹⁴ Green, A. S., and Green, M. I. Intracranial Chondroma, *Bull. Pract. Ophth.* 7: 85, 1937.

picture was either of no localizing value or gave incorrect information. Encephalograms were made in 15 cases, and in only 1 was the diagnosis missed. However, in 2 cases all other examinations gave incorrect information. The time that elapsed between the onset of symptoms and the diagnoses in those cases in which vision or mentality or both were subnormal varied from three weeks in case 18 to thirteen years in case 10. The longest span of time was in group 2, with an average of six and one half years, the shortest span of time was in group 3, with an average of twenty weeks.

While I do not attempt to say that examinations of the visual fields are of such value that other methods of examination could be dispensed with, I do feel that if done early enough they are of extreme importance. This is especially true in cases of tumor of the pituitary region and of the temporal and the parietal area. If every patient with persistent headache or with other signs suggestive of possible tumor of the brain had an examination of the visual fields, many more instances would be found in which the visual fields are of localizing value. If the visual fields at first are negative and the symptoms persist or are progressive, further studies should be made. It makes no difference if the symptoms are of three weeks' duration or are stretched out over a period of years, visual fields should be taken before blindness or mental deterioration develops. It has been shown how quickly blindness may occur once ocular symptoms develop.

I cannot say that all neurologic examinations should be dispensed with, because in this series they gave correct results in only 70 per cent. Yet I recently looked through the records of a large number of cases of tumor of the brain, and the percentage of cases in which proper visual fields were taken was surprisingly small, about the same percentage as was reported in the series by Globus.

In addition, one should consider the value of positive findings in the visual fields. If the results of the neurologic examination check with findings in the visual fields, it may often make encephalographic or ventriculographic study unnecessary, and for a person with tumor of the brain who is ill, it may mean the margin of safety. However, as shown in case 5, encephalography, when possible, should be carried out.

In closing, I shall say that I feel that examination of the visual fields is an indispensable aid in the diagnosis of tumor of the brain and should be resorted to early and more frequently.

Clinical Notes

RETINITIS PIGMENTOSA WITH "HOLE" IN THE MACULA

Report of a Case

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Pigmentary degeneration of the retina is a familial and hereditary disease in which the peripheral portion of the retina is most affected. Examples of this condition associated with central retinal changes are uncommon. A search of the literature reveals reports of a number of cases of typical or atypical retinitis pigmentosa combined with disturbances at the macula. Among the writers who have described such cases are Leber,¹ Carruthers,² Rieger,³ Spare⁴ and Pavia and Dusseldorp.⁵ The ophthalmoscopic picture of a "hole" in the macula in association with retinitis pigmentosa is rare. Leber⁶ in his monumental work on diseases of the retina discussed the presence of macular cysts in association with pigmentary degeneration of the retina and referred to cases reported by Hoffman (1885), Meyer (1889), Noll (1908) and Stock (1908). Leber's article included a photomicrograph of a section of the eye of Stock's patient, taken through the macula, showing multiple cysts of the internal nuclear layer of the retina and a large cyst in the fovea.

The patient whose case is reported here was brought to Dr. Charles H. May and has since been under our joint care.

CASE REPORT

Miss A. M. G., an Italian girl of 16, was seen in December 1935, complaining of night blindness and defective peripheral vision for nearly three years. She had consulted several European oculists, who had made the diagnosis of retinitis pigmentosa. Her parents were first cousins. The eyes of her parents

Presented before the New York Academy of Medicine, Section of Ophthalmology, May 16, 1938.

1. Leber, T. Ueber anomale Formen der Retinitis pigmentosa, *Arch. f. Ophth.* **17** 314-341, 1871.

2. Carruthers, J. F. Case of Retinitis Pigmentosa (Atypical), with Oedema of the Macula, *Proc. Roy. Soc. Med. (Ophth. Sect.)* **9** 103-105, 1915-1916.

3. Rieger, H. Ein Beitrag zur Kasuistik der tapeto-retinalen Degeneration, *Ztschr. f. Augenh.* **57** 429-463, 1925.

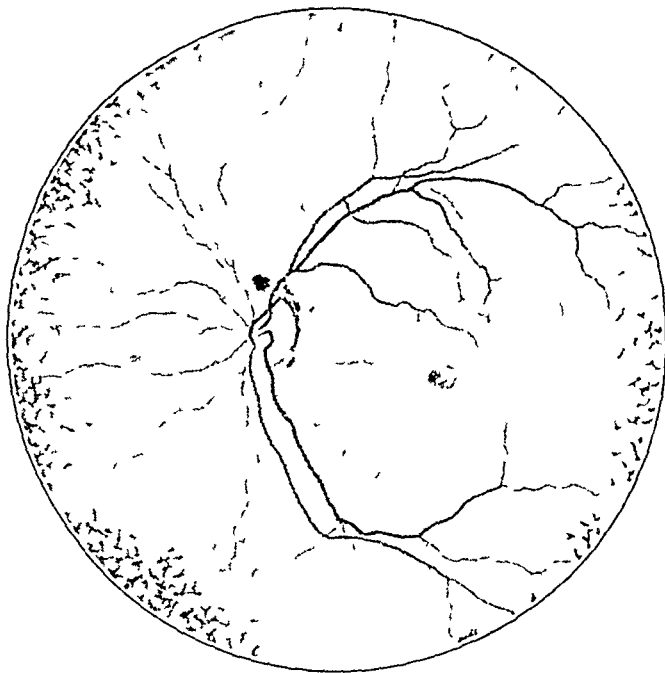
4. Spare, A. Tapetoretinal Degeneration of the Retina. Case Report, *Am. J. Ophth.* **11** 545-550, 1928.

5. Pavia, J. L., and Dusseldorp, M. Degeneracion pigmentaria de la retina con alteraciones de la region macular, *Rev. oto-neuro-oftal.* **9** 118-125, 1934.

6. Leber, T. Die Krankheiten der Netzhaut, in Graefe, A. and Saemisch, E. T. *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, pp. 1680-1682.

and those of her 2 brothers and 3 sisters were normal. The family history was otherwise irrelevant.

Physical examination showed a healthy young girl with no abnormalities except those found in a study of her eyes. Vision was 15/20 in each eye with correcting lenses (right eye, -3.50 sph -0.75 cyl, axis 15, left eye, -0.75 sph -0.75 cyl, axis 135). The ocular media were clear except for a few small opacities in the vitreous. Both fundi revealed the typical picture of retinitis pigmentosa, with narrowed vessels and peripheral deposits of pigment. Several small masses of pigment were seen adjacent to the left disk. The optic nerve heads showed limited atrophy, most evident in the left eye. The intra-ocular pressures and muscle balance were normal. The visual fields with a 3 mm white test object at 300 mm were contracted to 20 degrees from the fixation point.



Fundus of the left eye of a patient with retinitis pigmentosa, showing a "hole" in the macula and deposits of pigment adjacent to the nerve head.

During the following year and a half, the patient was seen at intervals of from four to six months, with no changes in the ocular picture. She was attending school and planned to enter college.

In June 1937 she reported to our offices, complaining of blurred and distorted vision of the left eye for several weeks. Examination showed the vision of this eye to have been reduced to 15/60. Examination of the left fundus revealed slight edema of the retina around the macula and a round faint reddish area in the fovea, suggestive of a beginning "hole." There was a small central scotoma in the field of the left eye. The right eye and the peripheral fields of both eyes were unchanged. Careful questioning elicited no history of trauma. Search for foci of infection was fruitless. The results of all laboratory tests, including a Wassermann test of the blood, were negative.

Within three months the vision of the left eye decreased to 15/400. The lesion in the left fovea took on the characteristic appearance of a hole, which has increased in size to a slight extent during the six months preceding the

writing of this report. The right eye has remained unchanged. The visual fields now show a large absolute central scotoma in the left eye but no further contracture. The accompanying photograph of a painting of the fundus by Mr Bethke depicts the lesions of the left fundus in December 1937.

COMMENT

The frequency of macular involvement in association with diseases of the retina is explained by the delicate and highly organized structure of this region and by the sluggish circulation due to absence of blood vessels. These factors make the macula particularly vulnerable to toxic and circulatory disturbances.

The so-called hole of the macula represents in most instances the ophthalmoscopic appearance of conglomerate or confluent cysts and has been reported to occur following contusion of the eyeball, penetrating injury of the globe, detachment of the retina, intraocular inflammation and vascular disease. The typical macular "hole" develops from cystic degeneration and is rarely the result of a retinal tear.

Most authors believe that the cystic changes leading to the formation of a "hole" have their origin in retinal edema and in the disappearance of retinal elements from insufficient nutrition. The pathologic picture of macular "holes" has been reviewed by Coats,⁷ Samuels⁸ and Reese.⁹ The last-named writer stated: "There may be cystic formations in the macula, usually in the early stages of the disease (retinitis pigmentosa). These may be transitory and are usually attributed to edema."

Schneck¹⁰ divided diseases of the macula into those due to extramacular processes (injuries, inflammations, glaucoma, myopia, thrombosis of the central vein of the retina, etc.) and those which occur without extramacular disease. He further subdivided the latter group into hereditary and senile or atherosclerotic diseases, including among the hereditary diseases the spontaneous, bilateral condition which Behr had described under the title of heredodegeneration of the macula (congenital, juvenile, adult, presenile and senile).

Pigmentary degeneration of the retina and macular dystrophies are classified together by many writers under the progressive dystrophies or abiotrophies. The latter term was coined by Gowers and refers to premature senility of the involved tissue due to defective vitality. Treacher Collins¹¹ wrote at length on the abiotrophic origin of retinitis pigmentosa, although the discussers of his paper (Mayou, Greeves,

7 Coats, G. The Pathology of Macular Holes, Roy. London Ophth. Hosp. Rep. **17**: 69-96, 1907.

8 Samuels, B. Cystic Degeneration of the Retina, Arch. Ophth. **4**: 476-486 (Oct.) 1930.

9 Reese, A. B. Defective Central Vision Following Successful Operations for Detachment of the Retina, Am. J. Ophth. **20**: 591-598, 1937.

10 Schneck, F., in Henke, F., and Lubarsch, O. Handbuch der speziellen Pathologie, Anatomie und Histologie, Berlin, Julius Springer, 1928, pt. 1, pp. 658-667.

11 Collins, E. T. Abiotrophy of the Retinal Neuro-Epithelium or "Retinitis Pigmentosa," Tr. Ophth. Soc. U. Kingdom **39**: 165-195, 1919.

A Crichtett, Morax and Parsons) expressed the opinion that vascular disease of the retina and choroid played a major role. Mann¹² described retinitis pigmentosa as a latent fault leading to the appearance of a postnatal degenerative process and related genetically to lesions of various regions of the fundus (including macular degeneration). She looked on both retinitis pigmentosa and macular degeneration as regional failures, with abnormal ectodermal and mesodermal elements.

The formation of a macular "hole" has been watched by many ophthalmologists, but only a few have reported their observation of the developing lesion. Crawford¹³ described an instance of this. I have recently studied the fundi of 2 elderly persons with senile degeneration of the macula and have watched the formation and development of a typical "hole" in one eye of each patient.

In the absence of more definite knowledge of the causation of retinitis pigmentosa and juvenile macular degeneration, it seems logical to consider the two processes described in this paper as due to the effects of vascular and nutritional disturbances on a congenitally abnormal and defective retina. If this is true, one must accept the possibility of future central degenerative changes in the retina of the eye with the uninvolved macula.

SUMMARY

Examples of retinitis pigmentosa associated with macular changes are uncommon. Instances in which a "hole" occurs in the macula are exceedingly rare. This combination of conditions is reported in a 16 year old Italian girl whose parents were first cousins. The macular lesion developed in one eye while the patient was under observation. I believe that the peripheral and central degenerative lesions in the retina of this patient were the results of local vascular disease and retinal deterioration. The relevant literature is reviewed.

12 Mann, I. Developmental Abnormalities of the Eye, Cambridge, England, Cambridge University Press, 1937.

13 Crawford, J. W. Hole in the Macula. Report of a Case in a Patient Under Observation, Arch Ophth **10** 793-799 (Dec.) 1933.

HYPERSENSITIVITY TO LAROCAINE

FREDERICK H. THEODORE, M.D., NEW YORK

Larocaine hydrochloride, *p*-aminobenzoyl-2,2-dimethyl-3-diethylamino-propanol hydrochloride, has gained considerable popularity as a local anesthetic in the United States and abroad in the past five years. It has been used successfully for many surgical procedures, particularly for those in the fields of otolaryngology and ophthalmology.¹ It is used satisfactorily in a 2 to 5 per cent solution for instillation into the eye.

1 Rich, B. Larocaine. A Summary, M. Rec **144** 419-423 (Nov. 4) 1936.

2 Mayer, L. Larocaine. A New Anesthetic, Arch Ophth **14** 408-411 (Sept.) 1935.

For subcutaneous injection, solutions varying in strength from 1 to 10 per cent have been used. Up to the present no ill effects from this drug have been reported. Because of this, it was thought worth while to report the following case of hypersensitivity.

REPORT OF CASE

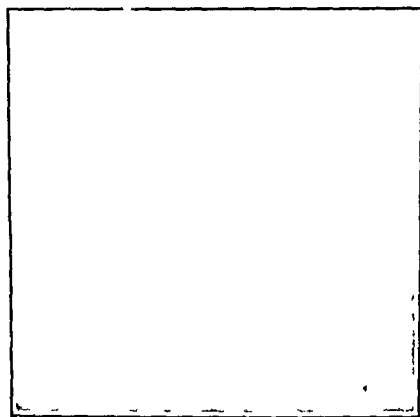
M T, a woman aged 70, was first examined in May 1937 because of opacities of the vitreous which had been annoying her. She had a high degree of myopia. Mild chronic conjunctivitis was noted as an incidental finding. Her past history was unimportant, except that in November 1934, during the course of a tinea infection of the skin, at the time unrecognized, she had undergone complete allergic tests and had been found sensitive only to chocolate. Six years previously a small dose of sodium barbital (soluble barbital U S P XI) had given her a rash.

She returned on December 2, because of acute conjunctivitis in each eye. Smears of conjunctival material revealed no organisms. Treatment consisted of the topical application of a 1 per cent solution of silver nitrate preceded by the instillation of a 2 per cent solution of larocaine hydrochloride. The patient was instructed to use a 25 per cent solution of mild protein silver at home. She returned one week later, considerably improved but not entirely well. A 1 per cent solution of silver nitrate, preceded by anesthetization with a 2 per cent solution of larocaine hydrochloride, was again applied. When the patient was next seen it was apparent that she was much worse. A number of fine punctate superficial corneal infiltrations had appeared in each eye, which stained with fluorescein. Again a solution of silver nitrate was used after anesthetization with larocaine hydrochloride. In the course of a week, during which almost daily treatments first with silver nitrate and then with the light application of a copper stick were given, each time after anesthetization with larocaine hydrochloride, the condition became increasingly worse. Finally, there was considerable massive edema of the eyelids and the upper half of the face, which, combined with swelling of the parotid glands due to stenosis of the parotid ducts, gave a leonine appearance to the patient. Both the bulbar and the palpebral conjunctiva were now diffusely injected, and there was much mucopurulent discharge, which was free from organisms. In the left eye, at the limbus, there was a complete ring of marginal infiltration of the cornea due to the coalescing of discrete catarrhal ulcers. In the right eye the marginal ulcers were less extensive. The corneal epithelium of each eye showed myriads of fine punctate infiltrations.

It was apparent that the treatment was aggravating the condition, and at this stage, fortunately, medication was stopped, and the patient was instructed to use a mild eyewash. In a few days she was much better, and within five days all the corneal lesions had disappeared. Thereafter, applications of a mild solution of silver nitrate, from 0.16 to 0.33 per cent, without the previous use of larocaine hydrochloride, were employed with benefit. On Feb 10, 1938, in the course of examining the right lacrimal passage, 1 drop of a 2 per cent solution of larocaine hydrochloride was instilled into the right eye only. Within a few hours the eye began to bother the patient, and twenty-four hours later both the bulbar and the palpebral conjunctiva were found to be diffusely inflamed, and two catarrhal corneal ulcers the size of a pinhead were present. Again, with the use of mild eyewashes the condition cleared rapidly.

It now appeared clinically certain that hypersensitivity to larocaine had been responsible for the entire disturbance from the beginning. Patch tests were made

with 10 per cent larocaine hydrochloride in an oxycholesterol petrolatum ointment base. There was no itching till twenty-four hours had elapsed. When the patch was removed after forty-eight hours, the skin was erythematous and indurated, and numerous vesicles were observed. Control tests with the ointment base and several other anesthetic ophthalmic ointments were negative. The sensitized skin became more inflamed, and the epidermis sloughed. One month later it had not entirely healed. All the previous solutions of larocaine hydrochloride and the ointments containing the drug had been made from the commercial tablets. In order to rule out any other factor, the patch tests were again done with pure crystalline larocaine hydrochloride³. This time ointments containing 0.5 and 0.1 per cent larocaine hydrochloride were used. With 0.5 per cent ointment there was a markedly positive reaction in forty-eight hours, with much vesiculation and, later, epidermal sloughing. This was even more marked than with the ointment which contained 10 per cent larocaine hydrochloride. Even the 0.1 per cent ointment produced a little erythema and then a few vesicles. These patch tests confirmed



Positive reaction after seventy-two hours to a patch test on the inner aspect of the arm with the 0.5 per cent larocaine ointment

the clinical impression that an increasing sensitivity to the drug had been occurring

COMMENT

Hypersensitivity to larocaine hydrochloride must be unusual, and its rare occurrence should not prejudice its position as an excellent anesthetic. Similar anesthetics likewise at times cause unpleasant reactions⁴. Had it been realized, however, that this drug is capable of producing untoward effects, considerable suffering might have been prevented. Moreover, the occurrence of constantly increasing sensitivity during the period of its administration may be of importance and should be noted. An appreciation of these facts is even more important in those cases in which larocaine hydrochloride may be used as an infiltration anesthetic, which, conceivably, might give rise to more serious consequences

³ This was supplied by Hoffmann-La Roche, Inc

⁴ Pfeiffer, R. Hypersensitivity to Pontocaine, *Arch Ophth* 18 62-64 (July) 1937

Ophthalmologic Review

EDITED BY DR FRANCIS HEED ADLER

BINOCULAR VISION AND ORTHOPTIC PROCEDURE

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The clinical application of orthoptic procedure in the treatment of imbalance of the ocular muscles has led to the publication of various reports which, because of differing criteria of success, incomplete records and inadequate control groups, show inconsistent results and justify skepticism as to the legitimate place of orthoptic training in ophthalmology. Indiscriminate use of ocular exercise by unqualified persons has contributed to the popular misconception of orthoptic procedure as a substitute for glasses or surgical treatment rather than a supplementary technic useful to the ophthalmologist for the diagnosis and treatment of defects of binocular vision. The physiologic basis for binocular vision and the clinical diagnosis of its anomalies will be briefly reviewed as a preface to a more detailed discussion of actual orthoptic procedure, which may contribute to more careful clinical work and at the same time indicate the need for controlled laboratory research in this field.

THE BASIS OF BINOCULAR VISION

Phylogenetically, binocular vision is correlated with the forward placement of the eyes, evident first in carnivora, giving overlapping fields of vision. Semidecussation rather than complete crossing of the optic fibers at the chiasm, giving bilateral cerebral representation of the eyes in higher forms, occurs in such a manner that the right portion of each retina is represented in the right cerebral hemisphere, while the left portion of each retina is represented in the left hemisphere¹. Ontogenetically, in the prenatal development of the human eyes, the angle between the optic axes is reduced from 180 degrees to about 60 degrees². The neurologic foundation for binocular fixation is present from birth, and though meager experimental evidence indicates a gradual develop-

From the Orthoptic Clinic of the Manhattan Eye, Ear and Throat Hospital

1 Adler, F H. Clinical Physiology of the Eye, New York, The Macmillan Company, 1933, p 230

2 Mann, I. Development of the Human Eye, Cambridge, University Press, 1928, pp 43 and 264

ment of coordinate movements during the first year, one cannot assume an arbitrary age at which binocular vision in all its aspects has reached maturity

Assumptions concerning the exact projection of the visual fibers in the occipital cortex have been based on evidence derived from defects in the visual field and localized central lesions resulting from gunshot wounds and other injuries. Rearrangement of the fibers in the lower part of the optic tracts is believed to take place in such a way that fibers from corresponding parts of the two retinas lie near each other,³ but since the time of Sherrington⁴ it has been assumed that fusion occurs in the final projection areas of the cortex. The typical sparing of the macula in severe defects of the visual field has given support to the widely accepted theory that the macular fibers have bilateral cortical representation, though Fox and German⁵ demonstrated widespread distribution of the macular fibers, which may make it unnecessary to postulate bilateral macular projection to account for such phenomena.

The earliest theories of fusion assumed actual connection of nerve fibers in or near the chiasm or alternation of the two eyes, so that one eye at a time actually responds to visual stimulation.⁶ Muller's postulation of "corresponding points,"⁷ which were assumed to have anatomically identical projection fibers, was widely accepted, though it is evident from simple demonstrations of physiologic diplopia that numerous double images must be suppressed at all times. Anatomic evidence of point to point correspondence in the central projection areas has never been shown. Moreover, Peckham⁸ proved that single vision is maintained when images are far from "corresponding" points. Henschen⁹ demonstrated that early loss of both eyes results in atrophy of both the superficial and the deep layer of cells in the area striata, leaving the middle, functionally independent layer intact, whereas early loss of one eye results in atrophy of the deep layer of cells on the opposite side of the brain and the superficial layer of cells on the same side of

3 Adler,¹ p 155

4 Sherrington, C S. Integrative Action of the Nervous System, New Haven, Conn., Yale University Press, 1911, p 381

5 Fox, J C, Jr, and German, W J. Macular Vision Following Cerebral Resection, *Arch Neurol & Psychiat* **35** 808 (April) 1936

6 Helmholtz, H L F. Helmholtz's Treatise on Physiological Optics, English translation, edited by J P C Southall, Rochester, N Y, The Optical Society of America, 1925, vol 3, p 482

7 Muller, J. Zur vergleichenden Physiologie des Gesichtsinnes des Menschen und der Thiere, Leipzig, Cnobloch, 1826

8 Peckham, R H. Foveal Projection During Ductions, *Arch Ophth* **12** 562 (Oct) 1934

9 Henschen, S E. Zur Anatomie der Sehbahn und des Sehzentrumms, *Arch f Ophth* **117** 403, 1926

the brain. Thus the middle layer of cells, bordered by cells connected with functionally related parts of each eye, is probably essential for fusion. These cells are fully developed at birth.

Verhoeff¹⁰ proposed a theory of binocular vision in which he accepted the assumption of corresponding points, which are presumably represented anatomically in the occipital lobes in close proximity, yet separately. He presented convincing evidence from stereoscopic charts that one eye is dominant for some parts of the visual field while the other is dominant for other parts, so that single impressions from corresponding retinal images are really made up of parts of one image and parts of the other or, in some cases, all of one image or all of the other. In stereopsis, in which there is apparent union of disparate images (those falling on noncorresponding retinal areas), Verhoeff demonstrated that one of the disparate images is completely replaced by its substitute without disturbing the effect of depth.

In addition to the empiric factors in the judgment of distance which operate for monocular vision alone, such as apparent size, motion, interposition, shadows, perspective and parallax by movements of the head, and possibly accommodation, in normal binocular vision the factor of binocular parallax, based on disparate images and enhancing the appearance of depth, acts for finite distances.¹¹ True binocular vision requires the presence of bifoveal fixation, a binocular field mediated by uncrossed retinal fibers, similar retinal images and approximately equal acuity in the two eyes.¹² Stereopsis is precluded by a difference in visual acuity when the ratio amounts to more than 6/6 to 6/9 according to Adler,¹³ though Verhoeff¹⁴ suggested a kinetic test for stereoscopic vision which is successful when the acuity is as low as 6/200 in one eye or in both eyes.

The maintenance of binocular vision in various fields of fixation and at various distances is dependent on the normal functioning and coordination of the intraocular and extraocular muscles. Cortical centers correlated with voluntary ocular movement have been demonstrated by electrical stimulation of the exposed cortex of the frontal lobes, centers for involuntary ocular movement, induced by visual stimuli, are situated in the occipital lobe.¹⁵ The extrinsic ocular muscles have been shown by experiment to exhibit the phenomenon of reciprocal innervation, the

10 Verhoeff, F. H. A New Theory of Binocular Vision, *Arch. Ophth.* **13** 151 (Feb.) 1935.

11 Helmholtz,⁶ p. 371.

12 Adler,¹ p. 229.

13 Adler,¹ p. 241.

14 Verhoeff, F. H. Kinetic Test for Stereoscopic Vision, *Arch. Ophth.* **15** 835 (May) 1936.

15 Adler,¹ p. 294.

contraction of one muscle being accompanied by simultaneous relaxation of its antagonist. The third, fourth and sixth cranial nerves, controlling the ocular muscles, are subject to the all or none law, the size of the response being dependent on the number of stimulated fibers in each motor nerve.¹⁶ The neural center for convergence, a disjunctive ocular movement, is assumed to be the median group of cells (Perlia's nucleus) in the nucleus of the third nerve,¹⁷ no anatomic center for divergence has been demonstrated. In emmetropic eyes, which are orthophoric, the amount of convergence in meter angles¹⁸ is theoretically equivalent to the amount of accommodation in diopters. There is evidence that the two functions, innervated by the third nerve, have become anatomically interrelated by the process of neurobiotaxis,¹⁹ so that accommodation causes simultaneous convergence. That the two functions can be dissociated to some extent is evident from the fact that refractive errors which increase or decrease the amount of accommodation necessary for clear vision may be present without disturbing the coordination of convergence. Nevertheless, complete dissociation of the two functions is not physiologically possible, and the application of orthoptic training is limited by this fact.

Worth's arbitrary classification of grades of fusion, based on artificial criteria, is in general use in discussions of binocular vision.²⁰

1 First grade fusion simultaneous binocular perception (This grade of fusion has been subdivided by some clinicians into two types simultaneous perception without superposition of images and simultaneous perception with superposition of images²¹ It is doubtful if binocular perception without superposition, i e, in which the observed images are widely separated subjectively, can be called fusion in the usual sense.)

2 Second grade fusion simultaneous binocular perception with effort to maintain fusion by ocular movements when fixation is changed ("amplitude" of fusion)

3 Third grade fusion stereopsis, or sense of depth mediated by binocular cues

ANOMALIES OF BINOCULAR VISION

Orthophoria, or normal muscle balance, like emmetropia, is the exception, since careful tests reveal heterophoria in over 80 per cent of cases,

16 Adler,¹ p 301

17 Adler,¹ p 298

18 Adler,¹ p 274

19 Stutterheim, N A. Indications for the Kinetic Treatment of the Eyes, London, G Pulman & Sons, 1931, p 10

20 Worth, C. Squint, ed 5, London, Bailliere, Tindall & Cox, 1921, p 12

21 Lyle, K, and Jackson, S. Practical Orthoptics in the Treatment of Squint, London, H K Lewis & Co, Ltd, 1937, chap 7

according to Bielschowsky²² Clark²³ found deviations from normal for the near point in 90 per cent of 191 college students tested. Mild degrees of muscle imbalance which cause no symptoms may be considered within normal limits. According to Berens, Losey and Hardy,²⁴ deviations for distance should not exceed 3 diopters of esophoria, 2 diopters of exophoria or 1 diopter of hyperphoria, deviations for the near point should not exceed 1 diopter of esophoria, 6 diopters of exophoria or 1 diopter of hyperphoria. Heterophoria, or latent deviation, demonstrable only by tests which dissociate the eyes by preventing fusion, probably precedes heterotropia, in which there is a manifest deviation, frequently called "strabismus" or "squint." The constant innervation necessary to maintain binocular vision in the presence of heterophoria may give rise to marked subjective symptoms, whereas no effort is made to maintain binocular vision in the presence of heterotropia, and symptoms consist of cosmetic defect or low visual acuity. The causes of nonparalytic strabismus have offered a fertile field for speculation, and present day treatment assumes no single factor, such as refractive error or fusion deficiency, as responsible for the defect.

No essential neurologic difference between monolateral and alternating strabismus has been demonstrated. That there is probably a close relation between the two types is evident from the fact that many persons who have alternating fixation at first later have a strong preference for monolateral fixation and persons who have monolateral fixation can be taught to alternate. Clinicians are agreed that patients with spontaneous alternating fixation, with equal acuity in the two eyes and, frequently, insignificant refractive errors, present greater difficulties in correction by optical, surgical or orthoptic methods than those who have a strong preference for monolateral fixation. It has been suggested that alternating fixation is accompanied by more firmly established functional changes²⁵ than those which accompany monolateral strabismus, but there may be central neurologic factors as yet unobserved which contribute to alternating fixation and interfere with successful treatment.

Significant functional changes which occur in cases of strabismus are suppression, defects in the visual field, amblyopia ex anopsia and anomalous projection. All of these factors operate to facilitate single vision and prevent diplopia, which interferes with accurate orientation. Sup-

22 Bielschowsky, A. Functional Disturbances of the Eyes, *Arch Ophth* **15** 589 (April) 1936

23 Clark, B. Heterophoria in College Students, *Am J Optometry* **12** 9, 1935

24 Berens, C., Losey, R. L., and Hardy, L. H. Routine Examinations of the Ocular Muscles and Nonoperative Treatment, *Am J Ophth* **10** 910, 1927

25 Lyle and Jackson,²¹ p 114

pression characteristic of strabismus is generally assumed to be a central process, though there is some evidence that peripheral mechanisms may contribute to suppression. Fry²⁶ demonstrated that voluntary suppression is accomplished by changes in accommodation, and Javal²⁷ pointed out that in cases of strabismus the deviating eye loses its ability to accommodate accurately for a given point of fixation, and this inaccuracy may simulate a considerable degree of amblyopia. Suppression scotomas have been mapped by Travers in cases of strabismus,²⁸ and Braun²⁹ showed that an absolute scotoma to the temporal side of the fixation spot can be found in cases of monolateral fixation, whereas the fields are normal in cases of alternating strabismus. Prolonged and constant suppression is assumed to cause amblyopia ex anopsia. Most important of the functional changes associated with strabismus of long standing is anomalous projection ("abnormal retinal correspondence," "secondary retinal correspondence," "incongruous retinas" or "false projection"), in which the foveal perception of the fixing eye is associated with peripheral perception of the deviating eye in an inferior type of binocular vision. When both eyes are in use, the projection of the deviating eye is interpreted as if the peripheral image were "fused" with the foveal image of the fixing eye and projected to the same point in space. Acuity of the peripheral region used under such conditions by the deviating eye does not increase above that to be expected in a peripheral retinal area. Except in rare cases of eccentric fixation, sometimes called "false macula," fixation returns to the foveal region in the deviating eye while the fixing eye is covered, even in cases of well established anomalous projection, so that the screen test remains a valid method of measuring the absolute deviation. It should be emphasized that anomalous projection is a binocular phenomenon, alteration in projection occurring for the entire field, as pointed out by Maddox.³⁰ The presence of anomalous projection invalidates measurements made with subjective tests (Maddox rod) in which there is no control of foveal fixation.

In anomalous projection, bifoveal stimulation, possible under artificial conditions, results in paradoxical diplopia. Heteronymous diplopia in

26 Fry, G. A. The Relation of Accommodation to the Suppression of Vision in One Eye, *Am J Ophth* **19** 135, 1936.

27 Javal, L. E. Du strabisme dans ses applications a la physiologie de la vision, Paris, Victor Masson et Fils, 1868, p. 53.

28 Travers, T. B. Concomitant Strabismus, London, G. Pulman & Sons, 1936, p. 46.

29 Braun, G. Gesichtsfelduntersuchungen bei Schielenden, *Klin Monatsbl f Augenh* **42** 600, 1934.

30 Maddox, E. The Ocular Muscles, ed. 2 Philadelphia, Keystone Publishing Company, 1907, p. 125.

convergent deviations, and homonymous diplopia in divergent deviations. Von Graefe³¹ reported postoperative paradoxical diplopia in 1854, it can easily be demonstrated preoperatively by controlled bifoveal fixation. Tschermak³² and others have used the projected foveal after-images as a test for anomalous projection.

It is probable that innate primary retinal correspondence is seldom replaced completely by secondary retinal correspondence since normal projection with bifoveal fixation eventually develops in many cases of postoperative paradoxical diplopia. Moreover, anomalous projection is frequently unstable, considerable variation in the angle of the anomaly (the position of peripheral fixation) being observed on repeated tests, though von Kries³³ stated that "in many instances the anomalous visual relation is a comparatively fixed one, strongly prevailing over the original, if the latter is still present at all."

Verhoeff³⁴ stated that he did not believe that there could be any retinal correspondence in anomalous projection and criticized the usual methods for demonstrating anomalous projection because of the highly artificial conditions set up for their use.

ORTHOPTIC PROCEDURE

Scattered references to orthoptic procedure can be found in the ophthalmologic literature for the last forty years without complete descriptions of the exercises used or the exact methods employed. Recently, more practical information concerning the technic of orthoptic training has been published, since the directions furnished with instruments are seldom adequate and courses in orthoptics, such as those given in several British clinics, are not generally available.

Worth's book on squint³⁵ in 1903 revived interest in the subject of orthoptics, which Javal³⁶ had treated in 1896. Wells'³⁷ emphasis on the use of the stereoscope in ophthalmology and the improvements in orthoptic instruments gave further impetus to the testing and training of binocular vision. Textbooks of ophthalmology mention the subject only

31 von Graefe, A. Ueber das Doppelsehen nach Schieloperationen und Incongruenz der Netzhäute, *Arch f Ophth* **1** 82, 1854

32 Tschermak, cited by Helmholtz,⁶ p 588

33 von Kries, cited by Helmholtz,⁶ p 588

34 Verhoeff, F. H. Anomalous Projection and Other Visual Phenomena Associated with Strabismus, *Arch Ophth* **19** 663 (May) 1938

35 Worth, C. Squint, Philadelphia, P. Blakiston's Son & Co., 1903

36 Javal, E. Manuel theorique et pratique du strabisme, Paris, G. Masson, 1896

37 Wells, D. W. The Stereoscope in Ophthalmology, ed 2. Boston Globe Optical Company, 1918

briefly, but in 1932 Cantonnet and Fillozat³⁸ published their book on strabismus and its reeducation, an English translation of which has appeared³⁹ British contributors have been foremost in the publication of articles and textbooks on orthoptic practice The works of Dobson,⁴⁰ Pugh⁴¹ and Lyle and Jackson²¹ may be referred to for descriptions of instruments and their use in orthoptics, Berens' "The Eye and Its Diseases"⁴² includes an article on orthoptics by Pugh, Travers²⁸ attempted to evaluate the place of orthoptics in the treatment of concomitant strabismus Articles in periodicals usually are confined to clinical studies without actual detail of orthoptic practice, and frequently incomplete records make evaluation of the studies difficult

Orthoptic tests can be carried out satisfactorily only if the proper surroundings contribute to quietness, isolation and unhurried examinations Individual supervision of testing and training is essential even when mechanical instruments are employed Half-hour periods several times weekly are usually recommended Cantonnet⁴³ suggested one office visit weekly with a full hour of home work daily Daily clinical work is usually impossible because of limited facilities, but fewer than three visits weekly, when no home work can be given, is hardly adequate to constitute "training"

No systematic studies of strabismic children for comparison with normal children in motor skills, personality traits, intelligence, scholastic achievement or social adjustment have been published Lippmann⁴⁴ found no evidence of more frequent stammering or lefthandedness in strabismic children, von Csapody-Mócsy⁴⁵ showed that children having convergent strabismus had symptoms of rickets more frequently than a general group of the same population Dor⁴⁶ suggested a possible relation between vitamin A deficiency and the development of strabismus

38 Cantonnet, A, and Fillozat, J Le strabisme, sa reeducation, Paris, Norbert Maloine, 1932

39 Cantonnet, A, and Fillozat, J Strabismus Its Reeducation, translated by M Coque, Chicago, M Wiseman & Company, 1934

40 Dobson, M Binocular Vision and the Modern Treatment of Squint, New York, Oxford University Press, 1933

41 Pugh, M A Squint Training, New York, Oxford University Press, 1936

42 Berens, C The Eye and Its Diseases, Philadelphia, W B Saunders Company, 1936, p 908

43 Cantonnet and Fillozat,³⁹ p 302

44 Lippmann, O Die Entartungszeichen und die Ueberwertigkeit einer Korperhalfte in ihrer Bedeutung fur das Schielen, Klin Monatsbl f Augenh **92** 370, 1934

45 von Csapody-Mocsy, M Ein Beitrag zur Pathologie des Begleitschielens, Klin Monatsbl f Augenh **92** 385, 1934

46 Dor, L Strabisme et avitaminose, Arch d'opht **50** 667, 1933

when a hereditary predisposition exists. Dull children are frequently poor subjects for orthoptic training, as their attention span is limited and their interest difficult to maintain. Cantonnet,⁴⁷ believing that the best results are to be expected from older children, accepted no patients under 7 years of age for orthoptic training. Accurate results in the testing of visual acuity by means of illiterate charts can usually be obtained when a child is between 3 and 4 years of age, and tests of binocular vision can be tried before a child is 2 years of age, though satisfactory tests require sufficient vocabulary to name simple objects. The age at which orthoptic training can be attempted depends on intellectual maturity and cooperation rather than on chronologic age.

Minimum orthoptic equipment should include a major amblyoscope with proper charts for measuring deviations and grading binocular vision as well as for duction training, supplementary fusion tests, and materials for visual training. The advantage of a major amblyoscope over the simple stereoscope lies in greater flexibility, easier control of macular fixation and greater convenience for rapid and accurate testing of binocular vision. In clinical work, additional equipment may be indicated, but the more complicated instruments employing moving targets have limited usefulness. Unilluminated stereoscopes and the simple amblyoscope are not readily employed to determine the type of projection used by the patient, since the examiner should have an unobstructed view of the subject's eyes during tests. The major amblyoscopes, like the simple stereoscope, contain convex spheres to give optical infinity at the distance chosen for the charts employed, prismatic effects are obtained by changing the actual position of the reflected images. Accommodation may be stimulated by the addition of concave spheres for near point tests or, in some instruments, by moving the charts closer to the eyes. Accurately prepared charts are necessary for test purposes but appropriate slides for duction training can be cheaply constructed by mounting tissue pictures in standard slide glass covers or tracing crayon pictures on etched glass. The angular size of the images can be determined by the use of the proper formulas, standard charts may be graded in angular size.

Complete records for orthoptic training are essential and should include general examination and preliminary treatment as well as accurate information concerning clinical visits and home work. A convenient record form for clinical use which can be printed on one side of standard 8½ by 11 inch (21 by 28 cm) sheets, or on 5 by 8 inch (12 by 20 cm) record cards if both sides are used, is shown in the accompanying table.

47 Cantonnet and Fillionat,³⁹ p. 228

Orthoptic Record

Number Name Address	Clinic Age Birth	Development Diseases Operations	Handedness I Q Cooperation
Strabismus, type Heredity Age of onset Mode of onset Cause Symptoms Fixing eye	Deviation Convergent Divergent Vertical Occasional Periodic Constant	Previous treatment Glasses Occlusion Atropine Orthoptics Operation	
Examination Fundus Media Fixation Motility Fields Amblyopia	Refraction and Prescription Cyclo Date plegic Static Rx		Binocular Vision Projection Suppression Fusion Distance Near Amplitude Distance Near Worth dots Distance Near Stereopsis
Head position Angle gamma P D * PcB †	1 2 3 4		Dates
Operations			
Recommendations			

Date	Time	Acuity		Screen		Deviation Synoptophore		Amplitude		Notes	Home Work	Return
		Ce †	Sc §	I ar	Near	Obj	Subj	Ad	Ab			

* Interpupillary distance
† Near point of convergence
‡ With correction
§ Without correction

In preliminary treatment of heterophoria and heterotropia, the significance of accurate correction of refractive errors cannot be overemphasized. Superficial reports of the "success" of orthoptic treatment frequently fail to indicate the importance of proper optical correction in aiding the coordinate action of the eyes, and it is probable that in many cases the correct prescription is of far greater effective value than orthoptic exercise. The value of any method of visual reeducation or coordinate exercise applied before optical correction of refractive errors has been used constantly for a period of several months is certainly open to question because spontaneous improvement in both acuity and coordination may occur without recourse to special training. No careful study of the length of the period during which correcting lenses should be worn previous to orthoptic treatment has been made. Guibor⁴⁸ and Bressler⁴⁹ suggested the wearing of the proper optical correction for

48 Guibor, G P The Possibilities of Orthoptic Training, Am J Ophth 17 835, 1934

49 Bressler, J L Orthoptic Treatment of Strabismus, Illinois M J 68 273, 1935

six months before orthoptic training Fuchs (Duane)⁵⁰ pointed out that the wearing of correcting lenses does not give an immediate effect "It usually takes some months for the full effect of the glasses to be secured" Maddox⁵¹ stated "The correction of the refraction does not always cure an accommodative squint at once, but lessens it by degrees" Parinaud⁵² recommended optical treatment for at least six months previous to other treatment Feldman⁵³ accepted patients for orthoptic training after glasses have been worn six weeks with no improvement Cords⁵⁴ in a statistical summary indicated that over 80 per cent of persons with convergent concomitant strabismus have hypermetropia, and since this defect carries with it the necessity for excessive accommodation, lenses which relieve this should certainly be worn before other treatment Guibor⁵⁵ found that 23.6 per cent of 185 persons with convergent strabismus responded to correction of the refractive error alone Of a group of 377 persons with convergent strabismus studied at the Manhattan Eye, Ear and Throat Hospital,⁵⁶ 26.25 per cent responded to correction of refractive errors without other treatment

Most authorities recommend for children the use of a 1 per cent solution of atropine two or three times daily for at least three days before retinoscopic examination For persons with strabismus, Thorington⁵⁷ recommended the daily use of atropine for two or three weeks, Howe⁵⁸ stated that in the nonoperative treatment of strabismus accommodation should be relaxed for months Parinaud⁵⁹ recommended the use of atropine for at least a week previous to tests, Javal⁶⁰ used atropine twice daily for one week, with atropine and cocaine used three times, three hours before examination Cords⁶¹ recommended the use

50 Fuchs, E. Textbook of Ophthalmology, ed 8, translated by A. Duane, Philadelphia, J. B. Lippincott Company, 1924, p. 335

51 Maddox,³⁰ p. 119

52 Parinaud, H. Traitement du strabisme, Bull. et mem. Soc. franç. d'opht., 1893, p. 50

53 Feldman, J. B. Orthoptic Treatment of Concomitant Squint, Arch. Ophth. **13** 419 (March) 1935

54 Cords, R. Die Physiologie der Augenbewegungen, in Schieck, F., and Bruckner, A. Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 3, p. 536

55 Guibor, G. P. Strabismus in Children Corrected by Refraction Alone, Am. J. Ophth. **18** 944, 1935

56 Keil, F. C. Summary of Orthoptic Work at the Manhattan Eye, Ear and Throat Hospital, Arch. Ophth. **17** 751 (April) 1937

57 Thorington, J. Refraction of the Human Eye, ed 2, Philadelphia, P. Blakiston's Son & Co., 1930, pp. 278 and 289

58 Howe, L. The Muscles of the Eye, New York, G. P. Putnam's Sons, 1907, vol. 2, p. 212

59 Parinaud,⁵² p. 9

60 Javal,³⁰ p. 147

61 Cords,⁵⁴ p. 529

of 2 or 3 drops of a 1 per cent solution of atropine for from three to eight days before refraction, pointing out that partial paralysis of the ciliary muscle leads to increased effort at accommodation and greater convergence when insufficient atropine is used. Both Howe⁶² and Thourington⁵⁷ emphasized that homatropine is unreliable and should not be used as a cycloplegic for children or young adults having muscle imbalance.

Apparent increase in static hypermetropia shown on successive tests under a cycloplegic is probably to be accounted for by imperfect paralysis of the ciliary muscle on preliminary retinoscopic examinations. Both Brown⁶³ and Vorisek⁶⁴ reported a slight average increase in hypermetropia in children on successive tests. There is generally assumed to be a tendency toward decrease of the normal hypermetropia of children as the eyes develop. When there has been prolonged and careful use of a cycloplegic, it is not usual to find increased hypermetropia on successive tests, though careless administration of drops by parents frequently results in inaccurate measurements, especially for young children.

Changes in refractive errors are to be expected after operations on the muscles. Marshall⁶⁵ made a systematic study of such changes and showed that they may occur in eyes not subjected to operation as well as in those operated on.

The question of the proper prescription for the patient with muscle imbalance is one in which clinical experience and theoretic considerations are not always in agreement. In general, convex lenses reduce convergent deviations in which there is an accommodative factor, while concave lenses which increase accommodation for finite distances tend to increase convergent, and reduce divergent, deviations. On the other hand, as reported by Parinaud⁶⁶ esotropia in myopic patients sometimes responds to optical correction, probably through a recession of the far point of accommodation and accompanying reduction in convergence, and occasionally divergent deviations in hypermetropic persons are reduced by the use of correcting lenses for the refractive error.

The appearance of the eyes in full cycloplegia is of considerable prognostic value, especially in cases of convergent strabismus. If

62 Howe,⁵⁸ vol 1, p 169

63 Brown, E. V. L. Apparent Increase in Hyperopia up to the Age of Nine Years, *Am J Ophth* **19** 1106, 1936

64 Vorisek, E. A. Changes of the Refraction of Children with Convergent Concomitant Strabismus, *Am J Ophth* **18** 820, 1935

65 Marshall, D. Changes in Refraction Following Operation for Strabismus, *Arch Ophth* **15** 1020 (June) 1936

66 Parinaud, H. Le strabisme et son traitement, Paris, Octave Doin, 1899, p 125

paralysis of accommodation results in orthophoria or divergence, optical correction can be expected to be effective, provided the refractive error is hypermetropic and convergent deviation is evident when the ciliary muscles are active. Postcycloplegic acceptance by children is completely unreliable as an indication of the proper correction because of active accommodation. Temporary use of atropine is sometimes necessary to reduce spasm of the ciliary muscle when full correction gives blurred vision for distance.

Sattler⁶⁷ demonstrated the effectiveness of prolonged occlusion of the fixing eye in improving the acuity of the deviating eye in cases of monolateral strabismus. In 3 of 89 cases amblyopia of the covered eye resulted from this treatment, constituting a strong argument for the existence of amblyopia ex anopsia. Abraham⁶⁸ distinguished congenital amblyopia from acquired amblyopia which may result from refractive errors (usually astigmatism of over 2 diopters) or active suppression in cases of strabismus. Smukler⁶⁹ suggested that retinal hemorrhages at birth (found in 10 or 15 per cent of the newborn) may cause amblyopia and, subsequently, strabismus. Hesse⁷⁰ demonstrated that in a large percentage of cases there are changes in the amblyopic eye which account for its diminished vision. Astigmatism, anisometropia and high hypermetropia are frequently associated with amblyopia. Peter⁷¹ expressed the belief that there may be visible pathologic changes in congenital amblyopia. As pointed out by Parinaud,⁷² it is erroneous to assume that improved acuity increases the tendency toward fusion, but because monocular suppression is readily maintained in the presence of unequal acuity, equalization of acuity through accurate optical correction and visual training is usually advocated as preliminary to orthoptic exercise. Complete and constant occlusion of the fixing eye, with frequent and careful tests of acuity (using illiterate charts, even for adults) for both eyes, is accepted as the most effective method of treating amblyopia ex anopsia. For children, such treatment should be delayed until the proper optical correction has been worn for two or three months, since spontaneous recovery of normal acuity may occur. A 4 year old child who showed occasional convergent strabismus was found to have acuity of 6/10 in the right eye and 6/20 in the left eye two

67 Sattler, C. H., cited by Cords,⁵⁴ p. 527.

68 Abraham, S. V. A Classification of Amblyopia, *Arch. Ophth.* **12**:402 (Sept.) 1934.

69 Smukler, M. E. Amblyopia and Squint from Head Injuries at Birth, *Pennsylvania M. J.* **37**:25, 1933.

70 Hesse, R. Ueber die Amblyopie schielender Augen, *Arch. f. Ophth.* **130**:375, 1933.

71 Peter, L. C. Technic of Orthoptic Training in Squint, *Arch. Ophth.* **14**:975 (Dec.) 1935.

72 Parinaud,⁵² p. 20.

weeks after the prescription of full hypermetropic correction (+ 6.00 sphere for the right eye, + 7.50 sphere for the left eye) There was gradual improvement in acuity, without occlusion, two months later vision was 6/6 in the right eye and 6/10 in the left eye⁷³ Pearson and Mayou⁷⁴ found that it is difficult to cure amblyopia which has occurred before the second year when treatment is delayed until after the fifth year, Peter⁷⁵ expressed the belief that amblyopia does not develop after the seventh year and that the best results in visual training can be expected before the seventh year

The age, duration of the amblyopia, probable cause for the defect, muscle balance and future treatment should be considered when visual training is recommended by forced use of an amblyopic eye Young patients respond most successfully (under 6 or 7 years of age), high refractive errors, corrected late in life, result in amblyopia which does not respond well to visual training, amblyopia in conjunction with hyperphoria of 5 diopters or over usually responds poorly to visual training, improvement in acuity is often temporary when binocular vision is precluded by high deviations from normal muscle balance

Complete occlusion of the fixing eye, which forces central fixation of the defective eye, results in the constant use of central vision in that eye There is no objective evidence that artificial stimulation by means of mechanical devices is more effective in improving visual acuity than constant use of the eye under ordinary conditions, when continuous stimulation and frequent changes in accommodation are present If central fixation of the deviating eye is not evident when the fixing eye is covered, occlusion is ineffective in improving acuity The use of atropine in the fixing eye is effective in transferring fixation to the other eye only if the acuity of the poorer eye is not below 20/50⁷⁵ In cases of severe amblyopia, fixation is maintained by the better eye in spite of the paralyzed accommodation, so that this method can rarely be recommended in the treatment of amblyopia

Orthoptic training should not be undertaken before the vision of the amblyopic eye has been brought to 20/70 or better When acuity is not over 20/100, complete macular suppression is easily established, and though fusion of large images is possible, stable binocular vision cannot be stimulated

Alternate occlusion, suggested by Javal⁷⁶ as a method of preventing the establishment of anomalous projection in cases of strabismus, has

73 Case record 153496 of the Manhattan Eye, Ear and Throat Hospital

74 Pearson, S, and Mayou, S Central London Ophthalmic Hospital Report of Squint Department, September, 1932-1933, Brit J Ophth 18 267, 1934

75 Fuchs,⁵⁰ p 336

76 Javal,³⁶ p 81

been recommended by some clinicians. Theoretically, if no simultaneous use of the eyes is permitted except during artificial bifoveal stimulation under controlled conditions, anomalous projection might be prevented or discouraged. No extensive clinical studies of this method have been published, but Travers,⁷⁷ who used alternate occlusion in 5 cases over a period averaging four and a half months, found it completely ineffective in the treatment of anomalous projection.

Occlusion in cases of strabismus does not change the coordinate movements of the eyes, the covered eye assumes the position ordinarily characteristic of the deviating eye. Prolonged occlusion in cases of heterotropia, which brings about equalization of acuity, may produce diplopia, which is considered an essential to the reestablishment of binocular vision. Actually, such diplopia can usually be observed under test conditions long before acuity is equal in the two eyes, and unless the position of the eyes is such that bifoveal fixation under ordinary conditions (i.e., without prism aids) is possible, observable diplopia is no indication that fusion may be stimulated. The difficulty in orientation caused by constant diplopia brings about prompt return of suppression for most persons. Observant subjects having anomalous projection can sometimes recognize both foveal and peripheral images (monocular diplopia) under test conditions; the images are unequal in clearness.⁷⁸

The following routine is suggested for rapid clinical testing of binocular vision with a major amblyoscope. The eyes should be allowed to recover fully from the effects of cycloplegia before tests are carried out, in practice, though the patient's correction may be placed on the instrument with the use of the proper correcting formulas, preliminary tests should be made with the subject's glasses on. This is particularly true for young patients with hypermetropia who may show spasmodic convergence associated with the stimulation of accommodation resulting from removal of the optical correction. The position of the subject should be relaxed and comfortable, with the instrument adjusted for vision in the primary field, since adduction and convergent deviations are greater in the lower field and abduction and divergent deviations are exaggerated in the upper field. It is unnecessary to hold the head in position with the hand except when marked head tilt or severe amblyopia is present. An intelligent child learns to report not what he actually sees but what he believes he should see, so that extreme care is necessary to avoid suggestion which may falsify tests. Control of illumination makes accurate checking of the responses possible.

1. The instrument is adjusted to the proper height and interpupillary distance.

⁷⁷ Travers,²⁸ p. 71.

⁷⁸ Travers,²⁸ p. 69.

2 Slides having small, centrally placed, dissimilar images are inserted, and the following tests are carried out

(a) Monocular fixation is tested by presenting the images separately to each eye, noting the angle gamma (or alpha) as positive or negative (In normal and hypermetropic eyes the angle between the optic axis and the visual axis at the nodal point of the eye is usually positive, the light reflex lying to the nasal side of the apex of the cornea and giving rise to apparent divergence, in myopic eyes the angle gamma may be zero or negative, in which case the light reflex lies temporal to the corneal apex and gives apparent convergence With appropriate slides, the size of the angle may be estimated in degrees)

(b) On testing monocular motility any limitation of motion in the lateral fields is noted

(c) The objective deviation is measured by alternate presentation of the images, prismatic correction being added by adjusting the images to neutralize lateral and vertical movements (In cases of concomitant strabismus the primary deviation, measured with the fixing eye in the primary position, is equal to the secondary deviation, measured with the deviating eye in the primary position) If foveal fixation is not present, objective measurements must be made by adjusting the corneal reflexes

(d) The subjective deviation is measured by noting the prismatic correction necessary for superposition of small, unlike images (When there is normal projection, subjective and objective measurements are equivalent, extinguishing either image after subjective superposition does not result in movement of the opposite eye When there is anomalous projection, the subjective deviation is less than the objective deviation Paradoxical diplopia is reported with the prism which corrects the deviation objectively, superposition is attempted by decreasing the prismatic correction, which makes the images appear to approach Actual superposition is frequently impossible, one image disappearing only to reappear on the opposite side of the other image The presence of anomalous projection is substantiated, first, if one image is reported to be less clear than when it is seen monocularly, second, if the corneal reflex of one eye is seen to lie in a peripheral position not compatible with central fixation, and third, if repeated tests indicate unstable binocular vision, with variability in the prismatic correction required for superposition)

3 Slides having similar images, with cues to indicate binocular fixation and fusion, are inserted, and the following tests are carried out

(a) Abduction is tested by adding prisms base in or reducing prisms base out until diplopia is reported or until one image is suppressed (Abduction measured in this way, on a major amblyoscope, is usually higher than that measured by loose prism methods)

(b) Adduction is tested by adding prisms base out or reducing prisms base in until diplopia is reported or until one image is suppressed (When subjective reports are unreliable, amplitude of fusion can be estimated by noting the points at which binocular fixation is replaced by alternation or monocular fixation) Convergence micropsia, the apparent decrease in the size of the fused images as prisms base out are added, is frequently reported spontaneously and is a supplementary indication of fusion, since when binocular fixation is lost the images appear larger ⁷⁹

(c) Supraduction or subduction is tested by adding prisms base up or base down, cycloduction measurements probably have no validity, as it has been shown by Beasley and Peckham that torsional movements are not made under such conditions ⁸⁰

4 Slides for testing stereopsis (disparate images) are inserted next

(a) Gross stereopsis is tested by ascertaining the depth relation apparent on slides having high binocular disparity, similar parts of the images being widely separated when the slides are held together Stereopsis is defective if disparate parts of the images appear displaced to one side (i.e., if a ball which should be centered in a ring is displaced to one side) Such displacement is usually observed when the acuity of one eye is much better than that of the other eye

(b) Fine stereopsis is tested by using graded slides with progressive reduction in the disparity of the paired images

The same tests may be repeated for the near point by adding appropriate concave lenses to stimulate accommodation or by moving the images closer to the eyes Since the optical effect of concave lenses decreases when they are moved from the eyes, the lenses employed must be selected accordingly With accommodation for the reading distance (33 cm), the person with an interpupillary distance of 60 mm must converge about 18 prism diopters Prism tolerance for near vision should be measured from this point and recorded as prism tolerance for base in or base out with accommodation

An attempt to define "normal" ductions is complicated by the high individual variations among those having no symptoms of muscle imbalance and by the fact that measurements of duction power vary with age, fatigue, practice and psychologic factors Moreover, the method employed influences the absolute measurements The ratio of adduction to abduction has been regarded as of greater clinical significance than the absolute values of positive or negative convergence It is usually stated that adduction with distance fixation should be from two to four

⁷⁹ Travers,²⁸ p. 66

⁸⁰ Beasley, W. C., and Peckham, R. H. "An Objective Study of "Cyclo-torsion," *Psychol. Bull.* **33** 741, 1936

times abduction to assure comfortable convergence reserve ⁸¹ Howe, pointing out that interrupted increases in prismatic addition give lower measurements than continuous increases, recommended what he called the minimum method of measuring ductions ⁸² by beginning with a prism too great to be overcome. This method was officially adopted by the American Medical Association in 1907, but is seldom employed ⁸³. Maximum adduction cannot be accurately determined because it is easily increased by practice. The published statistical studies of average duction power show great variability. In 1927 Berens, Losey and Hardy ²⁴ published average values for adduction and abduction for distance and near fixation for 218 young men having normal acuity and no ocular symptoms and for 104 patients selected as having normal muscle balance. All were measured by loose prisms, presented in racks, from low to high

Distance Fixation		Near Fixation	
Adduction	9 to 45D , average, 19D	Adduction	15 to 70D , average, 41D
Abduction	5 to 15D , average, 7D	Abduction	14 to 38D , average, 19D

For rapid indication of fusion at distance or at the near point ⁸⁴ Worth's four dot test is a convenient supplementary method ⁸⁵. Four illuminated circles, a white and a red circle and two green circles, are observed through red and green complementary filters. A subjective report of four lights indicates fusion, two or three, suppression, and five, diplopia. (If this test is prepared by the examiner, care must be used to secure strictly complementary filters, without which the test is invalid.)

The clinical training of binocular coordination implies the stimulation of normal projection, the training of adequate fusion amplitude and the encouragement of stereopsis.

It has been assumed that anomalous projection, in which binocular stimulation gives diplopia, is an acquired perceptual system and that normal projection can be reestablished by the use of appropriate orthoptic methods. This technic has been described in detail by Pugh, ⁸⁶ Lyle and Jackson ⁸⁷ and Travers ⁸⁸. To review briefly, two methods may be described.

1 Simultaneous stimulation of the two macular regions at the objective angle of deviation, with repeated lateral movements of the image

⁸¹ Taylor, E. A. *Controlled Reading*, Chicago, Chicago University Press, 1937, p. 246 (footnote)

⁸² Howe, ⁵⁸ vol 1, p. 299

⁸³ Wells, ³⁷ p. 25

⁸⁴ Worth Dot test (modified for near fixation), Clairmont Nichols Company, New York

⁸⁵ Lyle and Jackson, ²¹ p. 37

⁸⁶ Pugh, ⁴¹ p. 61

⁸⁷ Lyle and Jackson, ²¹ p. 58

⁸⁸ Travers, ²⁸ p. 68

2 Alternate presentation of images at the objective angle of deviation by manual or mechanical control of illumination

The infrequent success of orthoptic treatment in the correction of anomalous projection may be accounted for by the fact that even prolonged binocular stimulation under highly artificial conditions cannot bring about a stable projection system unless the eyes are in a position which makes constant bifoveal fixation possible. Travers⁸⁹ found that successful treatment of anomalous projection by orthoptic methods is so rare that it cannot be legitimately advocated in any case, since no treatment which did not include operation had any beneficial effect on patients with this type of projection. Lyle and Jackson⁹⁰ are more optimistic with regard to the use of orthoptic training in such cases, stating that in a favorable case projection becomes "true" in twelve training periods. Nevertheless, in their records of 24 cases of monolateral convergent strabismus in which the treatment did not include surgical measures, an average of eighteen periods was required to obtain "normal correspondence," and only 50 per cent of the patients showed single binocular vision at the end of training (after six to ninety periods). Pugh⁹¹ did not state how long training should be continued in cases of anomalous projection, but she pointed out that the first method, mentioned previously, "takes a long time to obtain a favorable result, too long for the perseverance of the average patient." In regard to the second method, with alternate presentation of images at the rate of from one to twenty per second, Pugh stated that the patient "soon accepts the images binocularly in their correct relationship and sees one composite image all the time." Nevertheless, in evaluating the results of treatment by orthoptic training, Pugh excluded all cases of anomalous projection (50 per cent of the cases)⁹²

Authorities are not in agreement concerning the value of orthoptic training in conjunction with surgical treatment. Unfortunately, it is not possible to isolate the specific contribution of orthoptic training toward the return of binocular vision in such cases, since binocular vision may be reestablished spontaneously after operation. Bressler⁹³ reported more favorable results in a group of 36 cases in which both preoperative and postoperative orthoptic training was used than in 150 cases in which operation alone was employed. His results, given in percentages, may be misleading because of the fact that the groups compared are not

89 Travers,²⁸ p. 77

90 Lyle and Jackson,²¹ p. 97

91 Pugh,⁴¹ pp. 62 and 85

92 Pugh,⁴¹ p. 85

93 Bressler, J. L. Treatment of Strabismus. Influence of Orthoptic Training on the Results of Operation, *Arch. Ophthalm.* 16:433 (Sept.) 1936

equivalent Berens, Payne and Kern,⁹⁴ in a study of orthoptic training and surgical treatment in cases of hyperphoria and hypertropia associated with lateral deviations, showed that prolonged periods of pre-operative and postoperative training, possible for patients in private practice, apparently contribute to more satisfactory binocular vision. They are of the opinion that anomalous projection, uncorrected pre-operatively, may cause the postoperative recurrence of the same deviation. They reported, however, that placing the eyes in approximate parallelism by means of surgical methods resulted in an increase of 40 per cent in the number of patients who could fuse.⁹⁴ Hicks and Hosford⁹⁵ reported spontaneous postoperative fusion in 18 of 24 cases in which no orthoptic training was used. Travers⁹⁶ stated "The idea that an abnormal correspondence will forcibly deviate the eyes after operation into their old position is also probably without justification."

If normal projection is present and fusion can be elicited on binocular instruments with or without prismatic aids, increased amplitude of fusion through training of adduction and abduction may be brought about by practice. Paired similar images with binocular cues should be used, and the patient must be taught to observe diplopia or suppression when binocular fixation is lost. It has been assumed, without adequate theoretic basis or controlled research, that practice in adduction will lessen exophoric deviations, while practice in abduction will lessen esophoric deviations. It is well established that voluntary control of adduction can be assisted by exercises with base-out prisms, and symptomatic relief in convergence insufficiency may be reported when adduction reserve is high, as indicated by Lyle and Jackson,⁹⁷ Berens²⁴ and Stutterheim.⁹⁸ Berens suggested that tolerance to a base-out prism of 75 prism diopters for both distance and the near point should be attained by home exercise with loose prisms. Such exercise often causes esophoria, which, according to Berens,²⁴ does not produce symptoms. In divergence excess (in which the deviation on dissociation tests is greater for distance than for the near point), adduction, both at the far and at the near point, can sometimes be stimulated, and voluntary control of binocular fixation can occasionally be established, but the deviation will remain unchanged with dissociation tests or when the

94 Berens, C., Payne, B. F., and Kern, D. Orthoptic Training and Surgery in Hyperphoria and Hypertropia Combined with Lateral Deviations, *Am J Ophth* **18** 522, 1935

95 Hicks, A. M., and Hosford, G. N. Orthoptic Treatment of Squint, *Arch Ophth* **13** 1026 (June) 1935

96 Travers,²⁸ p. 86

97 Lyle and Jackson,²¹ p. 153

98 Stutterheim, N. A. Eyestrain and Convergence, London, H. K. Lewis & Co., Inc., 1937

eyes are at rest. In such cases it may be necessary to make use of accommodative convergence by beginning adduction at the near point, in high divergence excess it is not always possible to establish any binocular vision for distance. Since the normal tendency toward divergent position of the eyes during growth prevents the spontaneous cure of divergence excess, early operation is sometimes to be recommended to prevent a cosmetic defect and to avoid the additional convergence insufficiency which may occur. Abduction, or tolerance to base-in prisms, is involuntary and must be stimulated reflexly through the relaxation of convergence. Javal⁹⁹ expressed the belief that strong adduction under voluntary control must be learned before abduction can be accomplished through the relaxation of convergence. Abduction is usually undertaken with accommodation relaxed (i. e., with distance fixation), but Pugh¹⁰⁰ reported more favorable results when accommodation, without convergence, is stimulated by placing the images close to the eyes, without additional base-out prisms (possible on the orthoptoscope or the stereoscope). Tait,¹⁰¹ who used controlled exercise with base-in prisms for 4 normal subjects, demonstrated that such exercise served to increase adduction power and to decrease exophoria. It is evident that since fatigue and other factors may be of considerable significance in duccion training, further research rather than haphazard clinical work should be undertaken.

Careful tests of muscle balance may disclose esophoria for distance and exophoria for the near point or, particularly in cases of post-operative convergent concomitant strabismus, exophoria for distance and esophoria for the near point. It is probable that in such cases duccion training should include practice with base-in and base-out prisms. Temporary esophoria for distance is frequent after extensive close work, even when there is marked exophoria at the near point. Persons who show exophoria at the near point, combined with esophoria for distance and low adduction, may overcompensate for a primary exophoric deviation, and both Wells¹⁰² and Lyle and Jackson¹⁰³ advocate exercise with base-out prisms for such persons.

Within narrow limits, it may be possible to train a hypermetropic person to accommodate without excessive convergence, but such exercise does not usually result in any permanent benefit, since persons predisposed to convergent deviations cannot accommodate sufficiently

99 Javal,³⁶ p. 111

100 Pugh,⁴¹ p. 54

101 Tait, W. J. Further Studies of the Fusional Convergence Amplitude, *Am. J. Optometry* **14** 4, 1937

102 Wells,³⁷ p. 70

103 Lyle and Jackson²¹ p. 152

for clear vision without stimulation of convergence. It is obvious that young hypermetropic persons whose eyes are orthophoric with convergence, with stable fusion at both the near point and at distance, should not be subjected to prolonged orthoptic training in an effort to establish the same coordination without correction of the refractive error. Parents are sometimes under the impression that exercise will make possible the discarding of children's glasses, but since this is rarely indicated, they should be correctly informed.

There is little basis for the assumption that stereopsis can be "taught" by orthoptic methods. Moreover, it is erroneous to assume that stereoscopic perception has a direct influence on muscle balance. Patients, for instance, who show periodic exotropia may demonstrate perfect stereoscopic perception under test conditions, and yet they do not employ binocular vision at all times. In fact, it is doubtful if even normal persons make use of stereoscopic vision constantly. Unless the conditions requisite to stereopsis are fulfilled—approximately equal acuity in the two eyes, bifoveal fixation, etc.—stereoscopic depth cannot be observed. Stereoscopic charts may be used in training ductions, and the subject may be encouraged to observe the depth relation mediated by binocular disparity, but "mental effort" cannot establish stereopsis.

Postoperative duction training does not differ essentially from preoperative training and can be undertaken as soon as healing has occurred. Passive tests of binocular vision and ocular position may be carried out shortly after operation, care being taken not to disturb the stitches by extreme movements. Changes in optical correction are frequently indicated postoperatively, and the influence of the correction on ocular position may be measured on a major amblyoscope. Trial lenses should be worn fifteen minutes before such tests.

Home exercise as a supplement to clinical training is frequently advocated, but most authorities do not report successful results, since even with competent instruction the exercises are seldom carried out regularly. It should be emphasized that home exercises based on prism technic or the use of a stereoscope can be attempted only when fusion with normal projection can be elicited on clinical tests. Accurate preliminary diagnosis of binocular vision is therefore necessary. Explicit directions, which must be modified to suit the individual patient, with an explanation of the purpose of the exercises, should be given. The following outline suggests instructions for simple home exercises.

1. Simple convergence exercises to stimulate convergence with accommodation, indicated in convergence insufficiency or in divergence excess to prevent secondary convergence insufficiency. (These exercises are not indicated if convergence cannot be elicited on repeated

tials) Convergence exercises are used to help sustain normal coordination when one eye has a tendency to turn out. The instructions follow

Use a black vertical line, a vertical row of letters or, for children, small toys or tiny pictures. Hold the object at arm's length, slightly below the level of the eyes, watch it closely with both eyes as it is moved slowly toward the nose. When the object appears double, or when one eye moves away from the nose, repeat the test, starting with the object at arm's length. Be sure that both eyes are focused on the object. (They should be seen to move toward the nose.) Move the object slowly. Repeat———times———periods daily.

(For children, ten repetitions three times daily may be recommended, for adults, three five minute periods. Presbyopic patients seldom attain any symptomatic relief from such exercise.)

2 Prism convergence exercises to stimulate convergence, with or without accommodation, indicated in convergence insufficiency, divergence excess or esophoria if adduction is low. The instructions follow

Obtain prisms no —— and a red glass filter from an optician for temporary use. Arrange a light (15 watt frosted bulb, flash light or candle) which can be observed from a distance of 20 feet (609 cm). Place a prism base out (the thick side toward the temple) and a red glass before either eye. If two lights are seen, make an effort to bring the lights together by turning the eyes toward the nose (possible by looking momentarily toward a near object with the prism in position). After fusion is obtained, so that a single light is seen through the prism, count five slowly, remove the prism and repeat. Practice———minutes ——times daily. Increase to no ——prism.

(Three five minute periods daily may be recommended. Exercise should begin with the highest prism easily overcome on clinical tests and increased weekly until 40 diopters or more can be overcome. If the use of a red glass prevents fusion, tests may be carried out without the filter. Monocular fixation is evident when a cover placed quickly before the fixing eye causes inward movement of the opposite eye.)

3 Stereoscopic duction exercises to increase fusion amplitudes and to dissociate accommodation and convergence, indicated in exophoria or esophoria when fusion can be elicited with normal projection and without high prism correction. The instructions for these exercises follow

Obtain from an optician a stand stereoscope and set of divided stereoscopic charts

Adduction A (a) Adjust the stereoscope for distance, placing the lenses —— inches from the chart holder

(b) Find the position in which fusion is attained by superposition of unlike images (pictures in which the two images have no similar parts)

(c) Substitute similar paired charts, placing them in the position found in 2, be sure the images are fused by looking for all the objects in the picture

(d) Move one or both charts toward the center of the chart holder until two pictures are seen or until some part of the fused picture disappears. Keep a record to show how close together the charts can be moved without losing fusion. Practice from ten to fifteen minutes daily.

Adduction B (a) Adjust the stereoscope for near fixation by placing the lenses——inches from the chart holder.

(b) Fuse the divided charts, leaving them as close together as possible, then move the stereoscope from the cards until fusion is lost. Practice from five to ten minutes daily.

Abduction A Instructions (a, b and c are the same as given for Adduction A)

(d) Move one or both charts away from the center of the chart holder until two pictures are seen or until some object in the fused picture disappears. Practice from ten to fifteen minutes daily.

Abduction B (a) Adjust the stereoscope for distance by placing the lenses——inches from the chart holder.

(b) Fuse the divided charts, leaving them as far apart as possible, then move the stereoscope closer to the charts until fusion is lost. Practice from five to ten minutes daily.

The foregoing exercises are simplified if a stereoscope having a calibrated shaft is used. Otherwise, the positions for distance and for near fixation must be calculated by finding the first focal plane ("optical infinity"), which is determined by the strength of the decentered convex spheres in a lenticular stereoscope.

Intelligent cooperation on the part of those responsible for the home care of strabismic children is essential and can be secured by giving information to the parents concerning the purpose and sequence of therapeutic measures. Parents are chiefly concerned with the child's appearance, and it is necessary to emphasize that restoration of binocular vision may contribute to the maintenance of normal ocular position. In clinical work at the Manhattan Eye, Ear and Throat Hospital, a booklet, "Helping Children See Straight,"¹⁰⁴ prepared to answer parents' questions, has been found useful.

SUMMARY

Binocular vision, of relatively recent development in the evolutionary scale, is based on complex neurologic relations which are not completely understood. Although the essentials for binocular vision can be isolated, the exact basis for the production and maintenance of fusion remains in the realm of theory. Clinical analysis of the anomalies of binocular vision which are associated with imbalance of the ocular muscles frequently reveals significant functional changes, including defects of the visual field (suppression scotomas), amblyopia ex anopsia and anoma-

104 Shaad, D. J. *Helping Children See Straight For Parents*, New York, The Manhattan Eye, Ear and Throat Hospital, 1937.

lous projection. Orthoptic procedure implies the application of measures designed to correct these defects and to promote normal coordination of the ocular muscles through the training of fusional ductions. If such treatment is to be established as a routine therapeutic measure in the treatment of strabismus, the value of its contribution depends on the proper testing of binocular vision, adequate records and careful control of other factors, such as correction of refractive errors, which form a part of the general treatment. Since clinical studies are frequently incomplete and confined to selected cases, the role of orthoptic methods in the restoration of normal binocular vision cannot be accurately outlined without further research.

Correspondence

SIMPLIFICATION OF THE O'CONNOR CINCH OPERATION

To the Editor —I read with deep interest in the August issue of the ARCHIVES, page 315, Dr O'Connor's comment on my article entitled "Simplification of the O'Connor Cinch Operation," published in the June issue

Dr O'Connor stated that I have "fallen into the common error" that he (Dr O'Connor) always divides the tendon into four strips. I know that he does not always divide the tendon into four strips. However, I invariably do for the type of case mentioned in my article, and for the following reasons

1 To remove the danger of tearing a strip of tendon. When the tendon is divided into more than four strips—Dr O'Connor even suggests up to twelve strips in some cases—one of these delicate thin strips (often 1 mm or less wide) is occasionally torn when the strip is pulled aside for looping, no matter how gently the narrow strip is handled. This is most likely to happen in operations on children, the muscle being often attenuated or the field of operation limited. Other surgeons have called attention to this danger (Berens, C. The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 1150).

2 As Dr O'Connor has himself stated, "Width of tendon strip. The more tissue in each, the greater the shortening." (The Cinch Shortening Loop in Surgery of the Extra-Ocular Muscles, *West J Surg* 39:670 [Sept] 1931).

If Dr O'Connor had read my article carefully, he would have noted that I made not the slightest reference to the use of the separators or to the number of strips into which the tendon is to be divided in the correction of vertical deviations.

I stated that it was my practice in all cases of divergent squint and properly selected cases of convergent squint of moderate degree to divide the tendon and muscle into three or four strips, using the separators. The advantages of this procedure are as follows:

- 1 A good view of the operative field is presented.
- 2 Less manipulation and less risk of tearing the strips are involved, as the strips do not have to be pulled aside to loop them.
- 3 The operative procedure is greatly facilitated and shortened.

The results have been uniformly gratifying in the type of case in which I suggested that my procedure be used.

M. E. SMUKLER, M.D., Philadelphia

A NEW MODEL GONIOSCOPE

To the Editor —In the report on a new model gonioscope in the June 1938 issue of the ARCHIVES (page 983) no mention was made of the portable gonioscope previously used and exhibited by Dr. Otto Barkan. No reference was made to his article in the December 1937

issue of the *American Journal of Ophthalmology* (page 1237) for the reason that the article by Mr Goebel and me was written before that date. The fact that he had exhibited his instrument before the American Academy of Ophthalmology and Otolaryngology and elsewhere and his paper in the 1936 *Transactions of the Academy of Ophthalmology and Otolaryngology* were inadvertently overlooked. The stand described in our paper is similar to Dr Barkan's, and he properly deserves full credit for it. However, I believe that the main features of our instrument, namely, the lighting arrangement, inverted suspension to allow free examination of the angle in its entire circumference and centered pivot to keep the instrument in focus as various segments of the angle are explored, are both new and definite improvements. There was no intention of taking any credit for the stand away from Dr Barkan, and I hope that this letter will correct any such impression that may have been given.

JOHN M McLEAN, M D, Baltimore

News and Notes

PERSONAL

Notice has been received of the death of Mr Jujuro Komoto, honorable professor of the Tokyo Imperial University. Professor Komoto was born on Aug 16, 1859, in Hyogo-ken. After graduation from the Tokyo Foreign Languages School and the Department of Medicine of Tokyo University in 1883, he was sent abroad by his government, where he continued his studies in Germany and Austria from 1885 to 1889. On his return from study abroad he was appointed professor of the College of Medicine of Imperial University, Tokyo, in June 1889 and was simultaneously appointed to the chair of ophthalmology in the same college. The former College of Medicine became the Faculty of Medicine in 1915, and Professor Komoto was appointed (in succession) again as professor of ophthalmology. Almost all of his important publications were in Japanese. He died on April 4, 1938, at the age of 79, after a long and painful illness.

SOCIETY NEWS

Journal of Social Ophthalmology.—The International Association for the Prevention of Blindness, is publishing a new organ, the *Journal of Social Ophthalmology*, for the purpose of keeping before the minds of social workers and health officials the importance of the health of the eye and the economic disability that arises from blindness. It is not a clinical journal. The first number is printed in English and French in parallel columns. As occasion suggests, other languages may be employed. The journal is sent free to subscribers to the association, 66, Boulevard Saint-Michel, Paris.

GENERAL NEWS

Exhibition at the Royal Eye Hospital—An exhibition dealing with the prevention of industrial injuries to the eyes was opened at the Royal Eye Hospital (Royal South London Ophthalmic Hospital, Inc.) on June 22 by the Earl of Athlone, K.G. Addresses were made at the opening of the exhibition by the chairman of the hospital, Mr L. Vernon Cargill, F.R.C.S., the Earl of Athlone and Mr J. Minton, F.R.C.S., medical representative of the Industrial Eye Injuries Committee.

The exhibition included a display of different types of goggles for such occupations as grinding, welding and riveting and metal-sputtered goggles, glare and dust proof, which are used by pilots of the Royal Air Force and Imperial Airways. A film was shown at the opening of the exhibition illustrating the protection of eyes in industry. It showed a workman suffering from an ocular injury received because he was not wearing his goggles or using a safety screen. Men working in factories doing various types of engineering work were shown using the necessary safety appliances.

Other demonstrations included one of oxyacetylene welding and electric welding by workmen wearing goggles and protective screens. Large photographs around the walls showed other workers either using or neglecting to use protective appliances.

A committee has been formed by the hospital to further the object of making employers conscious of the necessity of safety measures, and, a matter of perhaps equal importance, of combating prejudice or negligence among workmen whom the appliances are intended to protect. Part of the exhibition is to remain in the casualty department of the hospital as a permanent museum. It will thus come to the notice of the 7,000 workmen who are treated at the hospital every year for ocular injuries, many of them serious.

CORRECTION

In the abstract of the report by Dr I S Tassman entitled "The Use of Paredrine in Cycloplegia," which appeared in the proceedings of the College of Physicians of Philadelphia, Section on Ophthalmology for March 17, 1938, in the July issue of the ARCHIVES (20 156, 1938), the second sentence in the fifth paragraph should read

"After experimenting with solutions of different strengths, it was found that a 4 per cent solution of homatropine hydrobromide followed in three minutes by 1 drop of a 1 per cent solution of paredrine hydrobromide produced satisfactory results."

Abstracts from Current Literature

EDITED BY DR WILLIAM ZENTMAYER

Congenital Anomalies

COLOBOMATA OF THE OPTIC NERVE SHEATH IN RATS J V V NICHOLLS and K TANSLEY, Brit J Ophth 22: 165 (March) 1938

This study is based on the results of examination of histologic sections of the eyes of 10 rats. There was ectasia of the retina passing between the edge of the lamina cribrosa and the sclera into the optic nerve sheath. The essential feature in this series was poor development of the lamina cribrosa. There is some difficulty in evaluating the relation of the ectasia to the weakness of the lamina cribrosa. Whether the ectasia follows the collapse of the lamina or both conditions are part of the same process is difficult to determine. The second view is supported by Kayanaga. According to him, there is an abnormality in the condensation of the mesoderm about the neck of the primary optic vesicle, so that small pouches of neural ectoderm are nipped off about the future nerve head. Because of, or along with this, there is poor development of the lamina cribrosa. This explains how in some of the authors' cases the ectasia completely surrounded the optic nerve within its sheath, a previously undescribed condition.

W ZENTMAYER

Experimental Pathology

THE GALACTOSE CATARACT. CLINICAL OBSERVATIONS T SASAKI, Arch f Ophth 138: 351 (Feb) 1938

Sasaki made thorough clinical (including use of the slit lamp) studies of the ocular changes which occurred in young rats kept on a diet consisting of 50 per cent of galactose. The observations of Mitchell, Dodge, Yudkin and Arnold with regard to galactose cataract were confirmed. The galactose cataracts were found to have a characteristic clinical picture and to take a regular course. The first opacities developed in from three to seven days after the diet was started, these opacities were situated around the sutures and were subsequently covered by subcapsular radial (spokes and water clefts) opacities. The cataracts became complete within from twenty-two to twenty-eight days.

P C KRONFELD

THE PROPERTY OF GALACTOSE CATARACTS TO PROTECT VITAMIN C FROM OXIDATION T SASAKI, Arch f Ophth 138: 380 (Feb) 1938

The cataractous lenses of rats fed a diet consisting of 50 per cent of galactose protect (in in vitro experiments) vitamin C from oxidation. This property of galactose may be due to the diffusion of a reducing substance (glutathione?) out of the lens into the surrounding solution,

which contains vitamin C. The increased permeability of the capsule of a galactose cataract would facilitate this diffusion.

P. C. KRONFELD

General Diseases

OCULAR LEPROSY. REPORT OF TWENTY-TWO CASES. VILLARD, BOUNIOL, VIALLEFONT and FRIENTÈS, *Bull. Soc. d'opht. de Paris*, July 1937, p. 348.

Of 31 lepers examined in a colony at Valbonne, 22 presented ocular lesions. A short résumé of the ocular changes noted in each patient is given. The authors comment on the frequency and the gravity of ocular complications in cases of leprosy, on the fact that so many of the patients had contracted leprosy in France, and on the uncharacteristic nature of the ocular lesions as compared to those associated with tuberculosis. Except for a type of iritis with small white grains formed on the collarette of the iris, no characteristic sign of leprosy was noted. Scleral and episcleral lesions were not abundant in this group. Operative procedures were seldom used in the treatment of ocular leprosy, but of those necessary, iridectomy was most often required. The authors feel that complications from leprosy and tuberculosis are comparable in relation to involvement of the eyes.

L. L. MAYER

General Pathology

FURTHER CONTRIBUTION TO THE KNOWLEDGE OF GUNN'S SIGN. L. SALLMANN, *Arch. f. Ophth.* 138: 412 (Feb.) 1938.

A man aged 77 suffered from a decompensated hypertensive cardiovascular disease with arteriosclerosis and arteriolosclerosis. Ophthalmoscopic examination revealed narrow and straight retinal arteries with exaggerated reflex stripes and delicate white sheaths. Nicking of the veins was present at several places. Pathologic examination of these places revealed as cause of the nicking a definite thickening of the adventitia of both vessels at the place of the crossing. Nicking of the veins, or Gunn's sign, is, therefore, a sign of localized vascular sclerosis and not of displacement of the vein by the rigid artery.

P. C. KRONFELD

Lens

ARREST OF NUTRITIONAL CATARACT BY USE OF RIBOFLAVIN. P. L. DAY, W. J. DARBY and K. W. COSGROVE, *J. Nutrition* 15: 83 (Jan.) 1938.

Day and his associates gave young albino rats a diet deficient in riboflavin and examined their eyes at frequent intervals with the ophthalmoscope. Of 16 control animals receiving the deficient diet without supplement, cataract developed in 13 in an average of fifty-two days. The cataract proceeded to maturity in 12 of these rats in an average of sixty-seven days. The average time of survival was seventy-four days. When early cataractous changes were evident, 25 rats were given intra-

muscular injections of riboflavin in doses of 120 micrograms twice a week. The animals rapidly increased in weight, new hair appeared on those rats with alopecia, and keratitis cleared up slowly. In 11 of these rats cataract was arrested in both eyes. In each of 6 other rats the cataract proceeded to maturity in one eye, but its progress was definitely arrested in the other eye. The cataract proceeded to maturity in both eyes in 2 rats. Six rats were found to have clear lenses in both eyes after the keratitis cleared. It is thus apparent that the progress of the development of cataract was arrested by the administration of riboflavin in 17 of 19 animals exhibiting cataract. These data furnished additional evidence that riboflavin is the cataract-preventive vitamin.

J A M A (W ZENTMAYER)

EXFOLIATION OF THE LENS CAPSULE IN GLAUCOMA A GARROW, Brit J Ophth 22: 214 (April) 1938

Garrow gives a brief review of the literature on exfoliation of the capsule of the lens in cases of glaucoma, describes the several types of exfoliation which have been observed and gives a detailed description of his own cases with line drawings of the appearance in each case.

He gives the following summary:

Fifty-one glaucomatous patients were examined for exfoliation of the capsule. This condition was found in 8. Two of these patients had a congestive attack when they first came under observation.

No record was kept of the number of nonglaucomatous patients examined with the slit lamp during this investigation, but exfoliation of the capsule was found only twice in such patients.

The article contains several plates.

W ZENTMAYER

TECHNIC OF TOTAL EXTRACTION OF CATARACT WITH FORCEPS R DE SAINT-MARTIN, Ann d'ocul 174 750 (Nov) 1937

The April 1937 issue of the *Annales d'oculistique* contains an interesting article by Dr. Porte, of Geneva, on the intracapsular extraction of cataract. In this article he disapproves of the technic of extraction with forceps, which today is considered classic and is known under the names of the technic of Stanculeanu, Knapp, Torok and Elschmig. Elschmig renamed this technic the "Hungarian method."

The author of the present article accepts the superiority of this method without discussion. He gives his reasons why he considers the conclusions of Dr. Porte unreliable and liable to mislead operators who are not too familiar with surgical work on the lens and consequently should not undertake this type of operation.

S H MCKEE

Lids

STAPHYLOCOCCIC SEPTICEMIA FOLLOWING A SUPPURATING MEIBOMIAN CYST. REPORT OF A CASE R GOZBERK, Ann d'ocul 175. 159 (Feb) 1938

A furuncle or anthrax at any site may be the cause of staphylococcic septicemia. In this respect furuncle of the face merits special consider-

ation, as anastomosis of the facial vein with the superior ophthalmic vein renders the passage of micro-organisms toward the cavernous sinus easy. Suppurative phlebitis of the orbital veins and of the cavernous sinus is a not uncommon complication of furuncle of the side of the nose, the eyebrow, the forehead or the upper lip.

Different authors are cited as having published reports of cases from 1871 to 1933. A case is reported in detail. A boy of 11 years was seen because of an acute inflammatory exophthalmos, with most serious symptoms. The child had had measles the previous month, and convalescence had been interrupted by acute enteritis. One week before the child was seen, there appeared on the right upper lid a reddish swelling, and three days later chills and high fever suddenly developed, in two days a marked proptosis of the eye occurred. There followed a generalized staphylococcic septicemia, with abscesses in the orbit and conjunctiva. Postmortem examination showed abscesses in the brain, lungs and kidneys, all of which had the same character. It is believed that the measles lowered the resistance of the patient to such an extent that secondary infection progressed easily. Two illustrations accompany the article.

S. H. McKee

Methods of Examination

A SIMPLE ACCESSORY FOR THE DARK ROOM. J. SHERNE, *Brit J Ophth* 22: 109, 1938

The device described here is a reflector such as cyclists use instead of a rear light. It is affixed to the wall of the dark room directly opposite to and on the same level as the retinoscopic light. It produces a brilliant red fixation spot, this interests children and can be seen by even the most myopic. It is a great advance over the other methods for the production of relaxation of accommodation.

W. ZENTMAYER

THE USE OF SODIUM AND MERCURY VAPOR LAMPS IN OPHTHALMOLOGY. A. J. BALLANTYNE, *Brit J Ophth* 22: 204 (April) 1938

Ballantyne describes a convenient form of mercury vapor lamp of durable construction and of moderate cost. For most practical purposes, it is a satisfactory substitute for the Vogt red-free lamp, and it has the advantage that it is less disagreeable for the patient and creates less dazzle for the examiner.

The sodium vapor lamp has the important qualities that it is almost purely monochromatic and that the light is largely absorbed by the blood in the retinal vessels. These two properties give the light a notable resolving power.

The author describes the details of the fundus as observed under the two types of lamp.

W. ZENTMAYER

Neurology

NEURAL CORRELATIONS OF VISION AND THEIR SIGNIFICANCE FOR LOCALIZATION OF TUMORS OF THE BRAIN. C. A. ELSBERG and H. SPORNITZ, *Arch Neurol & Psychiat* 39: 315 (Feb) 1938

The authors present a preliminary report in which they describe functional visual tests such as dark adaptation flicker and the threshold

for light as related to minimal lighting and the area of illumination. They attempt to interrelate these visual functions in the normal person and in the person with a pathologic process of the visual tracts. Using white objects against a black background and white test objects with a black center, they found varying relations between the area of the test object and the minimum light required for vision, which, for each person, could be formulated into constants. This raised the question as to whether or not the constants were dependent on the amounts of energy received at the cones, bipolar cells and ganglion cells of the retina. The authors felt that the interrelations found did demonstrate that summation and inhibitory effects are important in retinal perception.

The use of these functional visual tests for the localization of lesions in the retina and visual tracts is discussed and illustrated. The authors realize that the study is as yet of theoretic interest only, but they are continuing the work, as there are indications of practical possibilities. Ophthalmologists should be interested in this article from the standpoint of visual physiology.

R IRVINE

A CASE OF INTRAOCULAR NEUROMA (RECKLINGHAUSEN'S DISEASE) OF THE LEFT OPTIC NERVE HEAD H B STALLARD, Brit J Ophth 22 11 (Jan) 1938

Stallard enumerates and describes in some detail the ocular complications of Recklinghausen's disease.

The author gives the following resume of the case reported. A young man aged 19 years had a neuroma affecting the optic nerve head of the left eye. His family history showed that in the two generations preceding his there had been 2 cases of multiple neurofibroma affecting the central nervous system and the nerves serving the special senses of sight and hearing. The histologic picture of the neoplasm affecting the optic nerve head is described, and photomicrographs illustrating these changes are shown. Three years after the disease had been discovered in the left eye, signs of raised intracranial pressure developed due to a neoplasm or possibly to several neoplasms inside the skull. The right auditory nerve was involved. A subtentorial decompression was followed two days later by death.

W ZENTMAYER

RELATION OF THE SENSORY VISUAL CORTEX TO THE OCULOMOTOR CORTEX E CLAES, Compt rend Soc de biol 127 1116, 1938

The experiments on cortical function were made on cats with the brain isolated by suboccipital section of the cord, a procedure which permits a truer physiologic response than general anesthesia. Each hemisphere has two oculomotor areas. The anterior, located in the sigmoid gyrus, produces when stimulated a bilateral and equal dilatation of the pupil, and the eyes move (up or down), reversing the position held before stimulation. The posterior area in the posterior portion of the suprasylvian gyrus on stimulation provokes a conjugate deviation to the opposite hemisphere. The posterior motor areas are mutually inhibitory but the motor areas of the same side are synergistic, and the anterior motor areas are apparently unconnected with each other. Wake-

fulness and activity of the striate and auditory areas have each a tonic cerebral action and effect a lowering of the threshold stimulus of either oculomotor area

J E LEBENSOHN

Ocular Muscles

ON THE VALUE OF ORTHOPTIC TRAINING F W LAW, Brit J Ophth 22: 193 (April) 1938

There are special reasons why orthoptic training should be scrutinized as to results. The recent interest in this branch of ophthalmology has resulted in a large number of medically unqualified persons undergoing a course of study subsequent to undertaking the training of squinting children, which training is usually of a protracted nature. Aided by the somewhat apathetic acquiescence of the majority of ophthalmic surgeons, the practice of orthoptics from some points of view has reached the position of a vested interest, and its uncritical acceptance is all that is needed to insure its persistence, quite apart from its excellence.

Law gives a detailed analysis of 91 cases in which orthoptic training was used.

After a consideration of the facts obtained, the author arrives at the following conclusions:

A far greater proportion of persons with squint than is generally supposed can be cured by glasses and, when necessary, by occlusion.

Only those persons for whom such measures have failed should be submitted to orthoptic training.

Patients should be selected for training on the basis of clinical merits and with due consideration for the end result desired and for the justification for the means necessary to obtain it.

The results of orthoptic training are far less dramatic and far less certain than they are held to be in some quarters, and the descriptions of the value of such training which have appeared in the medical press have all erred on the side of optimism.

W ZENTMAYER

A STUDY OF MINOR'S NYSTAGMUS RAYMOND S BROCK Brit M J 1: 443 (Feb 26) 1938

This paper is based on a study of nystagmus in 15 pits of North Wales over a period of ten years. The nature of the ocular movements is studied, and the various theories as to their cause are discussed. The importance of psychologic factors is considered, and suggestions are offered, first, as to the prevention of the disability and, secondly, as to a more secure basis on which the disability may be assessed.

The author's conclusions are summarized as follows:

1 Prolonged work below ground may bring about oscillation of the eyes which does not necessarily cause disablement.

2 When disability does arise it is usually due to an associated psychoneurosis.

3 The prevention of this depends on (a) recognition of the foregoing facts, (b) the treatment of contributory disorders, (c) dimin-

ished stress in the mine, (d) the barring of unsuitable applicants, (e) improved economic conditions, and (f) stricter assessment of disablement

ARNOLD KNAPP

Pharmacology

EFFECT OF AN EXTRACT OF CILIARY BODY ON BLOOD PRESSURE
D MICHAÏL and P VANCEA, *Compt rend Soc de biol* 127 453, 1938

An extract of ciliary body was made according to the general technic advised by Roger for the preparation of organic extracts. The intravenous injection of a small amount produced a slight fall in blood pressure followed by a notable rise, injection of a larger amount caused a slowly progressive rise from the start. These changes were unmodified by previous injection of atropine or ergotamine but were increased by previous section of the vagus nerves.

J E LEBENSOHN

EFFECT OF AN EXTRACT OF CILIARY BODY ON ISOLATED FROG HEART
D MICHAÏL and P VANCEA, *Compt rend Soc de biol* 127.455, 1938

The frog heart was isolated according to the technic of Straub and treated with an extract of ciliary body. Small doses produced a progressive depressive action, ending in complete ventricular arrest. Larger doses produced immediate cardiac arrest in diastole. In both instances reanimation ensued after irrigation with Ringer's solution.

J E LEBENSOHN

Physiology

A STUDY OF DIET IN RELATION TO HEALTH. DARK ADAPTATION AS AN INDEX OF ADEQUATE VITAMIN A INTAKE. J R MUTCH and H D GRIFFITH, *Brit M J* 2.565 (Sept 18) 1937

The association of night blindness with diet was accepted long before studies on vitamins were begun. With the rapid improvement in diet since the beginning of the century, obvious deficiency diseases have become more and more uncommon. As the important dietary sources of vitamin A are relatively expensive foodstuffs, it is evident that vitamin deficiency will be more common among the poorer people. Preliminary to a study of diet relative to health, tests have been made to discover the extent to which deficiency of vitamin A exists and the daily requirements of vitamin A to insure perfect health.

Given an accurate measure of the power of adaptation, the following two questions will arise: 1. To what extent does difference in the power of adaptation occur? 2. Can adaptation be improved by giving vitamin A?

The power of dark adaptation was estimated by Edmund's test type and Tscherning's photometric glass. A limit is shown below which all subjects react, with improved adaptation to administration of vitamin A. Those whose first performance is above this limit give no response to

the administration of vitamin A. The test is sufficiently simple to be applied to intelligent children of 6 years and so short that it is suitable for serial examination. The results are unaffected by practice.

ARNOLD KNAPP

The Pupil

THE INCIDENCE OF UNEQUAL PUPILS IN UNCONVICTED PRISONERS
H. K. SNELL and G. A. CORMACK, *Brit M J* 1:672 (March 26) 1938

Two hundred and ninety-five of 3,000 unconvicted prisoners showed persistent anisocoria. No cause could be found in 139. An error of refraction was the only anomaly in 62. There was a history of syphilis in 30, and in the remaining 64 the inequality was due to various causes. The anomaly was associated with mental disease in only 4. The condition was therefore of no significance in approximately two thirds of the subjects, and in the others it was associated with syphilis and a history of injuries to the head, conditions which may be related to mental disorders. If laboratory tests are not available, anisocoria should be regarded with suspicion and may be of medicolegal significance.

ARNOLD KNAPP

Refraction and Accommodation

TEMPORARY SPASMODIC MYOPIA DUE TO ARSPHENAMINE E. DE C. FALCÃO, *Ann d'ocul* 174:847 (Dec) 1937

The introduction of arspenamine into the human body is often followed by toxic conditions which manifest themselves in different forms. Of these complications, many have been observed and reports on them have been published, others have not been recorded, so that reports of new cases are always worthy of note.

The author reports what is to him an interesting case of arspenamine intoxication. Spasmodic contraction of the ciliary muscle appeared in a patient 33 years of age after the intramuscular injection of arspenamine. This caused a change in the refraction of the lens and transient acute myopia. A detailed description of the case is given.

The article closes with a summary of the previous case reports that have been published.

S. H. McKEE

INTRACAPSULAR ACCOMMODATION OF THE LENS F. BOSA, *Ann di ottal e clin ocul* 65:621 (Aug) 1937

The author calculated the dioptric power of the lens according to a modification of Gullstrand's equation. He found it to be 20.28 diopters in the nonaccommodated lens. In the accommodated lens without consideration of the increase in the total index of refraction, it was 28.30 diopters. When the increase in the total refractive index resulting from a change in the curvature of the nucleus was considered, the dioptric

power was increased to 32.25 diopters. The difference between the two latter figures, or 3.95 diopters, would then represent the increase in refractive power to intracapsular accommodation.

S. R. GIFFORD

Retina and Optic Nerve

EDEMA OF THE RETINA. P. BAILLIART, *Ann. d'ocul.* 175: 133 (Feb.) 1938.

From reading the ophthalmic classics one would think that edema of the retina is rare. It is characterized by an increase in the quantity as well as by an alteration in the makeup of the interstitial fluid. Edema naturally accompanies all hemorrhages, which are so frequently found in the center of the retina. There are many causes that bring on retinal edema. They may be circulatory or inflammatory and may originate within the tissues themselves. It must be understood that certain modifications, mechanical or chemical, in the composition of tissue make them capable of retaining an abnormal amount of interstitial fluid. According to Achard and Loeper and Widal and Lemierre, an excess of salt in the blood of an edematous person is deposited in the tissues. In some persons with swollen lids, edema of the retina is often seen without arterial hypertension. Achard believes that all chemical modifications of the interstitial fluid, whether they result in an abnormal amount of fluid or not, may provoke edema. Renal changes act in a similar manner. The action of cholesterol is also known.

The histologic constitution of the retina is less predisposed to edema than other tissues, as elsewhere there are zones of predilection. The author describes in detail some aspects of edema of the retina, the mechanism of the appearance of edema and the subjective symptoms.

S. H. MCKEE

TREATMENT AND DEVELOPMENT OF TWO CASES OF THE SYNDROME OF GRONBLAD AND STRANDBERG (ANGIOID STREAKS IN THE RETINA AND PSEUDOXANTHOMA ELASTICUM). R. FRYDMAN, *Ann. d'ocul.* 175: 154 (Feb.) 1938.

There are numerous articles on this subject, the most important of which is that of Gronblad and Strandberg. In a recent article, Franceschetti and Roulet gave a general description of the disease and its relation to other conditions.

Two cases of this syndrome are described in detail, and a chart showing the results of the treatment by injections of calcium in the first case is presented.

Frydman concludes from the course of the condition in these 2 cases that calcium and vitamins B₁ and C as prescribed by Franceschetti have given satisfactory results. This treatment has a remarkably rapid action, but its effect is nil in cases of lesions of long standing, and it does not prevent new hemorrhages from forming. Patients in whom angioid streaks of the retina occur have had repeated retinal hemorrhages. When a retinal hemorrhage has occurred, treatment by calcium and vitamin C should be begun. Such patients should receive a series of ten treatments every two months.

S. H. MCKEE

THE ANGIOSCLEROTIC AND ANGIOSPASTIC RETINOSES D CATTANEO,
Ann di ottal e clin ocul 65:721 (Oct) 1937

A number of forms of retinal disease are discussed which the author believes are caused by sclerosis or spasm of the retinal vessels. The literature on these conditions is reviewed, including that on circinate retinitis (Fuchs), senile macular exudative retinitis (Coppez and Danis), disciform degeneration of the macula (Kuhnt and Junius), central capillarospastic retinitis (Horniker) and retinal capillaritis (Bailliant). The close relation which exists between the first three conditions is emphasized, senile macular exudative retinitis and disciform degeneration of the macula being perhaps identical conditions which are often associated with the picture of circinate retinitis. The author believes these conditions to be based on organic changes in the arteries and the arterioles. The two last-named conditions described by Horniker and Bailliant represent the results of spasm affecting the small arterioles or capillaries of the macular region. At the onset there are edema and a paracentral scotoma. The edema disappears, and a number of white spots resembling Gunn's dots are left.

The author reports 17 cases in which the small lesions characteristic of capillaritis, or, as he prefers to call it, capillarosis, were combined with other changes due to involvement of the larger vessels. Complete studies were carried out in each case, including measurement of the retinal blood pressure with the dynamometer, determination of the capillary index according to the method of Fritz and in most cases endoscopic study of the circulation, the pressure which was required to obliterate the visible movement of corpuscles being recorded.

The small lesions characteristic of capillarosis were present in all cases. In 2, they were associated with central disciform degeneration, in 1, with senile cystic degeneration, in 1, with circinate retinitis, and in 2, with chronic glaucoma. The general systolic and diastolic blood pressure and the pressure in the retinal arteries were elevated in most but not in all cases. In 2 cases marked general and retinal hypertension were present, with no signs of arteriosclerosis, and in these cases the small lesions which were present must be considered due to vascular spasm. The lesions may best be interpreted as foci of degeneration due to an insufficient blood supply.

The author believes that the whole group of changes considered may well be named angiosclerotic and angiospastic retinoses and may be divided, according to location of the lesions, into three types. The macular type would include senile macular degeneration, senile exudative macular retinitis and central disciform degeneration. The paramacular type would include retinitis circinata. The disseminated type would include isolated and diffuse capillaritis.

A bibliography accompanies the article. SANFORD R. GIFFORD

PATHOGENESIS OF CHOKED DISK F SCHIECK, *Arch f Ophth* 138.
48 (Oct) 1937

Schieck performed the following experiment on a fresh clinically normal human eyeball with the whole orbital portion of the optic nerve attached to it. The specimen was obtained from a patient in whom a

recurrent sarcoma of the orbit necessitated exenteration. Immediately after the operation he injected india ink under moderate pressure into the intravaginal space of the optic nerve and then fixed, embedded and sectioned frontally the whole specimen. Microscopic examination of the sections revealed filling of the intravaginal space, or rather of the space situated between the inner surface of the dura and the pia covering the nerve. In addition, particles of ink extended along the outer surface of and into the strand of tissue which accompanies the central vessels, into the optic nerve. Except for this axial strand of tissue, the optic nerve was free of particles of ink. This experiment proves that "there is an open communication between the intravaginal space of the optic nerve and the tissue spaces of the axial strand." According to Schieck, in diseases which are associated with increased intracranial pressure the cerebrospinal fluid gains access to the distal portion of the optic nerve through this axial strand of tissue. No particles of ink were found in the orbital tissues outside the dura. Schieck, therefore, doubts the existence of the physiologic stream of tissue fluid which, according to Behr (*Arch f Ophth* 134: 249, 1937), goes from the intravaginal space by preformed channels into the orbital tissues.

P C KRONFELD

Trachoma

TREATMENT OF TRACHOMATOUS PANNUS BY SUBCONJUNCTIVAL AUTOHEMOTHERAPY. DELORD, Bull Soc d'opht de Paris, July 1937, p 372

The author reports the results obtained in the treatment of 6 patients with trachoma by means of subconjunctival autohemotherapy. From five to seven injections are advised at intervals of from ten to fifteen days, to allow for absorption.

The advantages of the method are listed as follows. It is simple, rapid, painless and efficient, and the patient need not be hospitalized. It is also analgesic and lessens photophobia.

L L MAYER

TREATMENT OF TRACHOMA WITH TRACHOZID. L KEPPICH-OLAH, Klin Monatsbl f Augenh 99 234 (Aug) 1937

Trachozid, prepared at the Serotherapeutic Institute at Vienna, is a derivative made from animal toxin by a special process. Its action is nontoxic for the eye and the entire organism. The author corroborated the favorable results obtained by other writers after treating 100 patients with trachoma at the ophthalmic department of the Royal Hungarian Hospital at Gyula. All her patients, mostly with stubborn and protracted trachoma, were benefited. The pannus receded promptly, lesions and ulcerations of the cornea healed promptly, and the tendency of the pannus to recur was diminished. The duration of the treatment was considerably reduced, so that the patients could return to work sooner than after other therapeutic procedures. Hospitalization was not required.

K L STOLL

Tumors

A MELANOMA OF THE IRIS WITH PATHOLOGICAL FINDINGS F T
TOOKE, Brit J Ophth 22:153 (March) 1938

In a man aged 24, a spot of pigment the size of a pinhead had appeared on the right iris seven years previously and had gradually increased in size until it extended from a point just beyond 9 o'clock at the pupillary margin below to within 2 mm of the corneoscleral margin above, and upward from 11 30 o'clock below at the pupillary margin to the limitations of the root of the iris above. The mass was melanotic and slightly elevated. It was removed by iridectomy. A detailed description of the histologic features of the tumor is given, and a critical résumé of the opinions advanced to date regarding the derivation of the cells responsible for pigmented growths is given.

The author states that "these mesoblastic melanoblasts of the iris stroma have the appearance of mesoblastic cells as our bleached sections show and many on very good grounds would call them mesoblastic. It is true that they are 'dopa' positive (i.e., they contain a specific ferment for producing melanin from dihydroxyphenylalanine) which is an ectodermal characteristic. If it is true that they are mesodermal then we have another instance, of which in the eye there are many, of a phylogenetic aberration. These are cells which partake of the characteristics of both ectoderm and mesoderm. One might mention as another instance the development of the smooth muscle of the iris from ectoderm."

The article is illustrated

W ZENTMAYER.

NEVOCARCINOMA OF THE LACRIMAL CARUNCLE CLINICAL AND ANA-
TOMIC STUDY F TERRIEN and P VEIL, Arch d'opht 53:721
(Oct) 1936

The rarity of malignant tumors of the caruncle and the variety of their anatomic appearance make them difficult to diagnose clinically. The authors describe a case in which the patient was followed until death, autopsy was not performed. Photographs of the gross specimen and microscopic sections are presented. In spite of its relatively benign clinical appearance and immediate extensive excision, recurrence took place in two months. As the patient refused exenteration, at first radium and roentgen therapy were tried, but without benefit. Later operation was done. The inefficacy of radiotherapy in these cases is generally recognized. The authors affirm that exenteration done early is the best treatment. The discovery of the presence of nevus cells and malignant changes on histologic examination implies a particularly gloomy prognosis for life.

S B MARLOW

TREATMENT OF EPITHELIOMA OF THE EYELIDS DUCUING, COUADAU
and LU-VAN-XUONG, Arch d'opht 53:800 (Nov) 1936

This report, which is divided into five sections, deals with the therapeutic methods available for the treatment of epithelioma of the eyelids, their advantages and disadvantages, the opinions expressed in

the literature by other writers, the therapeutic indications according to their technic and the results obtained by them in 55 cases. In cases of circumscribed epithelioma of the skin, preference is given to radium by surface application alone or combined with the use of radium needles. In cases in which the epithelioma involves the free border of the lid, radiopuncture is advocated. When the commissures are affected, if the lesion is limited to the skin, radium needles alone or combined with plaques are used. If the conjunctiva or the lacrimal sac is affected, extensive excision followed by the use of radium is indicated. If the palpebral conjunctiva alone is involved wide excision of the involved lid is advocated. If the bulbar conjunctiva or the globe itself is affected, enucleation with emptying of the orbit is selected. If the bones are invaded, wide resection by means of electrosurgery avoids extensive hemorrhage. The results obtained with these various methods of treatment in the 55 cases are as follows: roentgen irradiation in 1 case, with cure, the diathermic needle in 1 case, with cure, radium therapy and surgical treatment in 3 cases, with cure in 2 and improvement in 1, surgical treatment alone in 4 cases, with cure, radium therapy in 46 cases with cure in 34 (73.9 per cent), improvement in 5 and failure in 5. Two of the patients in the last group were lost from observation. These results are compared with those reported by others. The percentage of cure varied from 66 to 97.

S. B. MARLOW

GENERAL CONSIDERATIONS OF PSAMMOMAS APROPOS OF A CASE OF CYSTIC PSAMMOMA OF THE OPTIC PAPILLA. R. ARGAUD and A. COUADAU, *Arch. d'opht.* 53: 869 (Dec.) 1936.

Psammomas of the optic nerve are rare, and cysts of the papilla are more rare. The authors report a case in which after the subsidence of an attack of acute glaucoma ophthalmoscopic examination revealed a brilliant yellowish mass containing small crystals covering the disk. A subsequent attack of glaucoma necessitated enucleation. The histologic structure of the mass is described in detail, and a diagnosis of psammoma was made. A brief review of the probable mode of formation of such tumors is presented.

S. B. MARLOW

Therapeutics

FEVER THERAPY IN OPHTHALMOLOGY. L. HAMBRESIN, *Ann. d'ocul.* 174: 721 (Nov.) 1937.

Although the attention of oculists has been called to the therapeutic use of induced fever, it is not employed as a rule in ophthalmic practice. Articles on this subject are still scarce, especially in the French language.

Hambresin discusses the essential difference between fever therapy and the therapeutics of shock, as the two methods are often confused, and the latter is particularly employed in the practice of ophthalmology. The majority of oculists use shock medication, the most popular form being the injection of whole milk. Pyretotherapy is the treatment of disease by raising the temperature of the body. The fever cures by rendering the ordinary therapeutic measures more active or by making

the organism more receptive to these. With good reason, C. Richet and A. Meyer-Heime stated that "shock is one thing, pyretotherapy is another." Therapeutic shock may produce good results, even without the elevation of temperature.

The writer concludes that pyretotherapy, or fever therapy, is useful in the treatment of simple atrophy of the optic nerve. The results are not permanent or perfect, but it seems to be the only treatment that halts the progress of the lesion. Certain specific pupillary conditions are also helped, and good results may be obtained in cases of syphilitic parenchymatous keratitis, keratitis, iritis and iridocyclitis of all kinds.

S. H. McKEE

Society Transactions

EDITED BY W L BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W WHITE, M D, *Chairman*

March 21, 1938

RUDOLF AEBLI, M D, *Secretary*

CHOROIDAL HEMORRHAGE SIMULATING A CHOROIDAL TUMOR DR MARTIN COHEN

A K, a woman aged 51, had a history of marked systemic hypertension for the past six or seven years, but otherwise she was essentially normal. The menopause occurred at the age of 49. At the time of the last examination the patient had considerable pallor of the skin and conjunctivas. The heart was slightly hypertrophied. The Wassermann reaction was negative, and the renal function was normal. The blood pressure was 290 systolic and 140 diastolic. The hemoglobin content was 70 per cent.

The patient was referred to me on Jan 19, 1938, complaining of sudden excruciating pain in the left eye. Examination showed vision in the right eye to be 20/20. The fundus, aside from slight arteriovenous compression of the superior temporal vein above the disk, was essentially normal. Vision in the left eye equaled 20/20. With the exception of slight venous compression, the fundus appeared normal, but the ocular conjunctiva was markedly congested. Hot compresses, acetylsalicylic acid and absolute rest were prescribed with the object of alleviating the pain.

The patient returned the following day and reported that the severe pain still persisted. Slit lamp examination showed no iritic involvement, and the intraocular tension was normal. The previous treatment was continued, and a general physical examination was advised.

The pain in the left eye gradually subsided, and the patient returned two weeks later for reexamination, complaining of a cloud in front of the left eye. Vision in the right eye was now 20/30, and the fundus showed moderate edema of the temporal side of the disk. There were a few hemorrhages and lymphocytic infiltrations around the disk, with some dots of pigment in the periphery. Vision in the left eye had also diminished slightly (20/30). Ophthalmoscopic examination of this eye revealed a large, ovoid, flat blackish mass, about two and one-half times the size of the disk, in the fundus peripherally down and in, causing a scotoma above and temporally. The mass was slightly elevated, with an ill defined margin, and no vessels were visible on its surface. Transillumination was deficient over the dark area. The surrounding retina appeared hyperemic, and floating opacities could be seen in the vitreous. The intraocular tension was 18 mm of mercury (Schiotz) in each eye.

Recent examination of the right eye showed further diminution of vision (20/70). There was engorgement of the retinal veins and slight contraction of the arteries, with prominence of the central light reflexes, the smaller arteries were not perceptible. The outline of the disk was evident on all but the temporal margin, which was edematous. Several fresh hemorrhages bordered the venous branches, the lymphocytic infiltrations were more numerous, and the dots of pigment which resulted from previous hemorrhages were being absorbed. Vision in the left eye, which had at one time been reduced to 20/50 had improved to 20/30. The opacities of the vitreous had been largely absorbed, although the optic disk could not yet be perceived. The large blackish mass was now steel gray, owing to a commencing organization of the blood clot. Transillumination showed that the mass was contracting, and its outline at the upper margin was becoming evident. The surrounding retina appeared hyperemic, the retinal vessels were distinctly visible.

The diagnosis on the basis of the fundus picture was arterio-sclerosis of the right eye, and localized choroidal hemorrhage in the left eye. The general diagnosis was arteriosclerosis and essential hypertension of a severe type.

The treatment consisted of absolute rest and general measures for the systemic hypertension.

Conclusion—The excruciating pain in the left eye was probably caused by the localized pressure of the encapsulated blood clot on the surrounding neural elements. Choroidal hemorrhage has seldom been observed and reported in cases of disease of the vascular walls. It is difficult, therefore, to state definitely whether there is an underlying growth. However, from the progress of the condition, the accompanying systemic condition and the degeneration of the vascular walls in the opposite eye, it is inferred that the lesion in the left eye is probably a choroidal hemorrhage due to a venous thrombosis rather than a choroidal tumor.

DISCUSSION

DR LEWIS WEBB CRIGLER. Several years ago I reported before this section a case of subchoroidal hemorrhage in which a diagnosis of sarcoma of the choroid had been made by three ophthalmologists. Not until the enucleated eye was sectioned and examined microscopically was the true diagnosis made. Fortunately for both the patient and the oculist, the eye had to be removed on account of extreme pain and tension. All preception of light had been lost.

Investigation revealed that the patient was a free bleeder. At one time after a slight fall there had been a profuse subcutaneous hemorrhage between the knee and the hip. At another time after the extraction of a tooth there was bleeding from the socket for several days. With this experience before me, I have been constantly on guard to avoid a similar mistake.

Several months ago a patient 70 years of age was referred to me by Dr Beck, of New Rochelle. Dr Beck had seen this patient just a week previously and found both fundi normal except for mild signs of vascular sclerosis. On her second visit he found the vision in the left eye quite blurred and not being satisfied with the result of his examination he

referred her to me for consultation. There was no external evidence of inflammation. The anterior segment of the eye was normal, and tension was 18 mm of mercury. The pupil was dilated for examination of the fundus. The lens was clear and the vitreous turbid. By means of oblique focal illumination and a B B loupe a distinct elevation of the choroid and retina was seen in the inferonasal quadrant far forward near the ora serrata. The posterior aspect of the detachment could not be seen on account of the turbid vitreous. Transillumination over the detachment was dark.

The patient has been under observation by Dr. Beck and myself at weekly intervals. The tension has remained subnormal, and the vitreous is becoming less cloudy, but the detachment shows no material change. We hope to be able to report further details of this case at a later date. Whether the detachment is due to a transudate or to hemorrhage is a problem yet to be solved. In view of the clearcut history of the case, we think that a diagnosis of sarcoma can be excluded.

Dr. Cohen has had me see his patient with him on several occasions. The similarity of the picture presented in these 2 cases and the rarity of nontraumatic detachments of the choroid justify their presentation at this time.

DR. AUGUST LEO BECK, New Rochelle. The patient mentioned by Dr. Crigler is an unmarried woman 70 years of age. She was first seen in 1916, when she was 48 years old, at which time she was given a presbyopic correction. The fundi and the corrected vision were normal. She was again examined in 1919, and the glasses were changed. The fundi and the corrected vision were normal. She was examined in 1921, the results of the examination being the same as those of the preceding one. The glasses were changed. She was next examined in 1925, at which time the glasses were changed. She had a mild conjunctivitis, but otherwise the findings were the same as at the previous examination. She was examined in 1936, when she was 67 years old. The glasses were again changed. Her corrected vision was normal 20/20. There was a moderate sclerosis of both crystalline lenses, with barely perceptible visible opacification in the nucleus of the left lens. Examination of the fundi showed moderate angiopathic sclerosis in the retinas, with minute degenerative specks between the disk and the macular region of the left fundus.

The patient was next seen on Dec. 27, 1937, complaining of thread-like or filamentous specks before the left eye of several weeks' duration. There was a leash of injected conjunctival vessels on the nasal side of the left eye, which is still present. The right fundus was normal, except for the moderate angiopathic sclerosis. The left fundus was hazy, because of cloudiness of the vitreous. The vessels were visible, no hemorrhages were seen. No other abnormalities were noted, and the tension was not elevated. Potassium iodide was prescribed.

The patient was again seen on January 4, eight days after the last examination. During this interval the vision of the left eye was much more impaired. All fields were blocked out, except the lower one. No fundus reflex was present except from the extreme upper nasal field. The tension was low. A tentative diagnosis of retinal detachment was made. The patient was seen at this time in consultation with Dr. Crigler, who regarded the condition as choroidal detachment due to hemorrhage.

The Wassermann reaction was negative

The condition resembles nothing so much as an intraocular new growth, and such a diagnosis would have been made but for the foregoing observations. These, with the unvarying softness of the eye and the rapidity of involvement, make it reasonably certain that we are not dealing with a new growth.

The underlying general pathologic process in this case is not cardionephritis. According to the patient's family physician, there has never been any nephritis. There is, however, definite cardiovascular disease of some years' duration. The blood pressure has often been 180 or well over. The heart is enlarged, and on numerous occasions there has been a variation or difference in the blood pressure readings on the two arms. For example, the pressure has been read as normal on the left arm and as from 170 to 180 on the right arm on the same occasion. The patient is highstrung and overconscientious in temperament. The Wassermann reactions are negative. Chemical examination of the blood has not been done, chiefly for economic reasons.

RETINAL DETACHMENT WITH CYST FORMATION. REPORT OF A CASE. DR. WALTER P. GRIFFEY (by invitation)

This case of retinal detachment is presented because of the interesting history given and the fact that the visual acuity has remained normal in the presence of such widespread pathologic change and the associated cystic formation.

W. B., aged 31, a deckhand on board a tugboat, had no complaints referable to his eyes. During the routine physical examination he was told that the vision in the right eye was defective and was advised to obtain glasses. In the course of refraction, retinal detachment with a cystic formation was observed in the left eye, and the patient was admitted to the hospital.

The patient had enjoyed excellent health in the past, his habits had been regular. Alcohol and tobacco had been used moderately. He stated that this was the first time that he had ever been under the care of a physician. Two months before admission he recalled having been struck across the nose and the left side of the face by rope line. He continued at work. There was no change in the function of the left eye, nor were any external signs of trauma observed.

General physical examination revealed a robust man of medium stature with no abnormalities other than the condition of the eyes. Examination showed the vision in the right eye to be 20/50 which was improved to 20/20 with correction of astigmatism. The fundus was normal. Vision in the left eye was 20/20. Externally the eye was normal. On ophthalmoscopic examination floating opacities could be seen in the vitreous. A retinal detachment sharply demarcated by a zone of choroiditis extended from the disk temporally immediately below the macula to end at the periphery near 3 o'clock. On the nasal side the detachment was not so acutely limited but faded away in the upper quadrant from 10 to 12 o'clock. A translucent cyst, spherical in outline and about 3 disks in diameter was noted near the ora serrata between 7 and 8 o'clock. A second cyst, still farther forward and flat

in appearance, about 6 disks long and about 2 disks wide, was seen extending in an equatorial direction between 9 and 11 o'clock

The visual field showed an almost complete defect in the upper half

Urinalysis gave negative results. The Wassermann and Kahn reactions of the blood were negative. Repeated fecal analyses were negative for ova. The blood was found to be normal on study, and the differential counts were normal.

The problem of the etiologic basis, the treatment and the prognosis of this condition arises. Is the cyst the sole cause of the detachment? Could the trauma alleged have played a part in the cystic formation and detachment, and what outcome could be predicted on aspiration of the cyst and successful readjustment of the retina by operative means?

REPAIR OF AN OLD LACERATION AT MEDIAL END OF THE LOWER LID DR WENDELL L. HUGHES

(The plastic repair reported here was illustrated by a motion picture film)

This method of plastic repair is presented because of the frequency of the occurrence of this type of laceration across the canaliculus at the medial end of the lower lid. The method of replacement gives uniformly good results. The problems presented are the same whether the laceration is recent or old or is healed in an improper position. In a laceration of this type the torn section of lid usually retracts to give the appearance of an actual loss of tissue. However, if the end is picked up and stretched it will usually bridge the defect. In an improperly healed old laceration the incision is reopened. A tongue of tissue is prepared at the nasal end of the tarsus and pulled upward, nasally and as far backward as possible by a double-armed suture on large curved needles, which are entered nasally behind the normal attachment of the torn medial end of the canthal ligament and emerge above the canthus on the side of the nose. The sutures are tied over a small piece of gauze externally. The conjunctiva and edges of the skin are approximated with fine sutures. A pressure dressing is applied and left in place for one week.

A PRELIMINARY REPORT OF THE VALUE OF FUNCTIONAL VISUAL TESTS FOR THE LOCALIZATION OF SUPRATENTORIAL TUMORS OF THE BRAIN. DR CHARLES A. ELSBERG and DR H. SPOTNITZ (by invitation)

In order to learn whether functional visual tests are of value in the localization of tumors and other diseases of the brain, tests were made to determine the visual thresholds, the relations between area and light intensity required for threshold vision and the significance of dark adaptation and the refractory period in healthy persons and in those suffering from intracranial lesions.

The tests were made in a dark room with simple apparatus. The objects used for the visual tests consisted of white squares of different sizes on a black background and of white squares with inner black squares as objects with internal contrast. The test objects were illuminated by red light and were viewed from a distance of 3.75 meters through a red Wratten filter.

When white squares from 9 to 40 mm in size were used, the product of the cube root of the area and the threshold light intensity was found to be a constant. When squares of 40 mm with internal contrast were used, the square root of the area multiplied by the light intensity was a constant, and when objects of 9 mm were used, the area multiplied by the light intensity was also a constant. For all objects with internal contrast, the square root of the black area divided by the light intensity was a constant.

These varying relations between area and the minimum of light required for vision were found to be due to the quantitative functional relation between the cones, bipolar cells and ganglion cells of the retina. A formula was used which expressed the quantitative relations between area and light intensity for all of the test objects that were used and which showed the relative significance of excitation and of inhibition in vision.

The tests made it possible to study the changes that occur in dark adaptation and to show that the leveling of visual sensibility which occurs in passing from light to darkness is the result of the relative refractory period and the reciprocal relations between the intensity of the visual stimulus and the receptors in the retina and in the visual areas of the brain. It was found that when one eye is exposed in succession to a bright and a dim light, the duration of the refractory period is always longer than when the two eyes have been exposed in succession to a bright and a dim light and the test object is viewed with both eyes. In monocular vision after binocular stimulation by bright light the duration of the refractory period is regularly longer than in binocular vision after binocular stimulation and shorter than in monocular vision after monocular stimulation. The results of these tests showed that in so-called dark adaptation the central factor is of great importance.

By means of the tests it was found to be possible to determine the relative thresholds of vision in different parts of the fovea in healthy persons. Furthermore, in persons with diseases of the retina, and more particularly in those with papilledema and with primary atrophy of the optic nerve due to tumor of the brain, we were able to ascertain whether the central or the more peripheral parts of the fovea were more affected.

After a large number of healthy persons had been tested, patients with tumors and other lesions of the brain were studied in a similar manner. Up to the present time about 180 patients have been examined.

The functions of the two eyes were compared by means of tests of the monocular vision of each eye. The experiences thus far gained appear to indicate that in cases of tumor which cause pressure on the extracerebral visual pathways (optic nerves, chiasm and optic tracts) the threshold is raised, but the duration of the relative refractory period may be normal. When the growth is within the substance of the brain, the duration of the refractory period produced by successive exposure to a bright continuous light and to a dim light is prolonged in the contralateral eye. Therefore, it was possible by means of these functional visual tests to determine the hemisphere which was diseased. Furthermore the results of stimulation by flickering light appeared to indicate that the frontal lobes of the brain may inhibit the visual functions of the occipital and parietal lobes. The presence or absence of this

inhibition often made it possible to conclude whether or not the lesion was in the frontal lobe of one or the other side

Up to the present time our experiences appear to indicate that functional visual tests are important in the localization of diseases of the brain, but a much larger experience must be gained before the practical clinical importance of the tests can be evaluated

DISCUSSION

DR JOHN M WHEELER Dr Elsberg has a wealth of experience in the diagnosis of intracranial lesions and in surgical work on the brain and in performing autopsies After the eye has been exposed to a bright light and the person whose eye has been so exposed attempts to look at a dimly illuminated test object across the room, there is a delay in the period of recognition of that test object because of the previous exposure of the eye to light This delay the authors have called the dark adaptation time, referring to the bright illumination of the exposure of the eye as light adaptation, and they have used synonymously the terms "duration of fatigue" and "duration of recovery" and "time of adjustment for the subject to see the test object" across the room, after he has been influenced by the exposure of the eye to bright light They have capitalized on the dark adaptation phenomenon as an aid in the diagnosis of intracranial disease and particularly in the localization of intracranial tumors above the tentorium In reference to lesions of the frontal lobe, the point they have capitalized on is that a pathway exists (almost definitely established) that leads from the frontal lobe of one side to the occipital lobe of the other side The existence of this pathway has been used as an aid in the localization of these lesions

The authors have established this important fact The period of recovery from the exposure to the bright light (the dark adaptation time) is governed largely by the brain The eye has something to do with it, but, according to their findings, the brain has more to do with it, and there are several evidences of it Perhaps one of the best is that if one eye is exposed to bright light, the dark adaptation period is delayed in the other eye As there is no direct communication between the two eyes anterior to the external geniculate ganglions, the authors figure that the dark adaptation phenomenon is governed more centrally than ocularly That is rather disturbing to men who have spent large parts of their lives trying to determine the factors that influence a change from visual purple to visual red in the retina, but I think that it is well for one to think of the eye as an important part of the anterior portion of the brain and in this connection to think of the intracranial part as the governing part Dr Elsberg and Dr Spotnitz in the December 1937 issue of the *Bulletin of the Neurological Institute of New York*, refer to Dr Ames' work They stated "Ames has shown interest in errors of refraction that may result in incomplete fusion of the cortical images, and this may be the cause of a variety of disturbances" I should like to point out that incidentally Ames has been interested in refraction, because correction of refraction is a help in the relief of symptoms, but that has not been his main interest He has been interested in the difference in the size of the images in the two eyes, and he does not know—he says he does not know—where that difference is created, but he thinks that it is in the eye I should like to suggest that it never has

been proved that the difference originates in the eye. It may arise in the brain, and it may be that the method of Ames could be used in the localization of tumors of the brain. The work of Dr. Elsberg and Dr. Spotnitz has been on foveal vision and dark adaptation in relation to foveal vision. I dare say some hyper-scientific men might criticize their methods. They have gone ahead and have not been daunted by obstacles and have accomplished something in a relatively short time. Dr. Elsberg has talked to me about this work on several occasions, so I have tried to follow it.

I should like to make a suggestion. The effect of bright light on other parts of the retina might be important, and it may be that light adaptation (the exposure of the eyes to bright light) may have an important influence in bringing out defects in the visual fields. The aid of the ophthalmologist is of importance and should be of much greater importance in the diagnosing of intracranial lesions and their localization. The use of the ophthalmoscope has been important, the plottings of diplopia have been important and especially important are the plotting of the visual fields, so important that in some cases localization of a tumor of the brain has been dependent almost entirely on the defects in the visual fields. It may be that exposure to light as a preliminary to taking the visual fields may be helpful. That is just a suggestion, and I should like Dr. Elsberg to tell, if he will, what part the ophthalmologist is liable to play in carrying out of the ideas that he and Dr. Spotnitz are establishing in relation to the diagnosis of tumors of the brain, and whether he feels, as I do, that the effect of exposure to bright light may have an important influence on the visual fields, so that one may be led to diagnose earlier defects in the visual fields.

DR. CHARLES A. ELSBERG. What Dr. Wheeler has said regarding Dr. Ames' investigation is, I think, of great importance, because, as Dr. Wheeler mentioned, Ames' work had reference to the difference in size of the images. In binocular vision, images must be fused. The only place where they can be fused is centrally, and therefore the fact of Ames' investigation is another indication that the essential process in fusion and dark adaptation must be central. It is tremendously interesting that Dr. Wheeler should have picked up something that Dr. Spotnitz found in testing persons (with tumor of the temporal lobe, I think) who had no gross defects of the visual fields, that is, no defects that could be determined by perimetric examination. As the patients recovered after exposure to the bright light they saw the test object first on the good side and not on what should have been the side with the hemianopic defect. Exactly what Dr. Wheeler predicted might happen has happened in a few isolated instances. It may be of value in cases in which there is a possibility of a hemianopic defect in the visual field, first to expose the subject to a brighter light and then to ascertain the visual fields. I think that it is probable that this procedure which Dr. Wheeler has suggested and which has actually been tried in a few instances, will be of advantage to the ophthalmologist in the localization of lesions of the brain in examinations of vision. The method of examination and the apparatus to be used are both simple.

Book Reviews

Eleventh Annual Report of the Memorial Ophthalmic Laboratory Giza, Cairo, Egypt Schindler's Press, 1937

The eleventh annual report of the Memorial Ophthalmic Laboratory presents a record of the administration and an account of the scientific work carried out during 1936. The activities of the laboratory are considered as follows:

1 Postgraduate education. Since the laboratory was opened ten years ago, one of its functions has been to provide a postgraduate course of instruction in ophthalmology for all doctors entering the ophthalmic service of the Ministry of Public Health. The teaching is essentially practical, and special attention is devoted to those features of ophthalmic practice peculiar to Egypt.

2 Pathologic section. Five hundred and fifty-four pathologic specimens were submitted for examination. Of the 125 globes included, 84, or 64 per cent, had been destroyed through acute ophthalmias. This is 13 per cent less than the figure for ten years ago, and the reduction is believed to be a result of the activity of the government ophthalmic hospitals.

A total of 22 specimens are described in detail and illustrated with photomicrographs. Of outstanding interest is a case of bilharzial granuloma, the third seen at the laboratory. In each instance the patient was a young child with clinical symptoms strongly resembling infection of the conjunctiva with *Streptothrix*. Other interesting specimens include one of early sarcoma of the bulbar conjunctiva, one of cartilage in the conjunctiva, unassociated with dermoid tumor, and one of "hyaline" bodies in the caruncle.

3 Clinical section. The clinical material studied at the laboratory appears to have been unusually varied. Five cases of special interest are reported in full, and the colored drawings which accompany 4 of them clearly illustrate the special features which they represent. These include a case of pemphigoid infection of the conjunctiva, a case of spring catarrh associated with epithelial dystrophy of the cornea, a case of xanthomatosis bulbi associated with cerebral signs, a case in which interesting macular changes occurred in association with cilioretinal anastomosis and a case of tumor of the pituitary gland with typical changes in the visual fields and unusual neurologic signs. A number of other interesting cases, including 1 of fly-blown orbit and 1 of infection of the conjunctiva with *Streptothrix*, are illustrated by photographs.

4 Research section. As in former years, the cause of trachoma and the epidemicity of the acute ophthalmias have been problems for special attention. This year's work also included a study on spring catarrh.

The studies on trachoma include a "Note on Free Initial Bodies and Free Elementary Granules in Trachoma of Egypt, Madras and Hong Kong" by F. H. Stewart, the pathologist of the laboratory. Up to the end of 1935 free initial bodies (Lindner) have not been found by

Stewart in Egypt, nor were free elementary bodies found in sufficient number to allow of certain identification. During 1936 he studied 37 cases of early trachoma in infants all under 1 year of age, free initial bodies were found in 7 and free elementary bodies in 3.

A second study on the inclusion bodies of trachoma is reported by R. P. Wilson, director of the laboratory. At Bahtim Village, near Cairo, he was able to examine 39 infants for inclusion bodies at weekly intervals from birth. From his results he concludes that inclusion bodies are present in 100 per cent of the patients at the onset of the disease and that they may be found before trachoma can be diagnosed clinically.

Wilson's observations on the acute ophthalmias have been continued, both at Giza and in other parts of Egypt. His findings indicate that conjunctivitis due to the Koch-Weeks bacillus assumes epidemic proportions when the mean maximum temperature reaches approximately 25 or 26 C (77 or 78.8 F), while gonococcal conjunctivitis becomes epidemic only at temperatures from 32 to 35 C (89.6 to 95 F). The two principal fly-breeding seasons in Egypt are spring and autumn, during the hottest months of the summer the incidence of flies is reduced. Koch-Weeks conjunctivitis becomes epidemic soon after the flies begin to increase and reaches a maximum coincidentally with the height of the spring fly-breeding season, gonorrheal conjunctivitis, on the other hand, does not increase in incidence during the first fly-breeding season until the temperature reaches approximately 32 C. The summer minimum of Koch-Weeks conjunctivitis and the August decline in gonorrheal conjunctivitis coincide with the summer minimum incidence of flies. The maximum incidence of gonorrheal conjunctivitis and the height of the autumnal rise in Koch-Weeks conjunctivitis coincide with the second fly-breeding season. Wilson has therefore concluded that although factors of direct contagion by hand, clothing, etc., cannot be neglected, heat and flies are the two most vital factors in the epidemicity of conjunctivitis in Egypt.

An extensive report by F. M. Lyons entitled "Observations on the Pathogenesis of Spring Catarrh" is of distinct interest. Lyons calls particular attention to the characteristic sticky palpebral membrane in the disease, for its diagnostic importance and for the light it throws on the nature of the disease. In cases of mild involvement the membrane is not always apparent on evertng the lid but may be readily produced by various forms of irritation. Anything which gives rise to congestion and dilatation of the capillaries favors this exudation, which is accompanied by intense itching. Conversely, the formation of the membrane may be inhibited by any procedure which causes constriction of the capillaries, such as the application of ice or strong solutions of epinephrine hydrochloride. Freshly teased preparations and paraffin sections of the membrane were examined microscopically and found to consist of fibrin, mucin, some epithelial cells and considerable numbers of eosinophils. Lyons therefore concludes that the membrane is composed of two parts: (a) the true exudation of fibrin and blood cells, which must necessarily come from the subconjunctival capillaries, and (b) the mucin, a hypersecretion of the conjunctival goblet cells. He considers that the usual conception of the pathologic picture of spring catarrh is erroneous and that the lesions are due, not, as formerly

thought, to proliferation of preexisting connective tissue but rather to the invasion of the conjunctiva by new fibinous material and wandering cells. He concludes that the whole picture of spring catarrh results from an abnormal exudation of fibrin and blood cells from the capillaries of the conjunctiva, and his results indicate that a faulty calcium metabolism is not a primary cause of this capillary dysfunction.

The eleventh annual report meets the standard set by previous numbers and constitutes a most valuable addition to the ophthalmic literature.

PHILLIPS THYGESON

Citric Acid Studies Referring to the Eye By Herman Gronvall. Paper Pp 279, with 50 tables. Copenhagen. Levin & Munksgaard, 1937.

This monograph, published as the fourteenth supplement of the *Acta Ophthalmologica*, is concerned chiefly with the occurrence of citric acid in the intraocular fluids. It begins with a discussion of the application and limitations of the Thunberg enzymatic method for the determination of citric acid. The different qualitative tests which confirm the presence of the acid in ocular fluids and tissues are reported. The quantity of citrate in the intraocular fluids and serums of various mammals, birds and fish, of bovine fetuses and also of patients suffering from different ocular diseases is recorded. It also deals with the relation of blood, tears, corneal permeability and retinal dehydrogenases to the metabolism of citric acid.

Citric acid is found in intraocular fluid in all mammals, birds and fish, although the amount in birds is higher than in other animals. Unlike dextrose, citric acid is evenly distributed throughout the vitreous humor. In fetal life the citrate content of the intraocular humors slowly declines until birth. After intravenous injection of citrate, the amount in the aqueous humor rises and falls with that of the blood serum. The citrate in the serum seems to be linked chemically or physically with protein. In cases of iridocyclitis, glaucoma and melanoma of the choroid and after paracentesis of the anterior chamber the aqueous humor contains an abnormally large quantity of citric acid. There is no quantitative difference between the aqueous humor of cataractous and that of aphakic eyes. If the eye is kept in the dark, the amount of acid in the aqueous and vitreous humor diminishes.

The cornea shows an irreciprocal permeability to citrate which is dependent on the integrity of the epithelium. The corneal endothelium retards the diffusion of the salt less than does the epithelium.

Citric acid seems to be an intermediate product in metabolism of the ocular tissues. In the retina a dehydrogenase plays a part in this metabolism.

This well written monograph is the result of a carefully planned study of a special phase of ocular metabolism. More work of this kind is needed in ophthalmology.

ARLINGTON C. KRAUSE

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Place H B A Free Ophthalmic Hospital, Parel, Bombay 12 Time First
Friday of every month

* Secretaries of societies are requested to furnish the information necessary
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 Place Massachusetts Eye and Ear Infirmary, 243 Charles St, Boston Time.
 8 p m, third Tuesday of each month from November to April, inclusive

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 Secretary-Treasurer Dr C Allen Dickey, 450 Sutter St, San Francisco
 Place San Francisco Time June 19-22, 1939

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 Secretary-Treasurer Dr Purman Dorman, 1115 Terry Ave, Seattle
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 June, July and August

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 Place Saginaw or Bay City, Mich Time Second Tuesday of each month,
 except July and August

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Secretary-Treasurer Dr B M Merkel, 604 Locust St, Des Moines

Place Davenport

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Secretary-Treasurer Dr Edley H Jones, 1301 Washington St, Vicksburg, Miss

Place Gulfport, Miss Time May 8, 1939

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OTOLOGY AND RHINOLARYNGOLOGY

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Place Atlantic City Time June 1939

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 Secretary-Treasurer Dr Frank C Smith, 106 W 7th St, Charlotte
 Place Charlotte Time October

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 Secretary-Treasurer Dr F L Wicks, 516-6th St, Valley City
 Place Fargo Time May 1939

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 Secretary-Treasurer Dr Andrew J Browning, 418 Mayer Bldg, Portland
 Place Good Samaritan Hospital, Portland Time Third Tuesday of each month

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 Secretary-Treasurer Dr Linley C Happ, 124 Waterman St, Providence
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 second Thursday in October, December, February and April

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 Secretary Dr J W Jervey Jr, 101 Church St, Greenville
 Place Columbia Time Nov 1, 1938

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 Secretary-Treasurer Dr W D Stinson, 805 Medical Arts Bldg, Memphis

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 Secretary Dr O M Marchman, 1719 Pacific Ave, Dallas

UTAH OPHTHALMOLOGICAL SOCIETY

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 Secretary-Treasurer Dr E B Fairbanks, Boston Bldg, Salt Lake City
 Time Third Monday of each month

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 Secretary-Treasurer Dr M H Williams, 30½ Franklin Rd, S W, Roanoke

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AND THROAT SECTION

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LOCAL

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 Secretary Dr William F McKim, 317 Roseville Ave Newark
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 each month October to May

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 Secretary-Treasurer Dr C R Anderson, 106 S Main St, Akron
 Time First Monday in January, March, May and November

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 Secretary Dr Alton V Hallum, 478 Peachtree St, Atlanta, Ga
 Place Academy of Medicine, 38 Prescott St Time Second Friday of each month from October to May

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 Secretary Dr Fred M Reese, 6 E Eager St, Baltimore
 Place Medical and Chirurgical Faculty, 1211 Cathedral St Time 8 30 p m, fourth Thursday of each month from October to May

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 Secretary-Treasurer Dr Frank Mallon, 1135 Park Pl, Brooklyn
 Place Kings County Medical Society Bldg, 1313 Bedford Ave Time Third Thursday in February, April, May, October and December

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 Secretary-Treasurer Dr Meyer H Riwchun, 367 Linwood Ave, Buffalo
 Time Second Thursday of each month

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

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 Secretary Dr A H Benz, 706 Medical Arts Bldg, Chattanooga
 Place Mountain City Club Time Second Thursday of each month from September to May

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President Dr Georgiana Dvorak-Theobald, 715 Lake St, Oak Park
 Secretary-Treasurer Dr Earle B Fowler, 55 E Washington St, Chicago
 Place Medinah Michigan Avenue Club, 505 N Michigan Ave Time Third Monday of each month from October to May

CINCINNATI OPHTHALMIC CLUB

Chairman Each member, in rotation
 Secretary-Treasurer Dr E R Thomas, 819 Carew Tower, Cincinnati
 Place Holmes Memorial Library, Cincinnati General Hospital Time 8 15 p m, third Monday of each month except June, July and August

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 Secretary Dr Fred Dixon, 2060 E 9th St, Cleveland

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman Dr Paul Moore, Republic Bldg, Cleveland
 Secretary Dr G Leslie Miller, 14805 Detroit Ave, Cleveland
 Time Second Tuesday in October, December, February and April

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

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 Clerk Dr W S Reese, 1901 Walnut St, Philadelphia
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 Secretary-Treasurer Dr W A Stoutenborough, 21 E State St, Columbus, Ohio
 Place Deshler Wallick Hotel Time 6 p m, first Monday of each month

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman Dr Edgar G Mathis, 416 Chaparral St, Corpus Christi, Texas
 Secretary Dr E King Gill, 416 Chaparral St, Corpus Christi, Texas
 Time Second Thursday of each month from October to May

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President Dr Lester H Quinn, 4105 Live Oak, Dallas, Texas
 Secretary Dr J Dudley Singleton, 1719 Pacific Ave, Dallas, Texas
 Place Dallas Athletic Club Time 6 30 p m, first Tuesday of each month
 from October to June The November, January and March meetings are
 devoted to clinical work

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President Dr E G Linn, 604 Locust St, Des Moines, Iowa
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 Iowa
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 Time 6 30 p m, first Wednesday of each month

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

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 Secretary-Treasurer Dr Joseph L Holohan, 330 State St, Albany
 Time Third Wednesday in October, November, March, April, May and June

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President Dr R A Gough, 602 W 10th St, Fort Worth, Texas
 Secretary-Treasurer Dr Charles R Lees, 806 Medical Arts Bldg, Fort Worth,
 Texas
 Place Medical Hall, Medical Arts Bldg Time 7 30 p m, first Friday of each
 month except July and August

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President Dr Dewey R Heetderks, 405 Medical Arts Bldg, Grand Rapids, Mich
 Secretary-Treasurer Dr Robert G Laird, 116 E Fulton St, Grand Rapids, Mich
 Place Various local hospitals Time Third Thursday of alternating months,
 September to May.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND
 THROAT SECTION

President Dr Louis Dahl, 1215 Walker Ave Houston, Texas
 Secretary Dr Herbert H Harris, 1004 Medical Arts Bldg, Houston, Texas
 Place Medical Arts Bldg, Harris County Medical Society Rooms Time
 8 p m, second Thursday of each month from September to June

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President Dr J K Leasure, 23 E Ohio St, Indianapolis
 Secretary Dr Kenneth L Craft, 23 E Ohio St, Indianapolis
 Place University Club Time 6 30 p m, second Thursday of each month
 from October to June

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

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 Secretary Dr John S Knight, 1103 Grand Ave, Kansas City, Mo
 Time Third Thursday of each month from October to June The November,
 January and March meetings are devoted to clinical work

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 Secretary-Treasurer Dr Paul Nilsson, 211 Cherry Ave, Long Beach, Calif
 Place Professional Bldg Time Last Wednesday of each month from October
 to May

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President Dr Clifford B Walker, 427 W 5th St, Los Angeles
 Secretary-Treasurer Dr John P Lordan, 2007 Wilshire Blvd, Los Angeles
 Place Los Angeles County Medical Association Bldg, 1925 Wilshire Blvd Time
 6 30 p m, fourth Monday of each month from September to May, inclusive

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President Dr Gaylord C Hall, Brown Bldg, Louisville, Ky
 Secretary-Treasurer Dr Charles K Beck, Starks Bldg, Louisville, Ky
 Place Brown Hotel Time 6 30 p m, second Thursday of each month from
 September to May, inclusive

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OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman Dr Earle Breeding, 1801 I St, N W, Washington
 Secretary Dr Elmer Shepherd, 1606-20th St, N W, Washington
 Place 1718 M St, N W Time 8 p m, third Friday of each month from
 October to April, inclusive

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman Each member, in alphabetical order
 Secretary Dr Sam H Sonders, Medical Arts Bldg, Memphis, Tenn
 Place Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital Time
 8 p m, second Tuesday of each month

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President Dr John E Mulsow, 231 W Wisconsin Ave, Milwaukee
 Secretary-Treasurer Dr John B Hitz, 411 E Mason St, Milwaukee
 Place University Club Time 6 30 p m, second Tuesday of each month

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman Each member, in alphabetical order
 Secretary Dr M C Pfunder, 645 Medical Arts Bldg, Minneapolis
 Place Hennepin County Medical Society rooms Time 6 30 p m, fourth
 Monday of each month, October to May, inclusive

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 Secretary-Treasurer Dr Maitland D Place, 981 Reibold Bldg, Dayton, Ohio
 Place Van Cleve Hotel Time 6 30 p m, monthly, first Tuesday from October
 to June, inclusive

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President Dr L de G Joubert, 690 Dunlop St, Montreal, Canada
 Secretary Dr K B Johnston 1509 Sherbrooke St, W, Montreal, Canada
 Time Second Thursday of October, December, February and April

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Chairman Dr Guy Maness, 119-7th Ave, Nashville, Tenn
 Secretary-Treasurer Dr Andrew Hollabaugh, Doctors Bldg, Nashville, Tenn
 Place St Thomas Hospital Time 8 p m, third Monday of each month from
 October to May

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 Secretary-Treasurer Dr H F Brewster, 837 Gravier St, New Orleans
 Place Eye, Ear, Nose and Throat Hospital Time Third Thursday of each
 month from October to June

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

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 Secretary Dr Rudolf Aebli, 30 E 40th St, New York
 Time 8 30 p m, third Monday of every month from October to May, inclusive

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President Dr Percy Fridenberg, 38 W 59th St, New York
 Secretary Dr David Alperin, 889 Park Place, Brooklyn
 Place Squibb Hall, 745-5th Ave Time 8 p m, first Monday of each month
 from October to May, inclusive

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 OTO-LARYNGOLOGICAL SOCIETY

President Dr Philip Romonek, 107 S 17th St, Omaha
 Secretary-Treasurer Dr W Howard Morrison, 1500 Medical Arts Bldg, Omaha
 Place Omaha Club, 20th and Douglas Sts, Omaha Time 6 p m, dinner,
 7 p m, program, third Wednesday of each month from October to May

OPHTHALMOLOGICAL SOCIETY OF THE UNIVERSITY OF PITTSBURGH

President Dr W W Blair, 121 University Pl, Pittsburgh
 Secretary Dr George H Shuman, 351-5th Ave, Pittsburgh
 Time Second Monday in November, January, March and May

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President Dr R N Berke, 430 Union St, Hackensack, N J
 Secretary-Treasurer Dr T A Sanfacon, 340 Park Ave, Paterson, N J
 Place Paterson Eye and Ear Infirmary Time 9 p m, last Friday of every
 month, except June, July and August

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 Time First Thursday of each month from October to May

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 Secretary Dr George H Shuman, 351-5th Ave, Pittsburgh
 Place Pittsburgh Academy of Medicine Bldg Time Fourth Monday of each
 month, except June, July, August and September

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 Secretary Dr Richard W Vaughan, Medical Arts Bldg, Richmond, Va
 Place Westmoreland Club Time 6 p m, second Monday of each month from
 October to May

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President Dr R E Elliott, 78 S Fitzhugh St, Rochester, N Y
 Secretary-Treasurer Dr Raphael Farber, 280 Monroe Ave, Rochester, N Y
 Place Rochester Medical Association, 113 Prince St Time 8 p m, third
 Monday of each month from October to May

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 Secretary Dr Leslie Charles Drews, 508 N Grand Blvd, St Louis
 Place Oscar Johnson Institute Time Clinical meeting 5 30 p m, dinner and
 scientific meeting 6 30 p m, fourth Friday of each month from October to
 April, inclusive, except December

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 Secretary-Treasurer Dr Wilfred E Muldoon, 414 Navarro St, San Antonio,
 Texas
 Place Bexar County Medical Library Time 8 p m, first Tuesday of each
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EAR, NOSE AND THROAT

Chairman Dr Russell Fletcher, 490 Post St, San Francisco
 Secretary Dr Avery Morley Hicks, 490 Post St, San Francisco
 Place Society's Bldg, 2180 Washington St, San Francisco Time Fourth
 Tuesday of every month except May, June, July and December

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 Place Shreveport Charity Hospital Time 7 30 p m, first Monday of every
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President Dr P B Greene, 422 Riverside Ave, Spokane, Wash
 Secretary Dr O M Rott, 421 Riverside Ave, Spokane, Wash
 Place Paulsen Medical and Dental Library Time 8 p m, fourth Tuesday of
 each month except June, July and August

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 and August

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 Secretary Dr W R F Luke, Medical Arts Bldg, Toronto, Canada
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 D C
 Secretary-Treasurer Dr Frank D Costenbader, 1726 I St, Washington, D C
 Place Episcopal Eye, Ear and Throat Hospital Time 8 p m, first Monday
 in November, January, March and May

ARCHIVES OF OPHTHALMOLOGY

VOLUME 20 (old series Volume 77)

OCTOBER 1938

NUMBER 4

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PHYSIOLOGIC AND CLINICAL OPHTHALMOLOGIC PROBLEMS IN RELATION TO INDIVIDUAL VARIABILITY

A BRUCKNER, M D

Professor, Director of Ophthalmologic Clinic of University of Basel

BASEL, SWITZERLAND

Introduction

Problems of color vision

Dependence of color sensation on stimulus

Congenital color deficiencies

Partial color blindness

Anomalous trichromats

Diagnosis

Frequency of different types of color deficiency

Heredity of color deficiency

Total color blindness

Development of color sense

Phenomena of local adaptation

Phenomena of revulsive local adaptation

Black sensation

Importance of contrast

Local differences in adaptation

Measurement of the phenomena of local adaptation

Phenomenon of constancy

Seat of local adaptation

Light and dark adaptation

Diseases accompanied by disturbances of dark adaptation

Clinical methods

Focal illumination

Tonometry

Sensitiveness of the cornea

This article comprises lectures delivered in the Eye Department (Tennent Foundation) of the University of Glasgow in April 1936. General questions concerning light and color sense were treated. Special attention was paid to German literature, it was taken for granted that English authors were well known to the listeners, and they were therefore only mentioned in passing. Similarly, in this printed version of the lectures the citations are restricted to the most essential. Dr. Arnold Knapp, one of the editors of the ARCHIVES, suggested that the lectures be printed in this journal, which was not originally contemplated. Since the hearers in Glasgow were mostly practicing ophthalmologists, some principles which are well known to psychologists are treated rather fully.

Diseases of the eye dependent on constitution and on climate

Tuberculous diseases of the eye and general constitution

Scrofula of the eye

Diseases of the eye in relation to weather and climate

Hordeolum

Catarrhal ulcer of the cornea

Acute conjunctivitis

Herpes corneae

Scrofula of the eye

Acute glaucoma

Summary

INTRODUCTION

If a person goes from a brightly illuminated room into a dark one, he sees nothing at first. After a short time he becomes adapted to the darkness and is able to find his way about. If he returns to the brightly lit room, he is dazzled and all appears much brighter than before his stay in the dark room. Actually no change has occurred in the intensity of the illumination. The change of the impression is therefore related to a change in the human organism causing one to react in a different manner to the same surrounding circumstances. This phenomenon is well known. It is only one especially striking example of the relation between the living organism and its environment.

The ordinary man, starting from his own experience and with what is called the constancy of his own ego, naturally thinks that differences in his sensations to the same external influences are due to changes in these and not in himself. But, as already noted, it is often his own organism which does not react in the same manner to the same external influences. Therefore, in the case of different physical or psychic events one must always consider to what extent they are based on the environment and to what extent on the subject himself. The ancient philosopher Democritus expressed this idea in the following sentence: "In reality we perceive nothing certainly or infallibly but only impressions, which change according to the momentary condition of the body."

The distinction between environment and the inner perception of the organism—*die Umwelt und die Innenwelt*—has been especially clearly defined by von Uexküll¹. He convincingly showed that each person has his own environment, therefore, there is nothing absolute in the surroundings, in the sense of a constancy which is the same for all persons.

A nice exposition of this thought was given by the Baltic scholar Karl Ernst von Baer² about seventy years ago. He discussed how

1 von Uexküll, J. J. *Umwelt und Innenwelt der Tiere*, Berlin, Julius Springer, 1921.

2 von Baer, K. E. *Reden gehalten in wissenschaftlichen Versammlungen und kleinere Aufsätze vermischten Inhalts*, ed. 2, Braunschweig, F. Vieweg & Sohn, p. 254.

dependent on the duration of the life of the individual organism the view of the world would appear provided the organism had human understanding. A transient being, living but for a day, will conceive the day as happening but once with the rising of the sun on one side and its setting on the other side of the horizon, the longer-lived organism recognizes the rhythmic repetitions of the event and gives it another explanation.

The size of the organism is another aspect of environment which causes variations in visual concepts. One need not consider different species of animals. For when human beings of varying stature, children or adults, observe objects around them, the fact that the head is at a different height from the ground at varying stages of growth is sufficient reason for their receiving different images of the world. As a man grows there is a change in the zero of his system of coordinates, which zero, according to F. B. Hofmann,³ is situated in the articulation between the skull and the uppermost vertebra. This articulation is of the utmost importance for orientation in space. Its displacement, important as it is, escapes one's notice owing to the slowness of man's growth. Here again, there is no constancy of environment for the individual. This is true not only under normal conditions but with the pathologic reactions of the organism.

The fact that individuals consider the environment constant is a biologically important equalizing function of the whole constitution which first allows life in a fuller sense. A change of a primitive immediate reaction is followed by a correction in the organism itself, but one which often remains unperceived. The final behavior of the organism is the result of the interaction of these two processes.

However, this must not lead one to believe that the primary experience is unchanged when the same conditions are encountered at different times. It is a peculiarity of the living being that it perhaps never gives the same reaction to the same surrounding conditions twice because the organism does not remain the same. Ewald Hering⁴ mentioned this in one of his excellent lectures. He imputed to the living organism a kind of remembrance of all events which had occurred to it, not only those of the conscious mind but those of the unconscious mind.

If the human being is always changing his reactions to the same condition, it will readily be seen, from the extreme differences between human beings—even though one considers only these—how vastly different the biologic responses must be. This is true not only of physio-

³ Hofmann, F. B. *Raumsinn*, in von Graefe, A., and Saemisch, E. T. *Handbuch der gesamten Augenheilkunde*, ed. 2, Leipzig, Wilhelm Engelmann, 1901-1911, vol. 3, chap. 13, p. 393.

⁴ Hering, E. *Ueber das Gedächtnis als eine allgemeine Funktion der organischen Materie* 1870, in Hering, H. E. *Fünf Reden von Ewald Hering* Leipzig, Wilhelm Engelmann, 1921.

logic, but more especially of psychologic, response. How differently, for instance, do persons respond to esthetic impression! How changeable is the susceptibility to infections! These differences are in the last analysis based on the constitution as determined by heredity. This multiplicity of reaction presents some difficulty in every science. It is encountered in both the mental and the so-called exact natural sciences. Medical science may take a place between these two groups.

I shall discuss the multiplicity of reactions of organisms to their surroundings on the basis of normal functions and pathologic processes of the eye. The somewhat complicated matter will compel me to be theoretic and will lead to questions somewhat away from my special theme.

PROBLEMS OF COLOR VISION

I shall first consider some problems of physiologic optics.

In the sphere of light and color sense it is necessary to distinguish clearly between the objective and the subjective, or between the outer world and sensation. While Goethe according to his philosophy believed in an almost mystic unity of the light and the eye, today it is impossible to accept his view, although a higher unity in the sense of von Uexküll may be admitted.

According to the angle from which one approaches the problem, one will take as one's starting point either the physical processes or those of sensation. During the past fifty years there has been a dispute (now apparently ended) in the realm of physiologic optics as to the starting point. On the one side were the scholars, preeminently physicists, who sought to discover the relations between stimulus and sensation by means of physical measurements. Newton may be called the pioneer in this direction, Young followed in 1800 and Maxwell sixty years later. The principal representatives of this group in Germany were Helmholtz, König and von Kries. These scientists considered the physical data, namely, wavelength, intensity, mixture of defined lights and so on, the basis for scientific investigation. In opposition to this opinion, Ewald Hering, partly following the ideas of Ernst Mach, emphasized the necessity of starting not from physical events but from immediately given sensations. These are the primary principles. By critical judgment of sensations and determination of the various conditions under which a definite sensation is perceived, one may, according to Hering,⁵ draw certain conclusions as to the underlying physiologic events. Armin Tschermak⁶ designated this kind of research, with

5 Hering. Grundzüge der Lehre vom Lichtsinne, in von Graefe, A., and Saemisch, E. T. Handbuch der gesamten Augenheilkunde, ed 2, Berlin, Julius Springer, 1921, vol 3.

6 Tschermak, A. Der exakte Subjektivismus in der neueren Sinnesphysiologie, Arch f d ges Physiol 188 1, 1921.

an antithetic expression, exact subjectivism. A similar point of view has been taken by some psychologists and ophthalmologists, for example Georg Elias Müller, Hillebrand and Hess.

These two schools, while strongly opposing each other about forty years ago, made many contributions of the greatest importance to the knowledge of this subject. It seems to me that Hering's starting point makes it easier to understand the complicated matter in relation not only to the sense of vision but to the physiologic basis of all sensation. It may be emphasized that by this method a great deal is not immediately apparent but needs critical consideration to permit arrival at what was Hering's primary object, the delineation of perception by experiment.

Those of the other school emphasized that it was impossible to reproduce one person's experience in another. This was one of their chief reasons for starting from the physical conditions, since subjective sensations cannot be the foundation of knowledge. But herein lies the general problem of all mutual understanding and exchange of thought. Scientific research in general is possible only on the supposition of the existence of a psychologic organization of the same kind, although this supposition is not true. Even the physical study of physiologic optics is established on this supposition.

To make research in the psychology of sensations possible it is first necessary to determine the equality or the inequality of two sensations. This is done in the fundamental psychophysiologic investigations of Fechner.⁷ One judges simultaneously or successively two sensations, however called forth, with regard to their equality or inequality. If one wishes to learn something about the connection between a person's psychic behavior and the outer world, it is necessary to determine how the psychophysical substance answers to an exactly determined physical stimulus. This determination itself is, of course, only indirectly possible through sensations and perceptions.

The chief problem is: What is the functional relation in a mathematical sense between stimulus and sensation?

On the one hand, the more physically minded maintained it sufficient to define exactly and to reproduce the physical stimulus. For the psychophysical substance will always react in the same manner, provided the excitation is always nearly the same. The differences occasionally observed were considered to be accidental (von Kries⁸). On the other hand, the scholars starting from the psychophysical side, especially

⁷ Fechner, G. T. *Elemente der Psychophysik*, ed. 3, Leipzig, Breitkopf & Hartel, 1907.

⁸ von Kries, J. A. *Allgemeine Sinnesphysiologie*, Leipzig, F. C. W. Vogel, 1923.

Hering, emphasized the changes in the excitability of the living substance. Therefore, they did not in all cases exactly define the physical stimulus used in their experiments, for in view of the changes in the excitability they did not believe this to be of great importance.

Because of these two points of view differences in the results of research were bound to arise. The cause was often not clear and naturally led to violent disputes, since it was a question of sensations experienced by one person only. In determining experimentally the relation between stimulus and sensation, a single observation is of no value, the experiment must be repeated many times. The reconstruction of exactly the same conditions in relation to the physical stimulus is not too difficult, but special precautions are necessary to reproduce the same condition of the nervous substance. Strictly speaking, this is not always possible, because not only the change in the nervous system but repetition and recollection of former occurrences are important. Even if only a single observation is made, the manner of reaction is largely dependent on the immediately preceding excitation.

Therefore, using a simple expression for complicated things, one must consider the factor of time in all experiments dealing with the function of the senses.⁹

Since the results for different individuals seem to be within certain definite limits, these may be determined by the conformity of the decisive factors, namely, time, adaptation, psychologic condition and judgment. Further, the deviations may be equalized and a mean value obtained by the method of limits of error.

During the first investigations in physiologic optics, up to about the first decade of this century, it was usual to formulate general laws based on the experiments of only one person or of a few persons. But the previously mentioned point of view makes it necessary to use methods whereby a great number of persons may be submitted to the experiments. Thus the American literature, for instance, reports researches into physiologic optics which are founded on the investigations of many observers. To be sure, this condition can be fulfilled only when the problem is simple. When experiments require much time or are very complicated, as in the so-called gaging of the spectrum, they must still be limited to the investigations of a few specially trained and skilled observers.

⁹ That ophthalmologists do not sufficiently observe this point becomes clear when one considers the clinical method for examining the reactions of the pupil. The duration of the stimulus of light has hitherto been considered only by E. L. K. Zeldenrust (*Ueber die Chronaxie des Lichtreflexes der Pupille*, *Arch. f. Augenh.* **104** 585, 1931). Analogous considerations as to the reaction of the pupil in accommodation or convergence have never been made.

Only when the demand for a large number of persons for experimentation is fulfilled will it be known whether, besides the hitherto known types of light and color sense, there are other as yet unknown forms. Some investigations made in recent times seem to prove that such congenital variants occur, owing perhaps to hereditary differences. Generally, such differences are present not only in the nervous substance but in the physical makeup of the eye. For instance, Holm¹⁰ and Kravkov¹¹ found differences in the pigmentation of the macula which necessarily cause differences in the equation of spectral lights.

With increasing age the lens becomes more yellow and therefore the longer wavelengths predominate over the shorter ones in their action on the substance of the visual sense. If the crystalline lens is dark brown, as in the so-called cataracta nigra, there will be no perception of blue and violet. This is the so-called blue blindness, due to the yellow coloring of the lens. Hess¹² has suggested that the very intense blues found in the pictures painted by Titian in his old age—he was painting at the age of 90—are due to his not seeing blue well enough. In order to produce the necessary degree of blue in his pictures for himself, he was forced to apply this color heavily.

Variations in the rate of adaptation of the eye to low illumination are due, in addition to differences in the visual substance, to physical factors, such as differences in the width of the pupil, which becomes smaller with increasing age. Even in persons of the same age the size of the pupil varies according to whether the sympathetic or the parasympathetic system predominates in the innervation of the iris. Thus the quantity of light entering the eye will vary, and, as a result, eyes show differences in their adaptation to darkness.

I shall first consider the sensation of color as the sole psychic matter. There are colored and uncolored sensations, called in German *bunte und unbunte Farbenempfindungen*. By the expression uncolored is meant all shades of white, gray and black, by the term colored, the totality of the other colors. The German word *bunt*, "colored" in a narrower sense, was first used by Hering⁵. Other authors expressed the opinion that "*bunt*" signifies not a single color but a collection of different colors. This difference in the terminology is in my opinion of no importance, provided one knows what is meant. This expression is clearer than the division of the colors into those with hue and those without hue *tonfreie und tonhaltige Farben*. I shall use the words colored and uncolored when distinguishing between the

10 Holm, E. Das gelbe Maculapigment und seine optische Bedeutung, Arch f Ophth **108** 1, 1922

11 Kravkov, S. W. Ueber die Lichtabsorption der gelben Farbe der Maculagegend, Arch f d ges Physiol **210** 781, 1925

12 Hess, C. Ueber Blaublichtheit durch Gelbfärbung der Linse, Arch f Augenh **61** 29, 1908

color of the white, gray and black set and the others, although the expression uncolored color sensation is a contradiction

A psychologic analysis shows that four colors arise from the large number of possible color sensations—a pure red, a pure green, a pure yellow and a pure blue. These four principal colors in a psychologic sense—*Urfarben* oder *Grundfarben*, according to Hering—have always only a single color quality. In contrast thereto, the other colors contain two principal colors, for instance orange (yellow and red) and violet (blue and red). These colors lie between the principal colors, because they suggest two of them. These are called transitional colors—*Zwischen-* oder *Uebergangsfarben*.

It is of importance to note that there is no color sensation in which are seen red *and* green or yellow *and* blue. Therefore, these colors are named by Hering opponent colors—*Gegenfarben*.

There is no doubt about the character of the pure blue and the pure yellow. But that is not true of the pure green and the pure red, or *Urgrün* und *Urrot*. Therefore, many mistakes and unnecessary disputes have arisen in the literature. Many authors, for instance Ladd-Franklin,¹³ identified Hering's pure red as that which is produced by the longest wavelength of the spectrum. But this has always a slight yellowish tinge. In the spectrum pure red containing neither blue nor yellow does not exist. The pure red can be produced only by a mixture of a light of short waves and long waves from the spectrum—for instance, violet and red. Usually such colors are called purple. The same is true of the pure green. In nature is seen the green of leaves and grass. But this green contains a little yellow. One is therefore inclined to accept this as the pure green and to consider the pure green, which is neither yellowish nor bluish, as a bluish green.

Color sensations in a special sense—*die bunten Farben*—may be symbolically arranged in a circle in which yellow and blue, and red and green are situated at the opposite ends of two diameters drawn at right angles to one another. One can imagine that the colors in the circle are continuously changed. Red passes with the increase of the yellow component via orange into pure yellow, this through the yellowish green hues to pure green and so on, until there is a return again to red.

The set of uncolored sensations—white, gray, black—forms not a circle but a straight line. At one end is situated the darkest black, at the other end, the brightest white. Between them lie the gray colors of different brightness or darkness. This is illustrated in figure 1, which shows the increase of white and the decrease of black in the passage from black to white through the set of gray colors. Here also there

¹³ Ladd-Franklin, C. Eine neue Theorie der Lichtempfindungen, *Ztschr f Psychol* 4 211, 1892

is a continuous set, but without characteristic turning points as in color sensation. In the change, for instance, from a bluish to a yellowish red a characteristic point is to be found at the pure red. Dimmick¹⁴ suggested such a point in a middle gray, but I think this theory is not justified. If there were such a point, there would be an antagonism similar to that of the color sensations, and this would confirm Hering's theory, about which I shall speak later.

But here I wish to emphasize that black and white as sensations are always of the same kind as the other colors. The opinion of many physicists that black should not be considered a color because its sensation does not arise from a physical stimulus is not right. This point will be discussed in detail when I deal with the phenomena of contrast.

Experience shows that every principal or transitional color can join with each member of the set of black-white sensations. For instance, a pure red may join with white, with black or any shade of gray. In this way are produced a great number of nonsaturated colors—*verhüllte oder ungesättigte Farben*. According to the different brightness or darkness of the gray one distinguishes different sets, in which the relation between the pure red and the gray changes from the highest degree of saturation to the lowest, namely, the gray alone. In this manner results Hering's¹⁵ triangle of shades, *Nuancierungsdreieck*, as shown in figure 2.

Hering suggested by that triangle only a symbolic representation of possible sensations, without regard to the physical stimulus. William Ostwald,¹⁶ on the contrary, apparently without knowledge of Hering's idea, gave the color triangle an objective foundation. For him it was important always to reproduce the same color quality by the exact mixture of pigments. In this way he physically determined the waves reflected by the pigment and the content of physical white, the so-called albedo. Thus he sought to determine the physical stimulus, especially the composition of a pigment which, he supposed, always excites the same sensation. According to Fechner's law he distributed the physically defined pigments in sets with a number of logarithmically ordered sets. Ostwald's suggestion and his whole work concerning color sense were eminently practical. In this respect he brought about substantial improvement.

Considering that every member of the color circle can be enlarged to a triangle of shades, their total number will form a double cone as a symbolic representation of the sum of the sensations (fig. 3). On the axis thereof there are the uncolored sensations—at the top, white, at the bottom, black. At the equator the most saturated colors are to be

14 Dimmick, F. L. Minor Studies from the Psychological Laboratory of Cornell University. A Note on the Series of Blacks, Greys, and Whites, *Am J Psychol* **31** 301, 1920.

15 Hering, E. Zur Lehre vom Lichtsinne, in *Wissenschaftliche Abhandlungen*, Leipzig, Georg Thieme, 1931, vol. 2.

16 Ostwald, W. *Mathetische Farbenlehre*, Leipzig, Verlag Unesma G. m. b. H., 1921.

found, on the upper half of the cone, the bright, more or less saturated colors, in the lower half, the darker ones. Concerning this body of colors also the different opinions of both Hering and Ostwald are valid in the sense already mentioned with regard to the triangle of shades

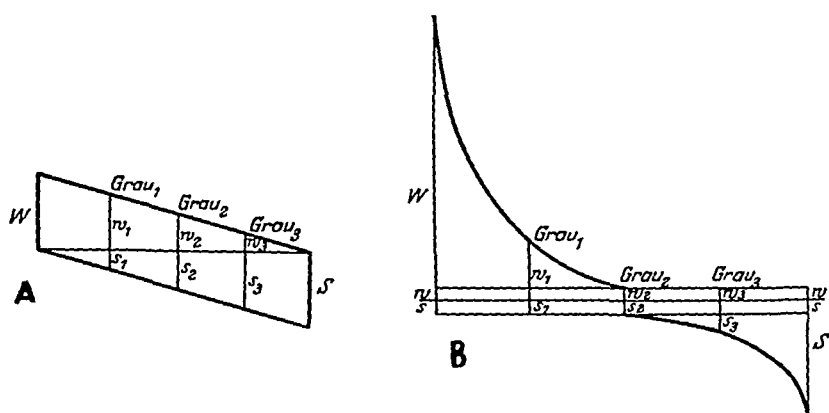


Fig 1—*A*, scheme of the white-black or gray set after Hering,¹⁵ with constant "weight" of sensation. *B*, scheme of the white-black or gray set after Tschermak (in Bethe, *A*, and others *Handbuch der normalen und pathologischen Physiologie*, Berlin, Julius Springer, 1929, vol 12, pt 1, p 300) with change of weight of sensation.

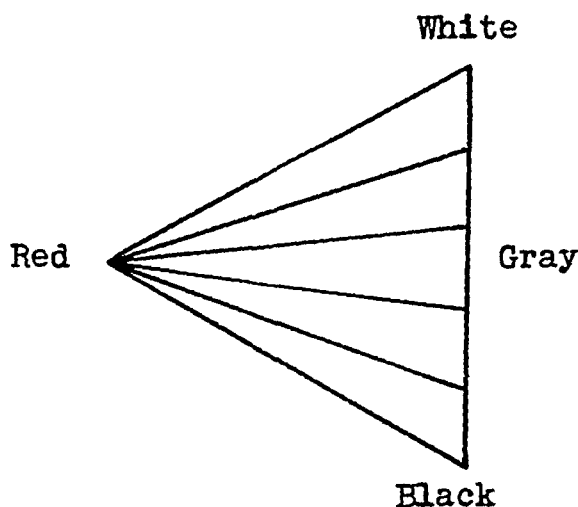


Fig 2—Hering's¹⁵ triangle of shades

Considering the double cone or only the circle of saturated colors, the possible number of steps between green and blue is apparently smaller than that between yellow and red or between red and blue. In other words, green and blue are more nearly alike than the other pairs of principal colors. Ostwald explained this on the basis of insufficient exercise for distinction, but certainly that is not satisfactory. Other authors have constructed oblique forms of double cones or

tetrahedra signifying the smaller or larger distances between the four fundamental colors. Some of these are shown in figure 4¹⁷

The body of colors is a continuum, therefore, theoretically, the number of the possible color sensations is infinite. In reality the number is limited, for in order to distinguish two colors there must be a certain minimum difference between them. An exact absolute number of different color sensations cannot be given.

Because the body of colors has three dimensions, every point thereof may be defined by three coordinates. Therefore, each single color sensation has been given a three-dimensional character. Long ago Grassman and Helmholtz¹⁸ distinguished three relatively independent qualities in each color sensation, namely, brightness, hue and saturation. Saturation means the relation of the pure color to the uncolored portion, hue, the specific color quality with regard to a definite member of

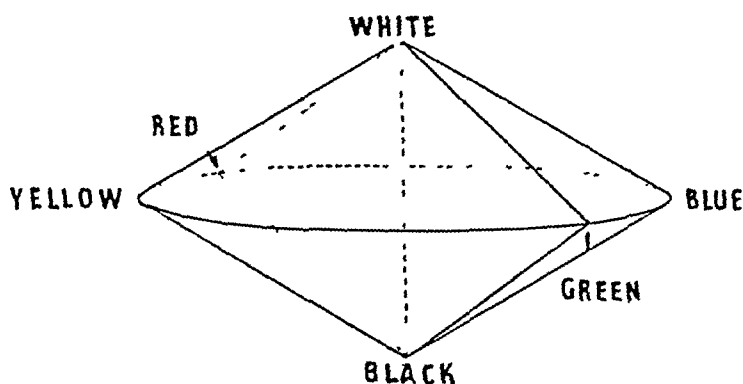


Fig. 3—Double cone as symbolic representation of the totality of colors (Color body after Ostwald¹⁶)

the color circle. Brightness is a quality not easy to define but denotes chiefly a degree of resemblance to a definite member of the white-black set.

In regard to the set of black-gray-white sensations, one cannot speak of saturation, because there is no colored portion. Therefore, the only question is the proportion of white and of black which simultaneously affects the degree of brightness.

Psychologically it is sometimes necessary to discriminate between brightness and darkness or between whiteness and blackness. That depends on the psychologic interpretation. Whether the brightness of a color is determined only by the uncolored part or also by its colored

17 Hofer, A. Zwei Modelle schematischer Farbenkörper und die vermutliche Gestalt des psychologischen Farbenkörpers, *Ztschr. f. Psychol. u. Physiol. d. Sinnesorg.* (Abt. 1) 58 356, 1911.

18 Helmholtz, H. L. W. *Handbuch der physiologischen Optik*, ed. 3, Hamburg, L. Voss, 1909-1911, vol. 2, p. 110.

part has been repeatedly discussed. Originally Hillebrand and Hering¹⁹ supposed blue and green to have a specific darkness and yellow and red a specific brightness. This is the theory of the specific brightness of colors. It was founded on the supposition that in the so-called scotopic vision of the dark-adapted eye there is only the uncolored effect of radiation on the nerve substance, under these conditions one cannot see the different colors. The spectrum at low luminosity appears as an uncolored band of differing luminosity with the maximum at a wavelength of 530 millimicrons, the point at which the light-adapted

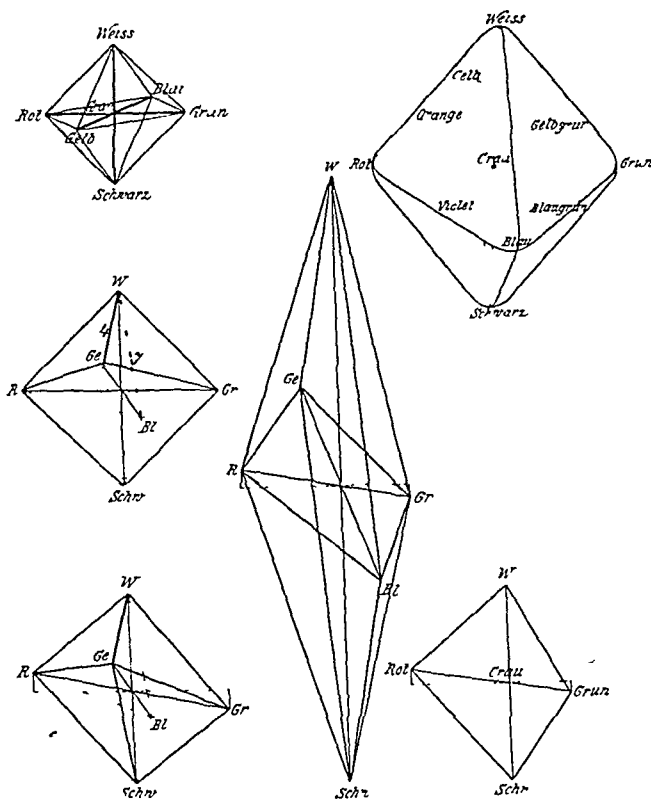


Fig 4—Different forms of the body of colors with reference to their greater or lesser resemblance or relation to the fundamental colors (After Hoffer¹⁷)

eye sees yellow-green. For the light-adapted eye the brightest point in the spectrum is at a wavelength of about 580 millimicrons, which for the dark-adapted eye appears rather dark, the red end of the spectrum is hardly visible to the dark-adapted eye, because red appears as black.

19 Hillebrand, F, and Hering, E. Ueber die spezifische Helligkeit der Farben, Sitzungsber d k Akad d Wissensch Math-naturw Cl 98 70, 1889

The fact that in dark adaptation the red and orange hues become darker and the blue and green brighter has for a long time been called Purkinje's phenomenon. By addition of red and yellow to the uncolored sensation, the long wave end of the spectrum should, according to the specific brightness of these variegated colors, become brighter, and by addition of the specific darkness of green and blue, the other end should become darker.

Since it has become known that there are both a dark apparatus and a light apparatus of the eye, the theory of the specific luminosity of colors has lost its probability. Nevertheless, blue and green may have some dark, and yellow and red some bright, quality. This is perhaps connected with a specific element which led to red and yellow being designated warm colors and blue and green cold, with respect to their esthetic value. However this may be, saturation and brightness of color cannot be considered independent variable factors of sensation.

The view that the three components, brightness, hue and saturation, were independently variable was founded on the theory of Helmholtz, who suggested that these three components were defined by some characteristics of the exciting light. The hue was said to depend on the wavelength, the brightness on the intensity of the radiation and the saturation on the proportions of homogeneous spectral light and of white light. According to this assumption the sensation produced by homogeneous spectral light, namely, light of a single wavelength or, better, of a very narrow band of wavelengths, was considered to be most saturated. But one can perceive after-images or, by contrast, sensations of still greater saturation, as I shall discuss later. When the intensity of light increases the hue changes, and it then has none of the characteristics of a pure primary color. This is a classic example of the confusing of the physical stimulus with the sensation produced thereby.

If, in a strictly psychologic sense, one desires to note the variables in color sensation which may change independently, it seems to me they are the following three: (1) the amount of black-white, (2) the amount of blue-yellow and (3) the amount of green-red sensation. Because green and red, and blue and yellow are by pairs antagonistic in a given sensation, only one partner of each pair can be present.

DEPENDENCE OF COLOR SENSATION ON STIMULUS

The experience of color is usually dependent, on the one hand, on external stimuli affecting the visual organ. These are called light rays. Their composition may be manifold. The experience is founded, on the other hand, on internal physiologic and psychic conditions which essentially determine the effect, the concrete sensation or perception.

Hering⁵ called attention to the fact that the interpretation of one's color sensations is, to a great degree, determined by experience. A piece of white paper on the floor may be perceived as such or as a spot on which the sun is shining. The interpretation depends on the circumstances. Often the color of an object the qualities of which are known is seen not as it really appears but as one remembers it. That sensation Hering called memory color (*Gedachtnisfarbe*). Therefore, in a room in which a reddish or yellowish illumination has been produced by a great many sources of artificial light, things are perceived as having almost the same color as in daylight. Nevertheless, the composition of the reflected light is greatly altered. But this correction does not always obtain, and therefore women are cautious about the shades of blue they wear in artificial light.

In a similar manner colors arising only from a physiologic basis, such as the contrast colors, are often suppressed because the memory colors predominate. The shadow on snow is to the keen observer blue or purple, and the artist paints it so, but the ordinary man does not see this contrast, he sees only the white color of the snow.

Into this variety of experience some order may be brought by following the suggestions of Katz,²⁰ who demonstrated that the so-called material of colors may be perceived differently in different circumstances.

By looking through a small hole at a colored field of any kind at such a distance that it is impossible to distinguish any details of its surface, such as irregularities in texture, one has a perception of what Katz termed "plain colors" (*Flächenfarben*). These colors have an absolute character, although they may show to some extent also in the third dimension. The blue sky, for instance, has this character. I should prefer to call this kind of color perception "absolute color," because the color has lost all factors of its usual appearance.

The second type of colors that one sees are the so-called surface colors (*Oberflächenfarben*). When one perceives a colored surface, for example a paper, with irregularities of texture, one has the impression that the color is bound to this surface.

Katz named some phenomena already described by Hering spatial colors (*Raumfarben*). A darkly shaded corner in a room seems to be entirely gray, when the room is illuminated this area takes on color. This kind of color perception has three dimensions.

The discrimination which Katz makes between the appearances of colors under different circumstances is not always possible, as this depends also on the disposition of the observer. Nevertheless, his state-

²⁰ Katz, D. Die Erscheinungsweisen der Farben, *Ztschr f Psychol*, 1911, supp 7.

ments seem justified, since neurologists have described cases of loss of perception of surface colors alone after injury to the brain (Gelb²¹)

But all these questions are of merely psychologic interest. The question as to whether a person is able to experience all color sensations which are called color material is one dealing with a primitive function

How can the essential factors of the color sense of a person be determined? As in all researches concerning sensation, investigation is founded on special tests. One considers how the tests are performed and especially if the person tested does them in the same manner as the majority of persons. If he does so, his color sense is considered normal.

In tests of the color sense the subject is asked to decide whether two colored fields are equal or not, and if they are not equal, in what manner they are unequal. This method is known as that of color equation.

Between the stimulus and the psychic sensation lies the transformation of the stimulus into chemical or electrical processes in the peripheral organ and their conduction along the visual pathway to the central organs in the brain. The electrical processes arising in the retina and conducted along the optic nerve have been explored recently by Adrian and Matthews²² and by Kohlrausch²³ and his pupil Sachs²⁴. The action currents are different according to the light which influences the eye. One must suppose that in the peripheral organs the process is specific, depending on the specific character of light as to wavelength and intensity. But there is no need to discuss this here.

To consider only the relation of the physiologic and the psychic final link is complicated enough. For clinical purpose, physicians are usually content to carry out some simple tests by means of cards, such as those of Stilling or of Ishihara.

Even if specially adapted spectroscopes are used, for example the so-called anomaloscope, only *one* equation is tested. As a rule, conclusions are drawn therefrom as to the entire color sense. But that can be allowed only for practical, not for scientific, purposes. If the color sense of a person is to be studied exhaustively in a scientific manner, it must be gaged. Theoretically, it would be of no importance whether for this purpose homogeneous or mixed light is used. The physical definition of homogeneous spectral lights is easier, and they are therefore more easy of exact estimation than mixed lights, whether they arise by

21 Gelb, A. Ueber den Wegfall der Wahrnehmung von "Oberflächenfarben," *Ztschr. f. Psychol.* 84 193, 1920.

22 Adrian, E. D., and Matthews, R. Action of Light on the Eye, *J. Physiol.* 63 378, 1927, cited by Kohlrausch²³.

23 Kohlrausch, A. Die elektrischen Vorgänge im Sehorgan, in Schieck, F., and Bruckner, A. *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1932, vol. 2, p. 118.

24 Sachs, cited by Kohlrausch,²³ p. 150.

reflection from pigment or by absorption when passing through filters. For general demonstrations the second method may be sufficient, but for gaging, the use of homogeneous spectral lights is indispensable. Therefore, I shall discuss only some experiments carried out by means of spectral lights.

By mixing three suitably chosen spectral lights it is possible to produce all colors, at least their hue. On this fact is based the three component theory propounded by Thomas Young and improved by Helmholtz (fig 5). On this basis rest the investigations of the majority of English authors and those of Helmholtz and his pupils in Germany. According to their experiments the colors red, green and blue or violet are usually taken as fundamental colors, because, as I have already said, by mixing these all other hues in the spectrum can be obtained.

The principle employed depends on the fact that the mixture of two lights which are not too far apart in the spectrum, for instance orange and yellowish green, gives the appearance of the color of some light lying between the two—in the sample chosen, orange, yellow, etc. The

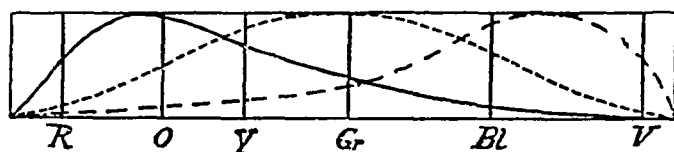


Fig 5—Scheme of the course of excitability curves for the three kinds of fibers, after Young and Helmholtz, with reference to different spectral lights. The colors in the spectrum are distinguished by their initials. The continuous line is the curve for red, the dash line, the curve for green, and the dash and dot line, the curve for violet.

color produced by the mixture depends on the proportions of the two components, and the resulting color will be nearer that of one of them.

When the two components are more distant from each other in the spectrum the resulting colors become less and less saturated, nevertheless, the hues accord with those of the colors produced by lights between the components. But at a certain distance the result of the mixture is colorless, without hue. These are the so-called complementary lights or antagonistic lights. The number of pairs of complementary colors is great—for instance, yellow and blue, and yellow-green and violet (fig 6).

One may object that the mixture of a yellow and a blue pigment is not white but green, and that is true, but here (fig 7), as was shown by Helmholtz, the wavelengths which affect the green sensation are residuary. The yellow pigment absorbs the short wave half of the spectrum, effecting a blue sensation. The blue pigment absorbs the red and yellow. When the two are mixed there remains only the middle part of the spectrum, which appears green.

If the two components in the spectrum are still farther apart, especially if they are situated at the two ends of the spectrum, there result sensations of color that are not in the spectrum, namely, the purple colors

Such color equations are given in immense number by different authors

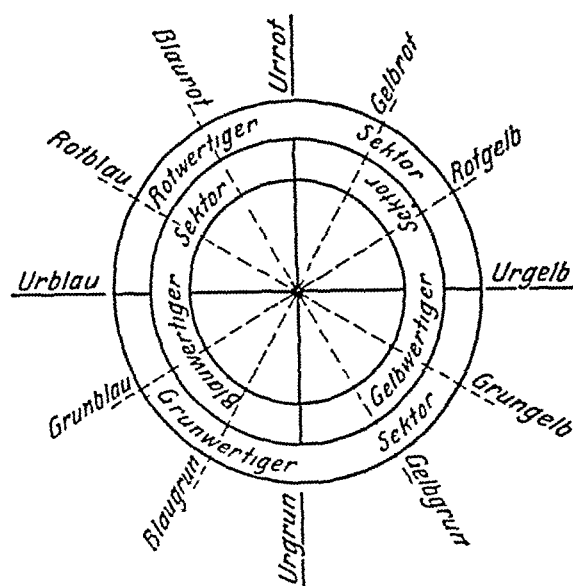


Fig 6—Complete color nuance sequence or color circle of Hering, with the antagonistic colors contrasted (After Tschermak, in Bethe, A, and others *Handbuch der normalen und pathologischen Physiologie*, Berlin, Julius Springer, 1929, vol 12, pt 1, p 300)

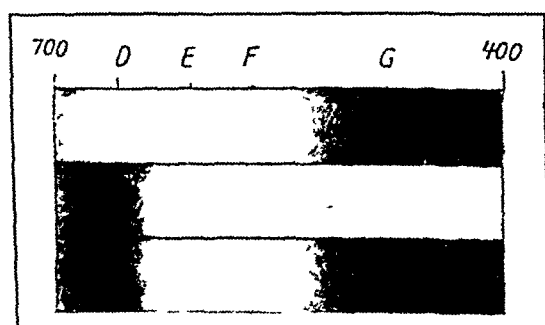


Fig 7—Scheme of spectral absorption by yellow pigment, by blue pigment and by a mixture of both green appearance of the residual rays of the green spectral region Yellow pigment + red light + yellow light + green light —blue light Blue pigment —red light —yellow light + green light + blue light Mixture of yellow and blue pigment + green light as residual physical subtraction (After Tschermak, in Bethe, A, and others *Handbuch der normalen und pathologischen Physiologie*, Berlin, Julius Springer, 1929, vol 12, pt 1, p 300)

In these experiments daylight was not used as a rule, on account of the change in the intensity of light caused by the variations in the clouds and in the altitude of the sun Therefore, artificial light was

usually employed. But here, with gaslight, for instance, the intensity of the different parts of the spectrum is dependent on the pressure of the gas and the temperature of the flame resulting therefrom. The same may be said of electric light, which varies in quality according to the intensity of the electric current. According to the law of Wien, the maximum of intensity of different wavelengths in the spectrum changes with increasing temperature, in the sense that the shorter waves predominate.

Theoretically, one may calculate the amount of each wavelength according to a standard spectrum, for instance, that of daylight with constant dispersion, as in the interference spectrum. But that cannot be achieved when the data concerning the spectrum used are not specifically given, such as the dispersion of the prism and the distribution of the intensity. Therefore, the results of different observers are only slightly comparable.

In regard to the constancy of the physiologic conditions of the visual organ, the earlier investigations are still more unreliable. The general adaptation of the eye especially has not been sufficiently taken into account. Therefore, dark adaptation has greatly changed the observations, and a great many former researches are worthless, because by increasing dark adaptation the colors become less and less saturated. A great number of those experiments were carried out before the importance of dark adaptation on color vision was realized. Recently Kohlrausch and van Meerendonk²⁵ emphasized the fact that in order to gauge the color sense good light adaptation is indispensable. Pitt²⁶ in his latest investigation remained ten minutes in a dark room before beginning his experiments. A remarkable degree of dark adaptation was obtained.

Thus this research is complicated. If, nevertheless, conclusions are drawn with some reserve as to the functional dependence between physical stimulus and color sensations, it is necessary to note that the results are not entirely satisfactory. This is true especially because for knowledge of the whole color system and its variations investigations should be made on a far greater number of persons than have been studied up to the present.

I shall first relate some experiments in spectrum gaging. In reducing the lights necessary for producing all colors to the smallest number, namely, to three lights, it is not always possible or necessary to use all three of those suitable gaging lights.

25 Kohlrausch, A., and van Meerendonk, P. Ueber den Geltungsbereich spektraler Farbgleichungen, *Ztschr f Sinnesphysiol* 66 45, 1935.

26 Pitt, F H S. Reports of the Committee upon the Physiology of Vision XIV Characteristics of Dichromatic Vision with an Appendix on Anomalous Trichromatic Vision, Medical Research Council, Special Report Series, no 200, London, His Majesty's Stationery Office, 1935.

The experiments of König and Dieterici²⁷ showed that for a stimulus to produce the colors at the two ends of the spectrum it is sufficient only to change the intensity of a light of 670 millimicrons at the red end and of about 430 millimicrons at the violet end of the spectrum. These so-called end spaces (*Endstrecken*) of the spectrum extend at the red end to 655 millimicrons and at the violet end to 430 millimicrons. Toward the middle of the spectrum there are two smaller spaces where it is necessary to mix two lights (e. g., 670 and 580 millimicrons or 475 and 433 millimicrons) in order to obtain the colors produced by stimulation with a homogeneous light. These spaces extend from about 655 to 630 millimicrons and from 475 to 430 millimicrons. Between those two spaces, the so-called *Zwischenstrecken*, lies the middle space. It occupies the range from 630 to 475 millimicrons. Here at least three lights are needed to produce a hue by mixture of the colors effected by homogeneous light in this part of the spectrum.

But it is much easier not to mix three lights on one side of the spectrum but instead to use two lights on one side and to add light from a third to the homogeneous one in the other half of the field. The light that is added can then itself again be gaged by another equation, and so on.

I shall here briefly touch on the manner of gaging. The equations obtained in this way may be treated mathematically. The amount of light used for the different wavelengths is measured by the width of the slits of the apparatus. By this method of gaging it is possible to express the physiologic value of each wavelength in the spectrum by the exciting effect of three suitable homogeneous spectral lights.

The investigations of König and Dieterici²⁷ concerned the dispersion spectrum of gaslight. The energy distribution of this light is known to be unequal, showing a greater dispersion toward the short wave end. Therefore, a conversion into the spectrum of equal dispersion, namely, the interference spectrum, is desirable. If one wishes to obtain gaging curves with regard to a standard light, such as sunlight, a further conversion is necessary. All these conversions have been carried out by König and Dieterici. Such curves have also been made by many other authors. I shall mention only Abney, Pitt, Lord Rayleigh and Kohlrausch.²⁸

²⁷ König, A., and Dieterici, C. *Die Grundempfindungen im normalen und anomalen Farbensystemen und ihre Intensitätsverteilung im Spektrum*, in König, A. *Gesammelte Abhandlungen zur physiologischen Optik*, Leipzig, J. A. Barth, 1903, p. 214.

²⁸ (a) Abney, W. de W. *Researches in Colour Vision and the Trichromatic Theory*, London, Longmans, Green & Co., 1913. (b) Pitt²⁶. (c) Rayleigh *Experiments on Colour*, Nature, London **25** 64, 1881. (d) Kohlrausch and van Meerlandonk²⁵.

I confine myself to the results of König and Dieterici, because they allow a comparison with curves obtained by another method, which I shall speak about later

From the curves in figure 8 which were made for persons with normal color sense, it is seen that those for the two observers König and Dieterici (the continuous and the dash line) agree well. These curves relate to the three gaging lights, red, yellowish green and blue. The unit of these is at first arbitrary. But the authors took comfort in the three component theory, which states that the simultaneous and equally great stimulation of three fibers produces the white, or colorless, sensation. Therefore, they took as the unit for each of the gaging lights the amount which was necessary to produce the white

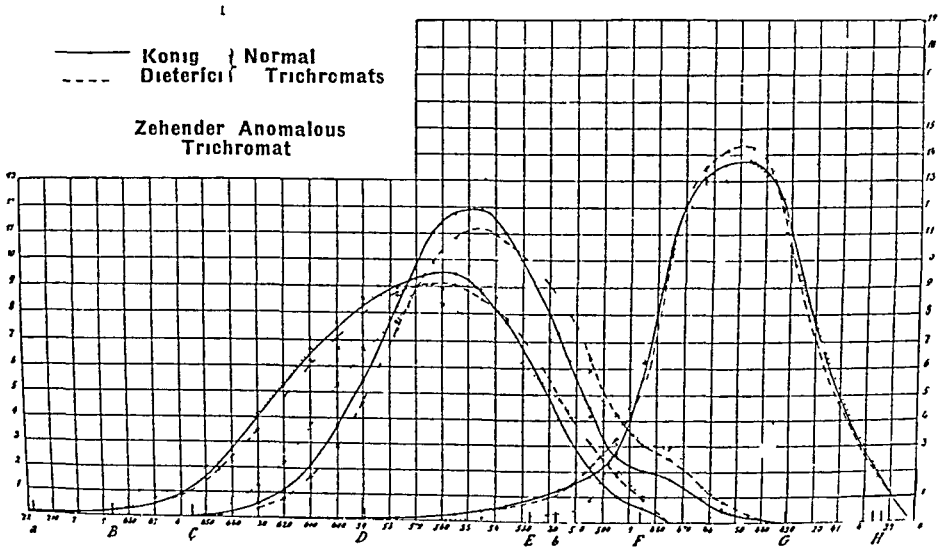


Fig 8—Curves for the three fundamental sensations (After König and Dieterici²⁷)

sensation by the mixture of all of them together. But this is arbitrary if not a *petitio principii*.

König and Dieterici tried to prove that in reality the three curves represent the course of the so-called fundamental sensations. By this name they designate a sensation which corresponds to a simple process in the peripheral organ. Donders²⁹ called it fundamental color. The ordinates of the curves of König and Dieterici should therefore represent for each point of the spectrum the amount of the excitation of the three fibers which the Young-Helmholtz theory suggested. König and Dieterici took as the fundamental sensation, first, a red which lies at some distance from the red end of the spectrum, toward the purple, second, a green corresponding to a wavelength of 505 millimicrons, and third, a blue of about 470 millimicrons. These authors

²⁹ Donders, F. C. Ueber Farbensysteme, Arch. f. Ophth. **27** 155, 1881

were of the opinion that these three colors correspond to three of Hering's original colors—the *Urfaiben*. But that is not correct. Hering called attention to the fact that the supposed red is still somewhat yellowish and therefore does not correspond to Hering's red, which is neither yellowish nor bluish. Also the green produced by 505 millimicrons contains some yellow. The original, neither yellowish green nor bluish green, is situated at about 495 millimicrons.

In contrast to the gaging of the spectrum by three lights, the gaging with four according to the four pure colors of Hering has hitherto been carried out only once. These investigations were made at Hering's suggestion in the Physiological Institute in Leipzig, Germany³⁰. They were based on the main idea of Hering's theory, namely, that on the one hand red and green and on the other hand yellow and blue are produced in the visual substance by antagonistic processes. If two lights, one producing the sensation of red and the other of green, are mixed, the processes compensate so far as they are of equal quantity. Only an excess of the process which corresponds to one or the other color is perceived as red or green. The analogy is valid also for blue and yellow. When the antagonistic excitations are equal the result is the colorless sensation. The white is, according to Hering's theory, a residuary process, according to the Young-Helmholtz theory, it is a process of summation.

In order to determine the content or value of a pure color produced by a homogeneous light, the amount of the pure antagonistic color necessary to extinguish the opposite color was determined. For instance, to an orange light of 615 millimicrons was added sufficient pure green light of 495 millimicrons to produce a pure yellow. Because it is not easy to decide if this yellow is really pure and does not contain some greenish or reddish color, the other half of the field in the eye piece of the spectroscope was filled with a pure yellow (of a wavelength of 580 millimicrons), taken from the other collimator of the instrument. By mixing this with a certain amount of white light any difference in the saturation was equalized. The width of the slit which gave sufficient green light to extinguish the red component of the orange light gave a value equivalent to the amount of red light present (fig. 9). In this way the values for red were determined for the parts of the spectrum containing red (the long waves from the end to the pure yellow at 580 millimicrons and at the violet end from the pure blue, corresponding to 480 millimicrons to the end).

In a similar manner the values for pure yellow and for pure blue were found.

30 Bruckner, A. Zur Frage der Eichung von Farbensystemen, *Ztschr. f. Psychol. u. Physiol. d. Sinnesorg.* (Abt. 2) 58:322, 1927.

In these equations a difficulty arose, because by certain mixtures a pure red resulted. For comparison it was not possible to use a homogeneous light, because there is none in the spectrum. Therefore, a filter consisting of a solution of carmine and gentian violet was used (fig 9), which gave a pure red. As this filter light was used for comparison only, and not for matching the values of the lights, there could be no objection to its use for this purpose.

In the mixtures containing light of short wavelengths (violet or bluish green) the yellow pigmentation of the macula interferes with exact measurement. Therefore, the observations were made with the paramacular part of the retina, a dark spot 1.3 mm in diameter in the

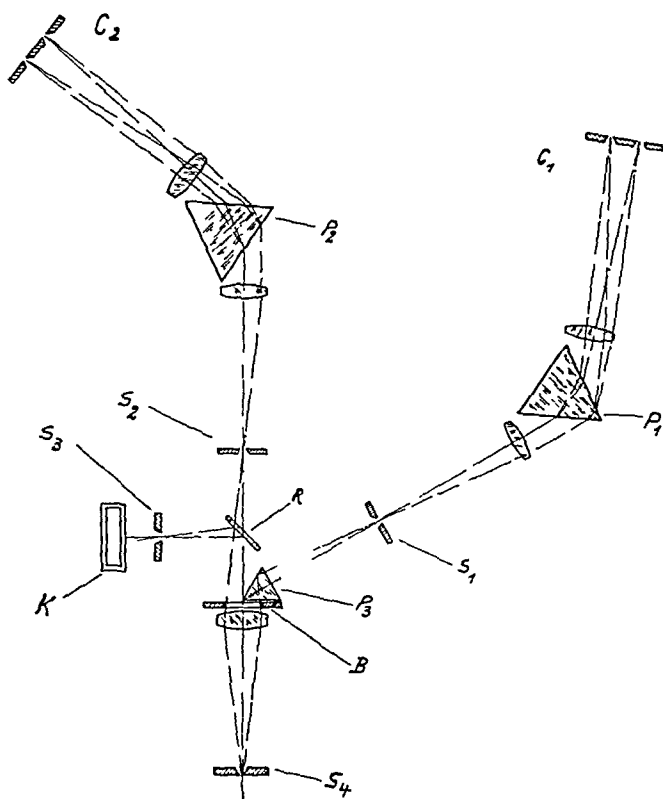


Fig 9— C_1 and C_2 , the two collimators each with two slits, so that two independent spectrums are thrown by the two prisms P_1 and P_2 . The rays are united in the slits S_1 and S_2 , and go directly or by complete reflection through prism P_3 into the ocular with the slit S_4 . K is a small glass vessel filled with color solution, the light from which after passing through the slit S_3 and reflection from the mirror R is united with the mixture of rays from the collimator C_2 . B is the diaphragm. (After Bruckner³⁰)

middle of the field of the eye piece excluded the central part of the retina. The results were then obtained on excitation of only peripheral parts of the retina by slightly excentric observation. After some trials the experiments were easily carried out.

Daylight was used, and as the duration of a series of experiments was from five to six hours, the intensity of light naturally changed. But

by carrying out several series of experiments and by changing the sequence of the tested lights, this influence was eliminated, as far as possible, by ascertaining averages

The values first found for the spectrum of dispersion were later converted into those of the interference spectrum of the sun with equal dispersion in all parts. The curves shown demonstrate averages of different curves. These are obtained by setting the highest values as 100 millimicrons and the average curve from 5 to 5 millimicrons (fig 10). The curves show the effect of different parts of the spectrum. According to Heining's suggestion of the antagonistic character of the two pairs of colors, the curves for red and yellow are above, and those for green and blue below, the zero line. The parts of the curves obtained by observation are continuous, those secured by graphic interpolation

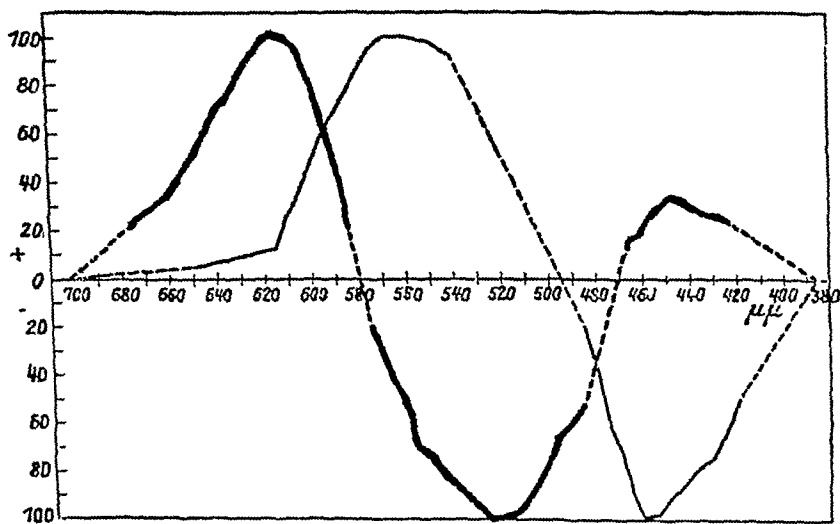


Fig 10—Gaging of color systems. Yellow and red are above the zero line, and blue and green are below. The parts of the curves obtained by observation are continuous, those by graphic interpolation are made up of dash lines. (After Bruckner³⁰)

are made up of dash lines. Here it was impossible to get good equations because the mixtures were insufficiently saturated.

The positions of the maxima are of some interest. That of the red lies in the orange at about 615 millimicrons, where one would not expect it. That of green is situated at 530 millimicrons, at a point where the green appears yellowish. It is remarkable that the highest point of the value of yellow is to be found at about 570 to 550 millimicrons, at a greenish yellow which does not seem to have such a great amount of yellow.

The curves agree with the position of the pure colors in the spectrum. The course of the red-green curve permits the conclusion that corresponding to the direct aspect of pure yellow lies at 580 millimicrons and the pure blue at 470 millimicrons. The position of the pure green

at 495 millimicrons is, owing to the interpolation, perhaps, not absolutely exact, but it corresponds to the results which were obtained by free determination in a spectrometer of the apparently pure green

If the theory of antagonistic colors is right, namely, that the antagonistic values cancel out in daylight, the area of the yellow curve and that of the red and of the green should be equal by pairs. This apparently is not so. The area of the yellow is larger than that of the blue, and the same is true as to red and green. This fact may be based on the color of daylight, namely, that it is not absolutely colorless or neutral but to the human eye has a yellowish red hue. That is perhaps caused by deflection of light in the atmosphere or by the light falling through the sclera into the eye. In the latter case reddish light is added to that passing through the pupil.

A short discussion of the so-called Bezold-Brücke phenomenon³¹ is of some interest. This consists in the alteration of the hue with increasing intensity of light. At low intensity yellow and blue occupy a very small space in the spectrum. If the intensity increases, the blue and yellow extend and correspondingly the red and green shrink.

When looking at the curves of the four pure colors in Hering's sense (fig 10), it is easy for one to understand why yellow, with its low value up to 610 millimicrons, remains under the threshold, in contrast with the high value of red in this region. The same may be said of the region from 520 to 490 millimicrons, where the excitation values of green are higher than those of yellow. As I have said, there is no common scale for the ordinates of the red-green and the yellow-blue curve. But the three component curves do not give a satisfactory explanation of this point.

A greater interest attaches to the relation between curves for Hering's pure colors and those of König and Dieterici. From the first it is supposed that the gaging of the spectrum with three lights cannot fundamentally give other results than with four components. This consideration suggested the attempt to transfer the König curves, obtained from three gaging lights, into curves of the four Hering colors. This proved to be possible. In the following discussion the curves of König, whose color system seems to possess great similarity to that of the observer of the four light gaging method, are transformed into Hering's colors.

In König's curves (fig 8) at about 575 millimicrons, where the green and the red curve cross, their ordinates are equal and the spectrum at this point appears pure yellow. Therefore, the two gaging lights must be a yellowish red and a yellowish green. The red and the green part extinguish each other, according to Hering's theory, otherwise, a pure yellow would never result. If it is supposed that the value for

³¹ Bezold, W. Die Farbenlehre, ed 2, Braunschweig, F. Vieweg & Sohn, 1921, p. 19.

yellow in the green and in the red curve are added together, by the addition of the ordinates of these curves the value for yellow in these lights is obtained

In view of König's blue curve it seemed permissible to take it as a measure also of the value of Hering's blue. Of course, it was essential to consider that beyond the point of pure green (at about 495 millimicrons) to the short wave end of the spectrum, in the red and green curves of König some yellow was present. If the ordinates of these two curves are added, the equivalent should be subtracted from the ordinates of König's blue curve.

In this manner the height of the ordinates of Hering's yellow and blue curves were determined from 10 to 10 millimicrons. The maximum in each curve was taken as 100, and the other values were

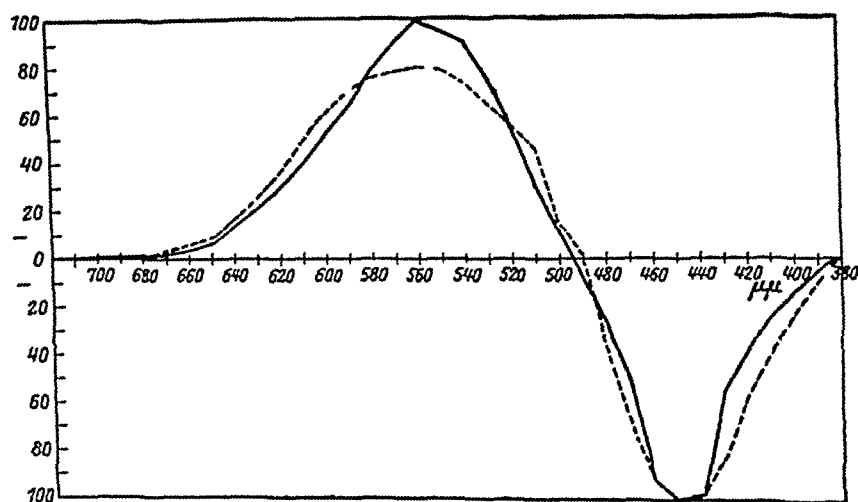


Fig 11—Gaging curves transformed from König (normal) and Zehender (deuteranomaly) for yellow and blue (After Bruckner³⁰)

reckoned proportionally. The resulting curves agree almost entirely in all respects with those obtained by experiment according to Hering's method (fig 11)

For the determination of Hering's green and red curves the procedure was similar. At the red end of the spectrum, where there are only König's red and green, according to Hering's suggestion of the antagonistic character of red and green, one counts only the difference of the ordinates of the two curves, representing the value of Hering's pure red. Accordingly, in the region from 575 to 495 millimicrons, from the difference of the ordinates the value of the pure green in Hering's sense is to be found. The proportional determination was made in the same manner as for the blue-yellow curve. The method of determining the red values at the violet end of the spectrum was more complicated, its consideration is not essential here.

The cuives for Heimg's red-green obtained from Konig's curves (fig 12) are similar to those found by direct observation The slight differences present may be due to the fact that Konig made his observations on the macula while the other obseervations were made on the paramacular region of the retina

As shown in table 1, the maxima of the curves obtained by the two methods are sufficiently close when one considers the different sources of light used by the authors

It is remaikable that almost at the same time similar transformations were carried out by Hiecke³² and Schrodinger,³³ which produced similar results

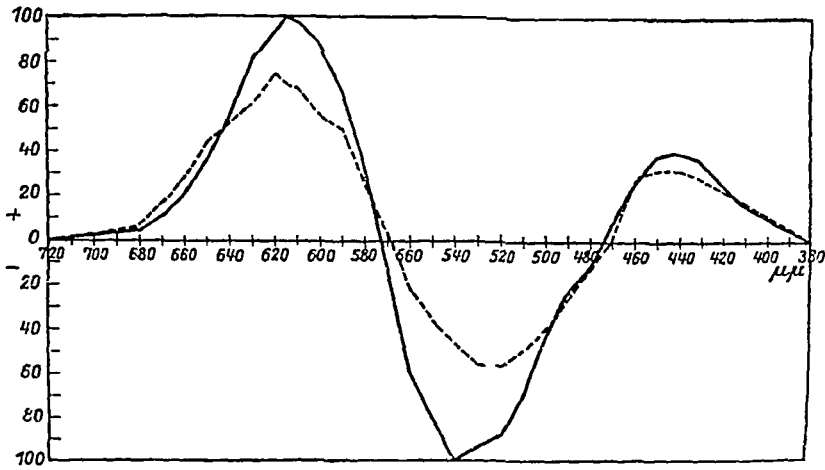


Fig 12—Gaging curves transformed from Konig (normal) and Zehender (deuteranomels) for red and green (After Bruckner³⁰)

TABLE 1—Position of the Maxima for Heimg's Fundamental Colors
Normal System

Colors	Hering, Millimicrons	Konig, Millimicrons
Red	615	615
Red in the violet	450	440
Green	525-520	540
Yellow	570-550	560
Blue	460 455	450

I may, perhaps, suggest that the discussion regarding the gaging of the light of the spectrum shows that it is not possible to conclude from these experiments whether a three or a four component theory is

32 Hiecke, R Neue Folgerungen aus den Farbenempfindungskurven von A Konig und C Dieterici, Ztschr f Psychol u Physiol d Sinnesorg (Abt 2) 58 111, 1927
33 Schrodinger, E Ueber das Verhaltnis der Vierfarben- zur Dreifarben- theorie, Sitzungsbd d k Akad d Wissensch Math-phys Cl 134 471, 1925

valid. By this method one can never determine which colors of lights are principal or fundamental in the sense that they correspond to a relatively simple process in the visual substance.

By gaging, nothing is learned concerning the curve of the white-black sensations. There is no proof of the Young-Helmholtz theory—that the simultaneous and equal stimulation of the nerve fibers, or whatever one calls them, producing the three color sensations gives a noncolored sensation. The same can be said of Hering's theory that the colorless sensations arise from an independent substance.

A further suggestion by Hering, that the distribution of the luminosity in the spectrum at low intensity and at dark adaptation of the eye represents the values of the effect on the white-black substance, is not correct. The maximum luminosity for the light-adapted eye is situated

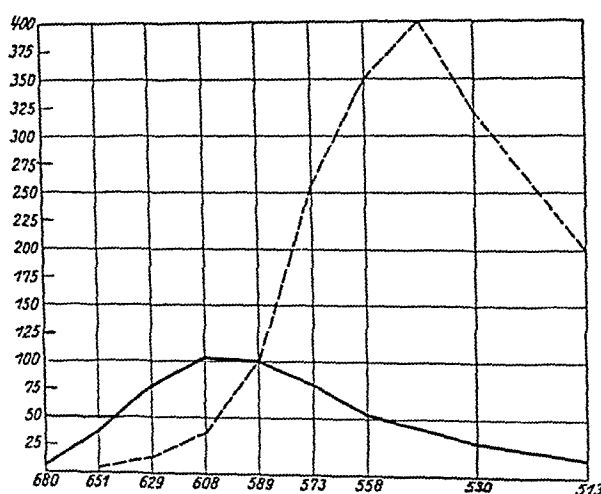


Fig 13—Equivalence curves of the distribution of the spectral luminosity (spectral dispersion of gaslight). The continuous line indicates so-called peripheral values, by daylight vision, the broken line, so-called scotopic values (extrafoveal average values). (After von Kries, in Bethe, A., and others. *Handbuch der normalen und pathologischen Anatomie*, Berlin, Julius Springer, 1929, vol 12, pt 1, p 685.)

in the spectrum at about 580 millimicrons, that for the dark-adapted eye, at the low intensity of the spectrum at about 530 millimicrons.

As I have already observed, Hering and Hillebrand would explain this fact by the specific luminosity of the various colors. But this cannot be accepted as correct, since in the scotopic eye a different apparatus functions from that in the light-adapted eye. The luminosity curve of the spectrum for the periphery of the retina, where colors are not perceived, is the same as that for the macular region of the light-adapted eye and does not in any way agree with the luminosity curve of the dark-adapted eye (fig 13).

It seems to me hardly permissible, however, to conclude that with peripheral vision the values of the white-gray-black component of the various colors are perceived, because one cannot know whether or not when they are not perceived the colored component has any influence on the luminosity. Therefore, at present it is not possible to determine by experiment the so-called white value of the different lights in the light-adapted eye. If one attributes to the colored component a constant value of luminosity, one may consider the luminosity curve as that of the white value.

This may be correct if the specific colors have some peculiar luminosity, but the "weight" (*das Gewicht*, Hering) of the amount which corresponds to them is small in proportion to that of the process corresponding to the colorless part of the sensation. Therefore, the influence of the colored part of the resulting brightness is small.

The fundamental thought of Hering's theory was founded on the idea that by stimulation of the peripheral organ in the visual substance some chemical substances of antagonistic character were produced proportional to the sensation.

This hypothesis of Hering seems to be supported by recent researches in general neurophysiology. E. T. von Brücke, professor of physiology in Innsbruck, Austria, has permitted me to discuss this point somewhat fully. Physiologic researches have proved that after excitation of a nerve certain substances are produced in the effector organ (e. g., the muscle). For instance, after excitation of the vagus nerve acetylcholine is present in the muscle of the heart, after stimulation of the sympathetic fibers a substance similar to epinephrine is formed. In these two cases substances are produced the effect of which is accepted as that of the stimulated nerves. American authors found also that by stimulation of the sympathetic chain a substance they called sympathin, which is probably merely epinephrine, was produced.

Loewi's pupils found that after stimulation of the parasympathetic nerves the effect in the organ concerned is due to acetylcholine, which is also found in the aqueous humor of the anterior chamber of the eye.

Similar processes have since been noted in the central nervous system, especially in the sympathetic ganglions. If these are produced by the central nervous system itself, the stimulation must be effected by humoral substances within this system. Therefore, Hering's theory of the formation of antagonistic chemical substances is in accord with the recent findings regarding the action of peripheral nerve endings where contractions are controlled by the antagonistic action of acetylcholine and epinephrine.

(To be continued)

NATURE OF THE FILTRABLE AGENT OF TRACHOMA

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It is now generally agreed that none of the bacteria which have been found on the trachomatous conjunctiva are concerned etiologically in the disease. In 1912 Nicolle, Blaizot and Cuénod¹ concluded, on the basis of two filtration experiments in which they had employed modified Berkefeld V filters, that the agent of trachoma was filtrable. In the first experiment a bacteria-free filtrate of trachomatous material produced a disease typical of experimental trachoma in a *Macacus mius* (Barbary ape) which was proved to be trachoma by transfer to a blind human eye. In the second experiment a bacteria-free filtrate proved infectious for a chimpanzee.

Confirmation of the filtrability of the agent of trachoma has come from the experiments of Thygeson and Proctor² with baboons, Thygeson, Proctor and Richards³ with a human volunteer and Julianelle, Morris and Harrison⁴ with *Macacus rhesus* monkeys. These investi-

Aided in part by a grant from the Committee on Scientific Research of the American Medical Association

Read before the Section on Ophthalmology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 15, 1938

From the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, and the Institute of Ophthalmology, Presbyterian Hospital, in cooperation with the Division of Health, Office of Indian Affairs, Department of the Interior, Washington, D C

1 Nicolle, C, Blaizot, L, and Cuenod, A. Le magot, animal réactif du trachome. Filtrabilité du virus, pouvoir infectant des larmes, *Compt rend Acad d sc* **155** 241, 1912

2 Thygeson, P, and Proctor, F I. Filtrability of Trachoma Virus, *Arch Ophth* **13** 1018 (June) 1935

3 Thygeson, P, Proctor F I, and Richards, P. Etiologic Significance of the Elementary Body in Trachoma, *Am J Ophth* **18** 811, 1935

4 Julianelle, L A, Morris, M C, and Harrison, R W. Studies on the Infectivity of Trachoma. Further Observations on the Filterability of the Infectious Agent, *Am J Ophth* **20** 890, 1937

gators have found that the agent filters inconstantly and that apparently certain conditions of filtration must be satisfied in order to obtain active filtrates

FILTRABILITY OF THE AGENT OF TRACHOMA

In table 1 are summarized the results of 22 filtration experiments performed by us. Six active filtrates were obtained under conditions designed to insure maximum activity of the material to be filtered and minimum losses due to adsorption in the filter. Maximum activity of

TABLE 1—Results in Twenty-Two Filtration Experiments

No of Experiments Performed	Type of Filter	Material	Filtrate Cultures	Subjects	Infectivity of Filtrate
3	Berkefeld V	Pooled expressed follicular material	No growth	M rhesus monkeys	All 3 negative
3	Berkefeld N	Pooled expressed follicular material	No growth	Human volunteers	All 3 negative
1	Chamberland L2	Pooled expressed follicular material	No growth	Human volunteers	Negative
2	Chamberland L3	Pooled expressed follicular material	No growth	Human volunteers	Both negative
2	Elford 0.70 μ A P D *	Epithelial scrapings, case of acute trachoma	No growth	M rhesus monkeys	1 of 2 positive
4	Elford 0.75 μ A P D	Pooled epithelial scrapings, cases of highly active trachoma	No growth	Baboons	All 4 positive
1	Elford 0.6 μ A P D	Pooled epithelial scrapings, cases of highly active trachoma	No growth	Human volunteer	Positive
2	Elford 0.6 μ A P D	Pooled epithelial scrapings sent by air mail (36 hr) to laboratory	No growth	Human volunteers	Both negative
2	Modified Berkefeld V	Epithelial scrapings with many inclusions, case of active trachoma	No growth	Baboons	Both negative
1	Modified Berkefeld V	Pooled epithelial scrapings, 3 cases of active trachoma	No growth	Baboons	Negative
1	Modified Berkefeld V	Pooled epithelial scrapings 2 cases of active trachoma	No growth	Baboons	Negative

Results 6 successful filtrations in 22 experiments

* Average pore diameter

the trachomatous material was attained in 5 of the 6 successful experiments by using pooled epithelial scrapings from trachomatous Indian children (selected for activity) from the Theodore Roosevelt Trachoma School, Fort Apache, Ariz. For the sixth experiment the material was obtained in New York from an adult white person with acute trachoma. Losses during filtration were minimized by the use of Elford graded collodion membranes of a pore size sufficient to retain the conjunctival bacteria (from 0.6 to 0.75 micron average pore diameter) and by employing a filtration surface of only 0.6 sq cm.

Of the 16 negative filtrates, 11 were obtained with unsatisfactory material and with the ordinary types of porcelain and Kieselguhr filters. Scrapings from unselected trachomatous subjects were employed in all 11 of the 16 experiments, and in 9 of the 16 expressed follicular material was used rather than epithelial scrapings. Since the available evidence now indicates that the agent of trachoma has an epithelial localization, the type of material utilized is probably important.

Characteristically, the material yielding active filtrates contained substantial numbers of epithelial cell inclusions (Halberstadter and von Prowazek,⁵ 1907)

RELATION OF THE FILTRABLE AGENT TO THE ELEMENTARY BODIES OF TRACHOMA

Minute bodies of a size consistent with filtrability were noted in trachomatous material by Halberstadter and von Prowazek.⁵ These bodies, which they considered to be the active agent of the disease, were seen both free in the secretion and massed together in the cytoplasm of epithelial cells to form the inclusions, which are now recognized as the most characteristic microscopic evidence of the disease. While these bodies have been accepted as etiologic by Axenfeld,⁶ Lindner,⁷ Taborisky,⁸ Rotth,⁹ Bengtson and Rolufs¹⁰ and others, the evidence advanced has been insufficient to gain universal acceptance of the theory.

The following sections of this report summarize the results of our studies on the elementary bodies of trachoma.

NATURE OF THE ELEMENTARY BODIES OF TRACHOMA

In Giemsa-stained epithelial scrapings from persons with active trachoma the elementary bodies were seen as minute, sharply defined, reddish blue bodies having a diameter of about 0.25 micron (fig. 1). They were gram-negative but failed to stain with the usual counterstains. Comparison with the elementary bodies of vaccinia and molluscum contagiosum revealed no striking morphologic differences but different staining properties. In contrast to the usual type of virus ele-

5 Halberstadter, L., and von Prowazek, S. Ueber Zelleinschlüsse parasitärer Natur bei Trachom, *Arch. f. Ophth.* **26** 1, 1907.

6 Axenfeld, T. *Die Aetiologie des Trachoms*, Jena, Gustav Fischer, 1914.

7 Lindner, K., in Berens, C. *The Eye and Its Diseases*, Philadelphia, W. B. Saunders Company, 1936, p. 437.

8 Taborisky, J. Die Prowazek-Halberstadterschen Körperchen und ihre klinische Bedeutung, *Arch. f. Ophth.* **124** 455, 1930.

9 Rotth, A. The Microbic Etiology of Trachoma, abstracted, *Rev. internat. du trachome* **15** 31, 1938.

10 Bengtson, I. A., and Rolufs, L. S. Observations on Epithelial-Cell Inclusions of Early Uncomplicated Trachoma, *Am. J. Ophth.* **19** 229, 1936.

mentary body, which requires mordants and special stains for demonstration, the elementary bodies of trachoma appear to have the general staining characteristics of the *Rickettsiae* with the Giemsa method they were demonstrable after staining with a dilute solution for as short a period as thirty minutes, the elementary bodies of vaccinia and molluscum contagiosum, treated similarly, remained unstained

On the other hand, the elementary bodies of trachoma are so like the granules of inclusion blennorrhoea and psittacosis in morphologic structure and staining reactions that a morphologic differentiation between them would seem to be impossible. A further point of resemblance between the granules of these three diseases, as well as a point of difference between them and the typical virus elementary body

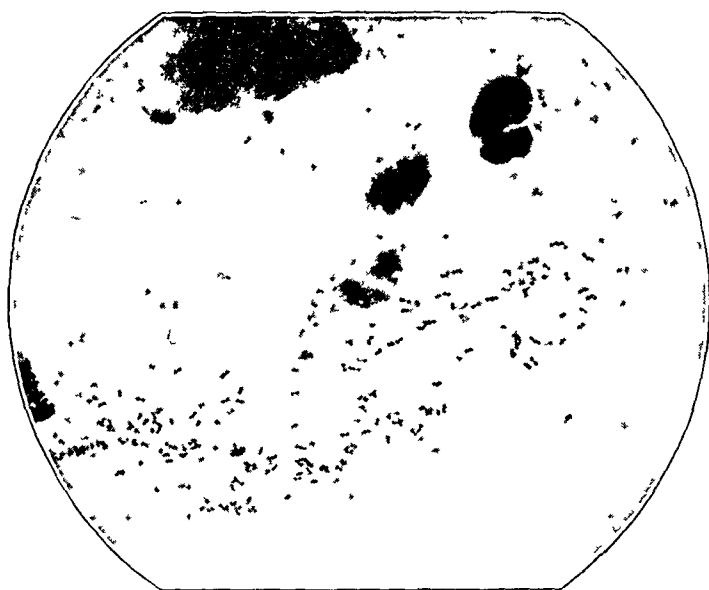


Fig 1—Free elementary bodies in an epithelial scraping from a patient with trachoma of high activity. Giemsa stain, $\times 2000$

(e g, of vaccinia), is the occurrence of large, coccobacillary forms (initial bodies), which take a pure blue stain as opposed to the reddish blue of the more numerous small forms

The minute bodies of trachoma form an inclusion body (fig 2) in the cytoplasm of the epithelial cells exactly like the inclusion body of inclusion blennorrhoea. Both have a glycogen-containing ground substance¹¹ (fig 3). The inclusion body of psittacosis, otherwise quite similar, contains no glycogen but in the young form possesses a basophilic-staining matrix

¹¹ Rice, C E. The Carbohydrate Matrix of the Epithelial-Cell Inclusion in Trachoma, *Am J Ophth* **19** 1, 1936. Thygeson, P. The Matrix of the Epithelial Cell Inclusion of Trachoma, *Am J Path* **14** 455, 1938.

On the basis of these observations it is believed that the elementary bodies of trachoma are virus bodies like those of psittacosis and inclusion blennorrhoea and that they are similar, except in staining reactions,

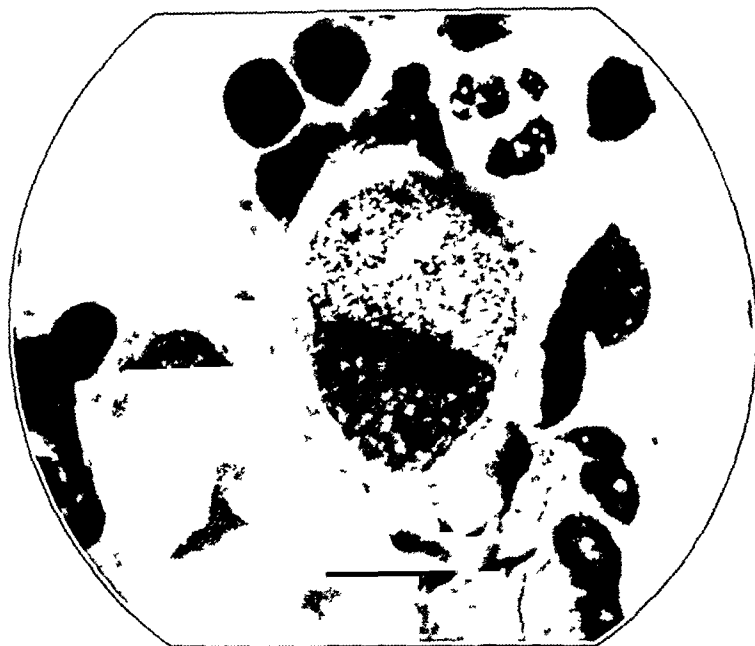


Fig 2—Epithelial cell inclusion body (Halberstadter-Prowazek) of trachoma Giemsa stain, $\times 2000$

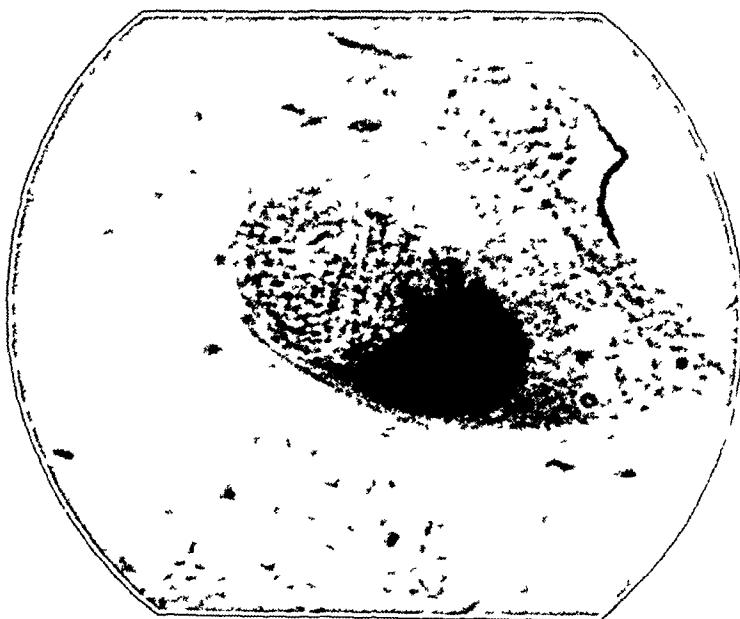


Fig 3—Inclusion body of trachoma stained with compound solution of iodine to show the presence of a glycogen-containing matrix, $\times 2000$

to typical virus elementary bodies, such as those of vaccinia, fowlpox and molluscum contagiosum

OCCURRENCE OF THE ELEMENTARY BODIES ON THE
CONJUNCTIVA

Giemsa-stained epithelial scrapings from 1,700 persons with conjunctivitis were examined for the presence of the elementary bodies of Halberstadter and Prowazek either free or as epithelial cell inclusions. They were found only in material from persons with inclusion blennorrhoea (61 patients) and with trachoma (320 patients). Since the material for study was derived from localities representative of widely separated sections of the country (New York, Iowa, Arizona and New Mexico), it would seem safe to assume that this type of elementary body occurs only in these two diseases in the United States.

FILTRABILITY OF THE ELEMENTARY BODIES OF TRACHOMA

Filter tests were made simultaneously with material from a patient with acute trachoma which exhibited relatively large numbers of elementary bodies and with a suspension of elementary bodies of molluscum contagiosum obtained by differential centrifugation of expressed material from the lesions of the disease. When the suspensions of trachomatous material were passed through Berkefeld V filters (small size, $\frac{3}{4}$ by $\frac{5}{8}$ inch [1.9 by 1.5 cm]) under ordinary conditions of filtration, no elementary bodies were demonstrable in the filtrate after centrifugation at high speed. This was equally true of the filtrates of molluscum contagiosum when the suspensions were diluted to approximately the same density as the suspensions of trachomatous material, with denser suspensions the elementary bodies were readily demonstrable in the centrifuged filtrate when suitably stained. The same experiment was performed with Elford graded collodion membranes of 0.6 micron average pore diameter and a filtration surface of approximately 0.6 sq cm. In this case elementary bodies were demonstrable in the filtrates from the suspensions of both trachomatous and molluscum contagiosum material.

It is evident, therefore, that elementary bodies of trachoma could have been present in the 6 positive filtrates of trachomatous material described earlier in this report.

OCCURRENCE OF ELEMENTARY BODIES IN AN INFECTIVE FILTRATE

The inoculation experiment, previously reported,³ in which a human volunteer (C. B.) was used to test the infectivity of a filtrate, served also to test the role of the elementary body. The experimental material consisted of pooled epithelial scrapings from 10 trachomatous Indian children (selected for activity from 164 trachomatous subjects) and was relatively rich in epithelial cell inclusions and free elementary

bodies After the material was ground and passed through rough filter paper to remove gross particles, the suspension was filtered through an Elford graded collodion membrane of 0.6 micron average pore diameter, and the filtrate was divided into equal parts, one-third for culture, one-third for inoculation and one-third for centrifugation at high speed The third used for culture was found to be free from bacteria, while sediment of the third used for centrifugation showed moderate numbers of elementary bodies Five days after the subject was inoculated, acute conjunctivitis developed, and epithelial cell inclusions and free elementary bodies were demonstrated in large numbers When the symptoms became chronic, about three weeks later, the inclusions and free bodies were demonstrable only in small numbers There was no secondary bacterial infection, and the disease ran the course of typical trachoma with pannus and scarring

RELATION OF THE ELEMENTARY BODY OF TRACHOMA TO THE ELEMENTARY BODY OF INCLUSION BLENNORRHEA

Inclusion blennorrhoea is now well established as a virus disease by virtue of the filtration experiments of Gebb,¹² Botteri,¹³ Thygeson,¹⁴ Tilden and Gifford¹⁵ and Julianelle¹⁶ It has been suggested that the elementary bodies of trachoma indicate merely a secondary infection with the virus of inclusion blennorrhoea and have nothing to do with the causation of the primary disease Since there is no morphologic means of differentiating the two types of elementary bodies nor any clinical means of differentiating the two diseases produced in monkeys, we have collected epidemiologic data in an effort to elucidate this question

Inclusion blennorrhoea is seen in this country principally in infants Of 50 cases studied, we have found evidence of spread in only 2, a proportion which indicates a low degree of communicability for the disease Inclusion blennorrhoea also occurs sporadically in adults, but the virus is in much lower concentration in the lesions In the 12 cases studied we found no evidence of transfer from one patient to another It seems¹⁴ that the disease does not become epidemic, that the infection

12 Gebb, H Experimentelle Untersuchungen über die Beziehungen zwischen Einschlussblennorrhoe und Trachom, *Ztschr f Augenh* **31** 475, 1914

13 Botteri, A Klinische, experimentelle und mikroskopische Studien über Trachom, Einschlussblennorrhoe und Frühjahrskatarrh, *Klin Monatsbl f Augenh* **50** 653, 1912

14 Thygeson, P The Etiology of Inclusion Blennorrhoea, *Am J Ophth* **17** 1019, 1934 Thygeson, P, and Mengert, W F The Virus of Inclusion Conjunctivitis Further Observations, *Arch Ophth* **15** 377 (March) 1936

15 Tilden, E B, and Gifford, S R Filtration Experiments with the Virus of Inclusion Blennorrhoea, *Arch Ophth* **16** 51 (July) 1936

16 Julianelle, L A Personal communication to the authors

of the infant takes place from the cervix of the mother and that the rare cases in adults occur, like gonorrhea, by direct transfer from the genitourinary tract to the eye. We have not observed the disease in epidemic form in swimming pools, but when this has occurred it has in all probability been a matter of transfer by genital secretions through the medium of the water to the eye.

These epidemiologic findings indicate that the inclusion virus could not possibly account for the high frequency of elementary bodies in trachoma. Furthermore, the occurrence of inclusion blennorrhea appears to be spotty. So far it has never been reported among the Indians of Arizona and New Mexico, although there the incidence of cases of trachoma in which inclusion bodies have been demonstrated is high. In Egypt the same situation holds. No case of inclusion blennorrhea has as yet been established there in spite of the high incidence of cases of trachoma in which inclusion bodies have been found. Of 65 cases of trachoma in Iowa in which bacteriologic studies were made,¹⁷ secondary infection with such organisms as *Diplococcus pneumoniae* and *Haemophilus influenzae* was found in approximately 50 per cent. Considering the fact that such bacteria are widespread and produce epidemic conjunctival infections, it seems unlikely that the much higher incidence of elementary bodies in trachoma (up to 100 per cent in the early and the acute stage) could possibly be accounted for by secondary infection with the relatively rare virus of inclusion blennorrhea.

This conclusion is shared by Roth,⁹ who has recently observed that inclusions cannot be demonstrated in a case of inclusion blennorrhea after the first few months of infection, whereas in a case of trachoma inclusion bodies may be demonstrated over a period of years.

FREQUENCY OF ELEMENTARY BODIES IN TRACHOMA

In table 2 are summarized the findings in 10 series of cases of trachoma in which examinations were made for inclusion bodies of Halberstadter and von Prowazek. In order to avoid error, only typical elementary body inclusions were included in the count. When one considers the difficulty of demonstrating the bodies due to their small size, to the lack of a cultural means of demonstration and to the chronicity of the disease, it would seem that the frequency reported here is consistent with etiologic significance. The results are comparable to the morphologic findings in other chronic diseases, such as tuberculosis, in which demonstration of the agent must usually be made by animal inoculation or culture. The finding of the bodies in all 12 of the only 12 cases of trachoma which were observed at the onset must be con-

17 Thøgerson, P. Unpublished data.

sidered significant. It is of interest in this connection to refer to the recent findings of Wilson,¹⁸ who demonstrated inclusions in 100 per cent of a series of 39 Egyptian infants observed at the onset of the disease, he had found them in considerably less than 50 per cent of patients with old established trachoma. It is our opinion that the elementary bodies are characteristic of active trachoma and that in any given case they can be demonstrated by repeated tests. We found them in the form of inclusions in 31 of 32 cases of chronic trachoma in which it was possible to make repeated examinations.

A good indication of the frequency of inclusions in trachomatous material is found in the results of the examination of the 107 Indian children (table 2). On the single slides examined per case there were

TABLE 2—Frequency of Halberstadter-Prowazek Inclusion Bodies in Cases of Trachoma

Description of Trachoma	Number of Cases	Number Positive for Inclusion Bodies	Percentage Positive for Inclusion Bodies
Trachoma at onset	12	12	100.0
Trachoma with acute or subacute symptoms, without superimposed bacterial infection	9	9	100.0
Chronic trachoma, stage IIa (repeated examinations in all cases)	32	31	95.6
Tunisian cases stage IIb, with superimposed bacterial infection	10	3	30.0
Tunisian cases stages I and IIa	28	11	39.3
Routine clinic cases, University of Iowa stages I, II and III (majority, stage III)	69	24	34.8
Indian children (Arizona, New Mexico) stages I, II and III, all with definite activity and all under treatment at time of examination (single slide examination)	107	9	54.8
Indian children (San Carlos, Ariz.) stages I, II and III, as in preceding group	18	4	22.2
Indian children (Fort Apache, Ariz.) stages I, II and III, as in preceding group	25	11	44.0
Trachoma, stage IV	10	0	0.0

no inclusions in 48 cases, only 1 inclusion in 16 cases, 2 inclusions in 14 cases, 3 in 4 cases, 4 in 3 cases, 5 in 2 cases, 6 in 2 cases, 7 in 1 case, 9 in 2 cases and numerous inclusions in 15 cases. In 1 of the 15 cases, a case of acute trachoma, 277 inclusions were found on one slide.

RELATION OF THE FREQUENCY OF THE ELEMENTARY BODIES TO THE DEGREE OF ACTIVITY OF THE TRACHOMA

In the 320 cases of trachoma which have been studied morphologically there has been in general a consistent relationship between the number of demonstrable elementary bodies and the degree of clinical activity of the disease as judged by the amount of conjunctival exudate. Thus, of 9 cases in which free elementary bodies were found in large

¹⁸ Wilson, R. P. in Eleventh Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo, Schindler's Press, 1937, p. 117.

numbers, acute symptoms were present in all but 1. This was a case of cicatricial trachoma of ten years' duration, there was a considerable amount of conjunctival secretion, but the conjunctiva was pale. In the cases of chronic trachoma of average duration the bodies were found consistently only in small numbers, and in cases of cicatricial and healed trachoma they were generally absent. There was also a definite correlation between the site of maximum concentration of inclusion bodies and the site of maximum intensity of the disease. While in cases of active trachoma inclusions could be demonstrated in all parts of the conjunctiva, including the limbal conjunctiva, they were almost invariably more abundant in scrapings from the retrotarsal fold, the point of maximum trachomatous activity.

OCCURRENCE OF ELEMENTARY BODIES IN CASES OF TRACHOMA OF LONG STANDING

Eight cases of trachoma have been studied in which the disease was known to have been present for over ten years. In 1 of these the disease was undergoing an acute exacerbation, and in spite of dense scarring inclusion bodies and free elementary bodies were found in large numbers. Preparations containing over 50 inclusion bodies per slide were observed repeatedly during the four days of study. In a second case in which there had been a subacute flare-up many inclusion bodies were also found. The condition in the remaining 6 was in the cicatricial stage, inclusion bodies were demonstrated, though in small numbers, in 5 of the 6 cases. It seems evident that the elementary bodies may persist throughout the active stage of the disease, even when it is prolonged.

ELEMENTARY BODIES IN HEALED TRACHOMA

In 10 cases of trachoma in which by all clinical criteria the lesion appeared to be healed, examinations were made for inclusion bodies. Repeated examinations of epithelial scrapings in each case failed to demonstrate them.

TRANSFER OF THE TRACHOMA ELEMENTARY BODIES

Trachomatous material containing inclusion bodies was transferred directly to 8 *Macacus rhesus* monkeys. Infection was obtained in all of them, though repeated inoculation was required in 5. The induced disease was mild and consisted of follicular hypertrophy without secretion or signs of inflammation. No free elementary bodies or inclusion bodies could be demonstrated with certainty in any of these animals even after repeated examination.

Trachomatous materials containing inclusion bodies were then transferred to 3 golden baboons. A follicular disease of considerable intensity developed, characterized by inflammation and secretion. In 2 animals a small number of characteristic inclusion bodies were demonstrable.

In a previously reported experiment¹⁹ trachomatous material containing inclusion bodies was transferred directly to the blind eye of a human volunteer. After an incubation period of five days, trachoma of subacute onset developed. There was no difficulty in demonstrating considerable numbers of both inclusion bodies and free elementary bodies in this experimental disease.

These experiments indicate that the bodies develop in the epithelial cells of the new host when transferred. It is of particular interest to note the ease with which they were demonstrated in the human subject and with what difficulty in the baboons. The failure to demonstrate them at all in the disease of the *Macacus rhesus* is believed to be due to the extraordinarily low intensity of the experimental disease in that animal.

RELATION OF THE INFECTIVITY OF CULTURES OF TRACHOMATOUS TISSUES TO THE PRESENCE OF THE ELEMENTARY BODIES

The experiments of Julianelle, Harrison and Morris²⁰ indicate that the virus of trachoma is not cultivable by ordinary methods of tissue culture. Our preliminary efforts have also given negative results. Repeated attempts have been made to grow the virus on the chorioallantoic membrane of the developing egg and in minced chick embryo tissues, with consistently negative results. We have obtained excellent growths of epithelium from normal human conjunctiva, but on ten attempts we have been unable either to show development of inclusion bodies in the inoculated tissue or to demonstrate infectivity of the tissue for monkeys. We have also been able to obtain excellent growths of epithelium and fibroblasts from material obtained from 14 patients with cicatricial trachoma and from 12 with follicular trachoma (showing epithelial cell inclusions), all Indian children. There was no development of inclusion bodies in the new growth nor demonstrable infectivity for monkeys.

LOCALIZATION OF THE ELEMENTARY BODIES IN THE EPITHELIUM

Our studies confirm the observations of other investigators that the localization of the elementary bodies and inclusions is strictly epithelial. In considering the question of the identity of the elementary bodies

¹⁹ Thygeson, P. Inoculation of Human Conjunctiva with Trachomatous Materials. *Am J Ophth* **16** 409, 1933.

²⁰ Julianelle, L. A., Harrison, R. W., and Morris, M. C. The Probable Nature of the Infectious Agent of Trachoma, *J Exper Med* **65** 735, 1937.

with the virus of trachoma it is of fundamental importance to determine whether or not the virus can multiply in the subepithelial tissues. In 1934 Michail and Vancea²¹ reported several observations in which trachomatous material of proved infectivity failed to produce a disease when inoculation was made through the skin of the lids without touching in any way the conjunctival epithelium. We have repeated this experiment with 6 baboons. In the first 2 a suspension of epithelial scrapings from a patient with trachoma which showed abundant inclusions was placed beneath the epithelium of the upper fornix by infection through the skin of the upper lid. A control test was made on a third baboon by simple deposition of the material on the conjunctiva followed by light friction. Lesions typical of experimental trachoma developed in the control animal, but the 2 baboons inoculated subepithelially were unaffected. Six weeks later susceptibility to trachoma was demonstrated for these 2 animals by inoculation of trachomatous material directly into the conjunctiva. Typical lesions developed after the usual incubation period of about eight days.

The fourth baboon was inoculated in the right eye by subepithelial injection of trachomatous material through the skin of the upper lid and in the left eye by inoculation directly on the conjunctiva. A typical experimental trachoma developed in the left eye after the usual incubation period, the right eye was unaffected. When this animal was examined five weeks after inoculation, however, both eyes were found to be involved. The infection of the right eye was probably the result of transfer from the left, but slow development of the lesion from the inoculation cannot be eliminated as a possibility.

In a fifth baboon similarly inoculated trachoma failed to develop, although the same material proved infectious for a sixth baboon by direct conjunctival inoculation.

These experiments are of fundamental importance and must be extended. All the evidence so far assembled points to the epithelium as the susceptible tissue.

EFFECT OF ANTITRACHOMATOUS THERAPY ON THE ELEMENTARY BODY

It is difficult to demonstrate the elementary bodies in patients with trachoma who have had daily treatment over long periods. Thus, at the Theodore Roosevelt Trachoma School, Fort Apache, Ariz., inclusions were demonstrated on a single slide of trachomatous material from only 7 of 151 children who had been receiving the usual types of treatment for seven months or longer.

²¹ Michail, D., and Vancea, P. Some Experimental Facts in Trachoma, *Rev. internat. du trachome* 9 33, 1932.

The recent observation of Loe²² that sulfanilamide has a rapid beneficial action in cases of trachoma led us, with W G Forster, to study the effect of the drug on the elementary bodies of trachoma. Accordingly, 10 Indian children with trachoma in the early stage were treated with average doses of the drug. Elementary bodies, in the form of epithelial cell inclusions, were demonstrated with relative ease in all the subjects prior to the onset of treatment. After fifteen days of treatment scrapings showed no inclusions. Examinations on the twenty-first and twenty-eighth days likewise gave negative results. Two untreated children with demonstrable inclusions, used as controls, still showed abundant inclusion bodies during the test period. These experiments will be reported in detail in a subsequent communication.

COMMENT

It is now generally recognized that the elementary bodies found in such virus diseases as vaccinia, fowlpox, molluscum contagiosum and psittacosis are parasitic in nature and constitute the agents of disease. Belief that the elementary body of trachoma represents the morphologic unit of the virus of trachoma rests, in our opinion, principally on the following findings: (1) the identity in morphologic structure and staining reactions of the bodies of trachoma with the similar bodies of inclusion blennorrhoea and psittacosis, established virus diseases, (2) the presence of the elementary bodies in the lesions of trachoma with sufficient constancy to indicate etiologic significance, (3) the presence of elementary bodies in an infective filtrate, (4) their multiplication in new hosts (man and baboon) when transferred directly or after filtration, and (5) their persistence in the lesions of trachoma throughout the period of activity of the disease.

The elementary body of trachoma has, in our opinion, the essential properties (filtrability, inclusion body formation and cytotropism) of a virus and should be classified, for the present at least, as a filtrable virus. It does, however, differ from the typical virus elementary body (such as that of vaccinia) in the ease with which it stains with Giemsa's stain and similar dyes and in its regular morphologic variation (elementary body, initial body). In these respects it resembles *Rickettsiae*, particularly *Rickettsia ruminantium* of Heartwater.²³

22 Loe, F. Sulfanilamide Treatment of Trachoma. Preliminary Report, to be published.

23 Jackson, C. The Microscopic Diagnosis of Heartwater, in Seventeenth Report of the Director of Veterinary Services and Animal Industry, Union of South Africa, August 1931, p. 161.

CONCLUSIONS

A series of studies on the etiology of trachoma are summarized and lead to the following conclusions concerning the agent (1) that it is filtrable under certain conditions, (2) that it has the characteristics of a virus (filtrability, inclusion body formation and noncultivability on nonliving mediums), and (3) that it is identical with the elementary body of Halberstadter and Prowazek

The virus of trachoma, with the viruses of inclusion blennorrhea and psittacosis, appear to form a group transitional between Rickettsiae and the typical viruses

ABSTRACT OF DISCUSSION

DR EDWIN WILLIAM SCHULTZ, Stanford University, Calif Investigations during recent years have definitely established that from the standpoint of size the filter-passing viruses range from easily visible bodies down to bodies measuring about 10 millimicrons, which is about twenty times as small as the smallest body one can see with an ordinary microscope Of the differences found to exist between parasitic bacteria and the so-called virus group, size is probably the least important A much more important difference is the marked cytotropism which characterizes the virus group and the absolute dependence of this group of agents on living tissue cells of one kind or another The high cell specificity of viruses, their intracellular relationship and especially their absolute dependence on living cells has evoked considerable speculation as to the exact nature of these agents It is not clear yet whether all the infectious agents classed under the heading of viruses are fundamentally related to each other or whether quite unrelated agents are included, but whatever the nature of some of these agents may be, it seems certain that some of the larger ones are organismal, at least in outward appearance Among these are the viruses responsible for pox diseases in animals, molluscum contagiosum, infective ectromelia of mice and psittacosis It is now established by ultrafiltration and by ultracentrifugation as well as by morphologic studies that the so-called elementary bodies of these virus diseases constitute the infective agents To this group of larger viruses evidently belongs the causal agent of trachoma

The difficulties experienced in demonstrating the filtrable nature of the virus of trachoma harmonize with the earlier experiences with some of the other larger filtrable viruses, especially with relation to the use of filter candles These difficulties have been largely surmounted by the use of collodion filters The fact that Dr Thygeson and Dr Richards obtained positive results when they used such membranes is, therefore, in harmony with experience with certain viruses The results of these filtration studies lend considerable support to the view expressed earlier by several investigators that the minute so-called elementary bodies which may be found in trachomatous material constitute the actual agent of this disease It is of considerable interest that Dr Thygeson and Dr Richards have found that these bodies in their staining reactions resemble Rickettsia somewhat more than the elementary bodies of vac-

cinia and molluscum contagiosum, which, together with the presence of coccobacillary initial bodies, suggests a relation to Rickettsia. This observation helps to tie together more closely the Rickettsia group and other filtrable viruses.

Dr Thygeson and Dr Richards have made an important contribution to the etiology of this disease.

DR HARRY S GRADLE, Chicago. It would be more than presumptuous for me to attempt to add to or detract from this excellent piece of investigative work. One unacquainted with the difficulties of animal experimentation in trachoma may have doubts as to what the experiments here cited actually prove and whether or not the authors have advanced one step further toward the fulfillment of Koch's postulates. It would appear to me that the answer is, "The case is not yet proved, but enough positive evidence has been added so that a solution may be in sight."

However, there exists considerable doubt in the minds of many whether or not the Prowazek bodies are the cause of trachoma or are even connected with the cause. As far back as 1910 Herzog advanced the idea that these bodies are merely the broken-down debris of attenuated gonococci and that trachoma is an aberrant form of that venereal disease. That idea was not sound. But at the International Congress of Ophthalmology, held in Cairo in December 1937, Grueter, of Marburg, came to the following conclusions:

"Inflamed trachomatous epithelial cells cannot be shown to contain any granular structures of a nature different from their own, whether in the Golgi zone, the interior reticulated body or in the cytoplasmic zone.

"A comparison of Heisberg's victoria blue stains with the results mentioned above, which were obtained with a number of my organic and inorganic cell toxins, in particular a comparison with epithelium conditions in trachoma, shows that the granular structures produced by many different techniques with all cell toxins are absolutely alike. They are seen particularly clearly when photographed in the dark field. It will then be seen again and again that the granules are primarily situated in the nodules, i. e., as the cell inflammation progresses, the mitochondrial granules become particularly clear. I cannot accordingly claim that the various granular structures described in the literature and tested by me by so many different methods are foreign substances, i. e., initial structures of a hitherto invisible disease germ in the cell.

"I hold the same view in regard to the finding of Busacca, who claims to have shown rickettsias in trachomatous epithelial cells on the strength of examination of cells in yellow fever. I consider that these trachoma rickettsias are inflammatory proliferations and divisions of granula which normally occur in the epithelial cells."

With such eminent authorities as the authors and Grueter presenting diametrically opposed views, what is the mere clinician to think? One must have an open mind and accept conclusive evidence such as Dr Thygeson and Dr Richards have presented. But until there is definite proof that Koch's postulates are fulfilled in toto, it cannot be said that the Prowazek bodies are the proved cause of trachoma.

DR PHILLIPS THYGESON, New York. We have considered the matter of the specificity of the inclusion bodies of Halberstadter and Prowa-

zek in great detail and believe that on the basis of the following findings these bodies must be considered of specific virus nature 1 They are not found in any bacterial or nonvirus type of conjunctivitis 2 They contain the two components typical of all specific cytoplasmic virus inclusions, i e, a matrix and the virus particles themselves 3 The virus components of the inclusions can be recognized outside the cells in the form of free elementary and initial bodies

Koch's postulates as applied to bacterial diseases are not applicable to virus diseases such as trachoma, since viruses cannot be grown in pure culture in the absence of living cells In virus diseases bacteria-free filtrates, which so far as can be determined contain no other agents, must be substituted for pure cultures These we have employed in our experiments, e g, in the experiment in which a human volunteer was employed and in which the elementary bodies of trachoma were demonstrated in the infective filtrate

AN EVALUATION OF HOMATROPINE-BENZEDRINE CYCLOPLEGIA

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Myerson and Thau¹ were the first to propose that benzedrine sulfate might be a useful adjunct to atropine in preparation of the eyes for refraction. The effects of benzedrine sulfate combined with homatropine hydrobromide were investigated by Beach and McAdams,² who concluded that the depth of cycloplegia obtained equaled that obtained by the usual method of administering homatropine but was of distinctly shorter duration. Further study of this subject was made by Powell and Hyde,³ who in their conclusions expressed entire agreement with Beach and McAdams.

The purpose of this study was to investigate cycloplegia produced with homatropine and benzedrine⁴ from the point of view of its depth and, further, to determine what part each drug contributes to the combined action.

Depth of cycloplegia is a problem which cannot be approached without an accepted method for its estimation. This is provided by the artificial myopia test. The fundamentals of the test are as follows:

- 1 The refraction of the eye under investigation has been determined.
- 2 The lens correcting the ametropia is in place before the eye.
- 3 To this correction is added a plus 3.00 diopter sphere.
- 4 The punctum remotum is measured by the use of 0.50 meter test type and a centimeter rule. The punctum remotum when so located falls at a point exactly 33 cm from the eye when the 6 meter correction is accurate, for then the eye has an artificial focus at this distance, otherwise the punctum remotum will not occupy this position.

There are certain inaccuracies innate in the artificial myopia test. The test object used is not ideal for its purpose, it intercepts an angle of 5 minutes at a distance of 50 cm, while at 33 cm it makes an angle

1 Myerson, A., and Thau, W. Human Autonomic Pharmacology. The Effect of Cholinergic and Adrenergic Drugs on the Eye, Arch Ophth 18:78 (July) 1937.

2 Beach, S. J., and McAdams, W. R. (a) Tr Am Ophth Soc 35:221, 1937, (b) Tr Am Acad Ophth 42:179, 1937.

3 Powell, L. S., and Hyde, M. E. (a) J Kansas M Soc 39:1 (Jan) 1938, (b) 39:57 (Feb) 1938.

4 The benzedrine sulfate used in this study was furnished by the manufacturer, Smith, Kline & French Laboratories, Philadelphia.

of 7.65 minutes. The ideal test object should be just visible to the normal eye at the artificial focus, where it should intercept an angle of 5 minutes. An object of ideal size is too small to be of practical utility, for it has to occupy a square each side of which measures 0.475 mm. Consequently, the larger object must be used and the inaccuracy which it imposes accepted. The resultant error should cause the test object to be recognized at a distance farther than 33 cm by the eye with normal or better visual acuity.

The punctum remotum will also vary, depending on the letters of which the test word is composed. Round letters are recognized with relative ease, while square letters are recognized with relative difficulty. Since most test words will contain letters of both forms, there is a tendency for the neutralization of the effect of this factor on the punctum remotum. For this reason it may be ignored.

Finally, there will be a variation in the location of the punctum remotum dependent on the mental attitude of the patient. The venturesome patient will read the test word at a greater distance than the cautious one. Individually, this factor is always operative, but in any larger number of tests it is collectively neutralized and may be ignored.

Since all of the test's inaccuracies except the effect of the size of the test type can be legitimately disregarded, the sum of them will cause the punctum remotum to be located at a distance greater than 33 cm from the eye. It would therefore appear that with cycloplegia complete the punctum remotum would be located at 33 cm or a distance greater than 33 cm, but never at a lesser distance. Similarly, with cycloplegia incomplete the action of the ciliary muscle induced by the effort to read the test word would add refractive power to the eye and so cause the punctum remotum to be located at a distance less than 33 cm.

This interpretation of the artificial myopia test is supported by experimental evidence presented by the data in table 1. In this table are listed data for 10 patients from 41 to 50 years of age on each of whom the artificial myopia test was carried out with the use of a cycloplegic and again at the postcycloplegic examination, which followed the former examination by not less than seven days. Three of these patients showed no difference in the punctum remotum. Of these 3, 1 required a reading addition of 2.25 diopters and 2 an addition of 2.00 diopters each. For 7 of the 10 patients the punctum remotum was found to be nearer at the postcycloplegic examination than at the cycloplegic examination and in each instance the distance was less than 33 cm. Of these 7 patients, 3 required no addition for reading, 1 an addition of 0.75 diopters, 2 an addition of 1.00 diopter each and 1 an addition of 2.00 diopters.

These data demonstrate that when the residual accommodation exceeds 1 diopter the artificial myopia test will show the punctum

remotum to be at a distance of less than 33 cm. Since there is general agreement that a residual accommodation not exceeding 1 diopter constitutes cycloplegia satisfactory for accurate refraction and since the artificial myopia test indicates exactly when this condition obtains, this test must be considered a clinically satisfactory measurement of the depth of cycloplegia.

A CLINICAL STUDY OF HOMATROPINE-BENZEDRINE CYCLOPLEGIA

Refraction was done for 25 private patients ranging in age from 18 to 57 years (average 32.6 years) one hour after the administration of 1 or 2 tablets containing $\frac{1}{50}$ grain (0.0013 Gm.) each of homatropine

TABLE 1—Results of the Artificial Myopia Test on Ten Patients

Age of Patient	Eye	Add	PR - H	PR - P	Difference
50	O D	2.25	33	33	0
	O S	2.25	33	33	0
42	O D	1.00	35	23	12
	O S	1.00	35	23	12
43	O D	1.00	33	28	5
	O S	1.00	33	28	5
46	O D	2.00	33	33	0
	O S	2.00	33	33	0
48	O D	2.00	33	33	0
	O S	2.00	33	33	0
49	O D	2.00	33	30	3
	O S	2.00	33	30	3
46	O D	0.75	33	28	5
	O S	0.75	33	28	5
42	O D	0.00	35	18	17
	O S	0.00	35	18	17
44	O D	0.00	33	25	8
	O S	0.00	33	25	8
41	O D	0.00	35	25	10
	O S	0.00	35	28	7

* Add refers to reading addition which is presented as indicative of the total accommodative ability of the patient, PR - H, to the punctum remotum measured in centimeters in the course of the artificial myopia test after refraction with homatropine hydrobromide cycloplegia, and PR - P, to the punctum remotum measured in centimeters in the course of the artificial myopia test at the postcycloplegic examination. All puncta remota are measured by Wells test type I (approximately 0.50 meter test type). The difference shows the number of centimeters by which the PR - H exceeds the PR - P.

hydrobromide and cocaine hydrochloride. In each instance completion of the refraction was followed by the artificial myopia test. The range in the puncta remota for both right and left eyes was from 33 to 40 cm. The average punctum remotum for the right eyes was 35.38 cm., and that for the left eyes was 35.42 cm. The difference between these average values was 0.04 cm.

Refraction was done with homatropine-benzedrine cycloplegia for 25 private patients ranging in age from 19 to 54 years (average, 33.9 years). The technic used was as follows:

(1) Conjunctival anesthetization by means of 2 drops of a 2 per cent solution of butyn.

(2) Instillation of 1 drop of a 5 per cent solution of homatropine hydrobromide.

(3) Instillation after from two to three minutes of 1 drop of a 1 per cent solution of benzedrine sulfate

(4) Compression of the canaliculi for not less than one minute after each instillation

(5) Refraction forty minutes after the last instillation

In each instance completion of the refraction was followed by the artificial myopia test. The range in the puncta remota for both right and left eyes was from 33 to 38 cm. The average punctum remotum for both right and left eyes was 34.48 cm.

Refraction was done for 25 private patients ranging in age from 18 to 52 years (average 28.8 years) after the administration in one eye of homatropine and in the other eye of homatropine and benzedrine. The technic follows:

A. Administration of homatropine

(1) Conjunctival anesthetization by means of 2 drops of a 2 per cent solution of butyn

(2) Instillation of 1 drop of a 5 per cent solution of homatropine hydrobromide at seven and one-half minute intervals

(a) A total of 4 drops for patients up to 40 years of age

(b) A total of 3 drops for patients from 40 to 50 years of age

(c) A total of 2 drops for patients from 50 to 60 years of age

(3) Compression of the canaliculi for not less than one minute after each instillation

(4) Refraction forty minutes after the last instillation

B. Administration of homatropine and benzedrine

(1) Conjunctival anesthetization by means of 2 drops of a 2 per cent solution of butyn

(2) Instillation of 1 drop of a 5 per cent solution of homatropine hydrobromine

(3) Instillation after two to three minutes of 1 drop of a 1 per cent solution of benzedrine sulfate

(4) Compression of the canaliculi for not less than one minute after each instillation

(5) Refraction forty minutes after the last instillation

In each instance completion of the refraction was followed by the artificial myopia test. Homatropine was used in 12 right eyes and 13 left eyes, homatropine and benzedrine, in 13 right eyes and 12 left eyes. The puncta remota ranged from 33 to 40 cm. for the eyes treated with homatropine and from 30 to 40 cm. for the eyes treated with homatropine and benzedrine. The average punctum remotum for the former was 35.42 cm. and for the latter, 35.29 cm. The difference between these values was 0.13 cm. It was observed that the pupillary diameters were greater in the eyes treated with homatropine and benzedrine than in those treated with the homatropine.

The eyes under homatropine cycloplegia (tablets) had an average punctum remotum with the artificial myopia test of 35.40 cm. The eyes under homatropine-benzedrine cycloplegia had an average punctum remotum of 34.48 cm with the artificial myopia test. The punctum remotum of eyes under homatropine-benzedrine cycloplegia was, on an average, 0.92 cm shorter than that of eyes under homatropine cycloplegia alone. However, since two different groups of subjects are considered, there is great likelihood that this difference is in great part accidental. Such accidental factors should be vastly reduced in number in the control group in which the two methods were used, one on each eye of the same person. This group had an average punctum remotum with homatropine-benzedrine cycloplegia which was nearer than that with homatropine cycloplegia by 0.13 cm. However, the right and the left eyes under homatropine cycloplegia showed a difference in the average punctum remotum of 0.04 cm. This suggests that even with the usual homatropine hydrobromide cycloplegia there is some variation in the punctum remotum with the artificial myopia test. With correction for this variation, it would appear that the eyes of the control group treated with homatropine and benzedrine had an average punctum remotum which was nearer than that for the eyes treated with homatropine hydrobromide by only 0.09 cm.

Further, it can be seen that all three clinical groups showed average puncta remota with the artificial myopia test which were well in excess of 33 cm. Therefore, it can be concluded that residual accommodation in all subjects was no greater than 1 diopter. Because residual accommodation in no patient exceeded 1 diopter, all patients may be said to have had cycloplegia satisfactory for refraction. There was, however, in the control group of patients a slight difference in the depth of cycloplegia in the eyes treated with homatropine and benzedrine as compared to that in those treated with homatropine. This difference is represented by a difference in the average punctum remotum of 0.09 cm which can at most indicate only a fraction of a diopter. It is therefore concluded that cycloplegia with homatropine and benzedrine is satisfactory for refraction and is only slightly less profound than cycloplegia produced by the usual technics of administering homatropine.

EXPERIMENTAL INVESTIGATION OF THE CYCLOPLEGIC EFFECT OF HOMATROPINE HYDROBROMIDE AND BENZEDRINE SULFATE

The purpose of the experimental procedures which were carried out on 15 patients at the clinic of the Indianapolis City Hospital was to determine the exact cycloplegic effect of a 5 per cent solution of homatro-

pine hydrobromide and a 1 per cent solution of benzedrine sulfate when used separately. An outline of the experiment follows.

- (1) Refraction with homatropine cycloplegia
 - (a) Instillation of 1 drop of a 2 per cent solution of homatropine hydrobromide in each eye every ten minutes for six instillations
 - (b) Artificial myopia test after refraction with homatropine cycloplegia
- (2) Examination not less than one week after refraction with homatropine cycloplegia
 - (a) Determination of the visual acuity through a 3 mm pinhole
 - (b) Measurement of the puncta proxima
 - (c) Anesthetization of the conjunctiva with 2 drops of a 2 per cent solution of butyn
 - (d) Instillation of the experimental drugs followed by compression of the canaliculi for not less than one minute
 - (1) Instillation of 1 drop of a 5 per cent solution of homatropine hydrobromide in the right eye
 - (2) Instillation of 1 drop of a 1 per cent solution of benzedrine sulfate in the left eye
 - (e) At fifteen and at thirty minutes after instillation of the drugs
 - (1) Determination of visual acuity through a 3 mm pinhole
 - (2) Measurement of the puncta proxima
 - (f) At forty minutes after instillation of the drugs
 - (1) Measurement of the pupillary diameters
 - (2) Refraction
 - (3) Artificial myopia test

The experimental data are recorded in their entirety in table 2. The visual acuity was taken in all instances through the 3 mm pinhole in order to exclude the influence of a variable pupillary area. Examination of the 15 right eyes fifteen minutes after the instillation of homatropine hydrobromide showed no significant change in the visual acuity of 14 of the eyes and a diminution in that of 1 eye. Examination of these eyes thirty minutes after the instillation of the drug showed no significant change in 8 eyes and a diminution of the visual acuity in 7. Examination of the left eyes fifteen minutes after the instillation of benzedrine sulfate showed no significant change in the visual acuity of 14 of the eyes and a diminution in the visual acuity of 1 eye. Thirty minutes after the instillation of the drug examination showed no significant change in the visual acuity of 12 eyes, diminished visual acuity in 1 eye and increased visual acuity in 2 eyes.

The accommodative power of all eyes at each stage of the experiment, expressed in diopters, is shown in table 3, the data for which were computed from table 2. Examination of the right eyes fifteen minutes after the instillation of homatropine hydrobromide showed no change in the accommodative power of 3 eyes, a decrease in that of 11 eyes and an increase in that of 1 eye. Thirty minutes after the instillation

TABLE 2—Results of Experimental Investigation of Cycloplegic Effect of Homatropine Hydrobromide and of Benzedrine Sulfate

After the Administration of the Experimental Drugs													
		15 Minutes			30 Minutes			10 Minutes					
Race	Sex	Cycloplegia with Homatropine Hydrobromide						Preceding Administration of Drugs		Pupil Diameter, etc.	PR		
		Age	Vision	Refraction	Vision	PR	Vision	PR	Vision			Refraction	Vision
W	M	26	O D	6/6-1	33/I	11.00 (-1.00)	6/6-2	15/I	18/I	6/9	23/I	6/6-1	33/I
			O S	6/6-2	33/I	+1.00 x .97	6/6-2	20/I	20/I	6/6-2	20/I	6/6-1	23/I
G	M	27	O D	6/6	33/I	+0.37	6/6	25/I	23/II	6/6	25/IV	6/6	33/I
			O S	6/6	33/I	+0.37	6/6	25/I	23/I	6/6	25/I	6/6	28/I
W	F	10	O D	6/6	33/I	+1.50	6/6	30/I	30/VII	6/6-2	30/I	6/5-2	33/I
			O S	6/6	33/I	+1.75	6/6	30/I	30/I	6/6-1	30/I	6/6-1	20/I
O	F	16	O D	6/6	33/I	+1.25	6/6	38/VI	31/VI	6/6-1	30/VII	6/6-1	33/I
			O S	6/6	33/I	+1.25	6/6-1	38/VI	28/VI	6/6-1	20/VI	6/6-1	30/I
O	M	27	O D	6/6	33/I	+0.50 (-0.62)	6/5	15/I	18/I	6/5	23/I	6/6	33/I
			O S	6/6-2	33/I	+0.37 x 180	6/6	15/I	15/I	6/6	15/I	6/6	18/I
W	F	31	O D	6/6	31/I	+0.50 x .97	6/6	20/I	33/II	6/9+2	20/VI	6/6	33/I
			O S	6/6	31/I	+0.50 (-0.12)	6/6-3	20/I	23/I	6/6-1	23/I	6/6	25/I
W	F	27	O D	6/6	28/I	+1.75 (-1.50)	6/12-1	15/I	15/I	6/12	15/I	6/9+3	20/I
			O S	6/6	33/I	+0.50 (-0.75)	6/6-1	15/I	15/I	6/6-1	15/I	6/6-1	18/I
O	F	35	O D	6/6	33/I	+2.00 (-1.20)	6/6-2	18/IV	23/IV	6/12	30/VII	6/6	33/I
			O S	6/6	33/I	+2.00	6/6-2	18/IV	18/IV	6/3-1	18/IV	6/6	25/I
W	M	33	O D	6/6	35/I	+5.00 (-1.50)	6/6	20/VII	0/VII	6/30	0/VII	6/6	33/I
			O S	6/6	35/I	+1.50 (-1.00)	6/6	15/VII	18/VII	6/9-2	18/VII	6/6	33/I
W	F	35	O D	6/6-1	33/I	+1.00 (-0.62)	6/9	20/I	22/I	6/12	18/VI	6/6-1	33/I
			O S	6/6-1	33/I	+0.62 x 120	6/6	18/I	15/I	6/6	18/I	6/6-1	25/I
O	F	25	O D	6/6	33/I	+1.75 (-1.50)	6/6-1	10/I	13/I	6/9	23/I	6/6	33/I
			O S	6/6	33/I	+1.50	6/6-1	10/I	10/I	6/6	10/I	6/6	25/I
W	F	25	O D	6/9	33/II	+1.75 (-1.00)	6/9	33/IV	28/VII	6/30	0/VII	6/6-1	33/I
			O S	6/6	35/I	+1.25 (-0.37)	6/6	28/II	23/II	6/9	33/II	6/6-1	33/I
O	F	21	O D	6/6	33/I	+0.75 (-0.75)	6/6	13/I	18/I	6/6	20/I	6/6	30/I
			O S	6/6	33/I	+1.00 (-1.00)	6/6	15/I	15/I	6/9	15/I	6/6	23/I
O	F	28	O D	6/6	33/I	+1.25 (-1.25)	6/9	22/I	22/I	6/9	22/I	6/6	33/I
			O S	6/6	33/I	+1.25	6/6	22/I	22/I	6/6	22/I	6/6	27/I
O	F	18	O D	6/6	33/I	+0.50 (-0.25)	6/6-1	10/I	10/I	6/9+2	15/I	6/6-1	33/I
			O S	6/6-1	33/I	+0.25 (-0.25)	6/9+2	10/I	10/I	6/9+1	10/I	6/6-2	30/I

* PR refers to the punctum remotum measured by the artificial myopia test. The punctum remotum (PR) and the punctum proximum (PP) were measured by the use of Wells test type and are expressed in centimeters. The Roman numerals indicate the test type. The pupillary diameters are expressed in millimeters. Refraction in the right eye was repeated forty minutes after the instillation of 1 drop of a 5 per cent solution of homatropine hydrobromide. Refraction in the left eye was repeated forty minutes after the instillation of 1 drop of a 1 per cent solution of benzedrine sulfate.

of the drug, only 2 eyes showed no change in the accommodative power, while 13 showed a decrease. Examination of the left eyes both fifteen and thirty minutes after the instillation of benzedrine sulfate showed no change in the accommodative power of 13 eyes, a decrease in that of 1 eye and an increase in that of 1 eye.

The pupillary diameters of 14 of the 15 patients were measured forty minutes after the administration of the experimental drugs and

TABLE 3—*Accommodative Power of All Eyes, Expressed in Diopters**

Eye	Preceding Administration of Drugs	15 Minutes After Administration of Drugs	30 Minutes After Administration of Drugs
O D	6 66	5 55	3 00
O S	5 00	5 00	5 00
O D	4 00	0 00	0 00
O S	4 00	4 00	4 00
O D	3 33	0 00	0 00
O S	3 33	3 33	3 33
O D	2 63	3 00	0 00
O S	2 63	3 50	5 00
O D	6 66	5 55	4 35
O S	6 66	6 66	6 66
O D	5 00	0 00	0 00
O S	5 00	4 24	4 24
O D	6 66	6 66	6 66
O S	6 66	6 66	6 66
O D	5 55	3 50	0 00
O S	5 55	5 55	5 55
O D	5 00	0 00	0 00
O S	5 55	5 55	5 55
O D	5 00	4 54	0 00
O S	5 55	5 55	5 55
O D	10 00	7 69	4 24
O S	10 00	10 00	10 00
O D	3 00	0 00	0 00
O S	3 50	3 50	3 00
O D	7 69	5 55	5 00
O S	6 66	6 66	6 66
O D	4 54	4 54	4 54
O S	4 54	4 54	4 54
O D	10 00	10 00	6 66
O S	10 00	10 00	10 00

* The accommodative powers have been computed from the puncta proxima recorded in table 2. When the punctum proximum recorded in table 2 had receded beyond 40 cm with the same test type, the accommodation was recorded here as zero.

just prior to refraction. The diameters of the pupils of the eyes treated with homatropine were equal to those of the eyes treated with benzedrine in 3 instances. The pupillary diameters of the eyes treated with homatropine were greater than those of the eyes treated with benzedrine in 11 instances. In no instance was the pupillary diameter of an eye treated with benzedrine larger than that of an eye treated with homatropine. The average increase in the pupillary diameter of the eyes treated with homatropine over those of the eyes treated with benzedrine was 1.71 mm.

In table 4 are compared the results of refraction with the usual homatropine hydrobromide cycloplegia with the results of refraction with cycloplegia produced by 1 drop of a 5 per cent solution of homatropine hydrobromide and 1 drop of a 1 per cent solution of benzedrine sulfate. The data are computed from those recorded in table 2. Of the eyes treated with homatropine, 7 showed no decrease in the hyperopia uncovered in the greater meridian, while the remaining 8 showed a decrease ranging from 0.12 to 0.75 diopters. The average loss in hyperopia uncovered in the greater meridian was 0.19 diopters. The efficiency of refraction with cycloplegia produced with a 5 per cent solu-

TABLE 4—Results of Refraction with Two Types of Cycloplegia *

Diopters Loss of Hyperopia		Cylinder Axis Deviation		Difference in PR, Cm	
O D	O S	O D	O S	O D	O S
0.00	2.00	7.0	8.0	0	10
0.25	0.62	No cylinder		0	5
0.00	1.25	No cylinder		0	13
0.00	0.50	No cylinder		0	3
0.12	0.75	0.0	0.0	3	15
0.00	1.00	7.0	82.0	1	9
0.50	2.00	0.0	90.0	8	15
0.00	0.62	0.0	No cyl	0	8
0.00	0.50	No cylinder	0.0	2	2
0.37	0.50	0.0	No cyl	0	8
0.12	0.75	No cylinder		0	5
0.00	1.12	0.0	0.0	0	2
0.75	1.25	0.0	0.0	3	10
0.50	0.75	15.0	No cyl	0	6
0.25	0.50	30.0	Cyl lost	0	3

* All data in this table were computed from those in table 2. The decrease in hyperopia was computed by subtracting the hyperopia in the greater meridian uncovered by refraction with cycloplegia produced with a 5 per cent solution of homatropine hydrobromide or with a 1 per cent solution of benzedrine sulfate from that uncovered in the greater meridian by refraction with the usual homatropine hydrobromide cycloplegia. The cylinder axis deviation shows the deviation in degrees of the cylinder axis found by refraction with cycloplegia produced with a 5 per cent solution of homatropine hydrobromide or with a 1 per cent solution of benzedrine sulfate from the axis determined by refraction with the usual homatropine hydrobromide cycloplegia. The difference in the punctum remotum shows the number of centimeters by which the punctum remotum measured with the artificial myopia test after refraction under the usual homatropine hydrobromide cycloplegia exceeded that found by refraction with cycloplegia produced with a 5 per cent solution of homatropine hydrobromide and with a 1 per cent solution of benzedrine sulfate.

tion of homatropine hydrobromide as compared with that of refraction with the usual homatropine hydrobromide cycloplegia was 90 per cent. All eyes treated with benzedrine sulfate showed a decrease in the hyperopia uncovered in the greater meridian. The decrease ranged from 0.50 to 2.00 diopters, and the average loss was 0.94 diopters. The efficiency of refraction with cycloplegia produced with a 1 per cent solution of benzedrine sulfate as compared with that of refraction with the usual homatropine hydrobromide cycloplegia was 46 per cent.

Investigation of the change in cylinder axis showed that with a 5 per cent solution of homatropine hydrobromide the axis for each of 10 patients was found where it was previously located by refraction with the usual homatropine hydrobromide cycloplegia. Six of these 10 patients showed no change in the cylinder axis. Four showed a

change of from 7 to 30 degrees. The average deviation in axis for the 4 patients was 14.75 degrees. With refraction with cycloplegia produced with a 1 per cent solution of benzedrine sulfate the axis for 7 of 8 patients was found where it was previously located by refraction with the usual homatropine hydrobromide cycloplegia. Four of the patients showed no change in the cylinder axis. Three showed a change in axis ranging from 8 to 90 degrees, with an average of 60 degrees.

Comparison of the punctum remotum shown by the artificial myopia test at the time of refraction with the usual homatropine hydrobromide cycloplegia with that found at refraction with cycloplegia produced by a 5 per cent solution of homatropine hydrobromide revealed no change in the position of the punctum remotum for 10 of the 15 patients. For the 5 remaining patients the punctum remotum was decreased. The range of decrease was from 1 to 8 cm. The average decrease for these 5 patients was 3.40 cm. Comparison of the punctum remotum shown by the artificial myopia test at the time of refraction with the usual homatropine hydrobromide cycloplegia with that found at refraction with cycloplegia produced by a 1 per cent solution of benzedrine sulfate showed a decrease of the punctum remotum for all of the 15 patients. The decrease ranged from 2 to 15 cm. The average decrease for the 15 patients was 7.73 cm.

These data lead to certain conclusions concerning the two drugs under investigation. First to be considered will be the 5 per cent solution of homatropine hydrobromide.

- 1 Homatropine hydrobromide in 5 per cent solution is an effective cycloplegic, for it decreases the visual acuity, definitely diminishes the accommodative power and provides cycloplegia of such depth as to uncover 90 per cent of the hyperopia in the greater meridian, reveal the correct cylinder axis and leave the eye with little residual accommodation, as evidenced by the artificial myopia test.

- 2 As a mydriatic, a 5 per cent solution of homatropine hydrobromide has, forty minutes after instillation, a greater effectiveness than a 1 per cent solution of benzedrine sulfate, for 11 of 14 patients showed a greater pupillary diameter in the eye treated with homatropine hydrobromide, and in no instance was the pupillary diameter of the eye treated with the benzedrine sulfate greater than that of the eye treated with homatropine hydrobromide.

Second to be considered are conclusions concerning a 1 per cent solution of benzedrine sulfate.

- 1 The 1 per cent solution of benzedrine sulfate has no cycloplegic effect whatever, for it does not diminish the visual acuity nor does it decrease the accommodative power, under its influence refraction is utterly unreliable, as it uncovers only 46 per cent of the hyperopia in

the greater meridian, shows marked variation in the cylinder axis which it reveals and leaves the eye with a residual accommodation of high degree, as demonstrated by the artificial myopia test

2 As a mydiatic, a 1 per cent solution of benzedrine sulfate has, forty minutes after instillation, a lesser power than a 5 per cent solution of homatropine hydrobromide, for the pupillary diameters of 11 patients were less with the use of the former than with the use of the latter, 3 patients had equal pupillary diameters, and in none was the pupillary diameter of the eye treated with benzedrine greater than that of the eye treated with homatropine

Last to be considered are conclusions concerning the combined use of a 5 per cent solution of homatropine hydrobromide and a 1 per cent solution of benzedrine sulfate to produce cycloplegia

1 These drugs have no synergistic action as regards the production of cycloplegia. It has been shown that a 5 per cent solution of homatropine hydrobromide is sufficient to produce satisfactory cycloplegia for routine refraction, while a 1 per cent solution of benzedrine sulfate has been shown to have no cycloplegic effect whatever. Accordingly, it cannot be expected that a noncycloplegic drug can, by addition, augment the effect of a true cycloplegic. Moreover, the depth of the cycloplegia obtained with this combination of drugs is not necessarily the result of the combination. Indeed, this may be most doubtful when it is known that the cycloplegic element of the combination is by itself able to produce the depth of cycloplegia observed

2 A synergistic relation between these two drugs does exist to produce mydriasis. A 5 per cent solution of homatropine hydrobromide has been demonstrated to be a potent mydiatic. A 1 per cent solution of benzedrine sulfate has been shown to be a less active mydiatic. Hence, these two drugs combined might reasonably be expected to produce greater mydriasis than either alone. Indeed, this synergy in mydiatic effect has been observed in the course of refraction with homatropine-benzedrine cycloplegia

3 Previous workers, judged wholly by their published reports, have been in error in their belief in a synergistic action of homatropine and benzedrine to produce cycloplegia. This belief has no basis in experimental observation. These authors noted the synergistic effects of these drugs to produce mydriasis, they observed correctly that cycloplegia under their influence was profound and of short duration and enabled satisfactory refraction to be performed, so they came to the erroneous conclusion that these drugs used in combination acted in a synergistic fashion to produce cycloplegia. This conclusion is false, because it was arrived at without consideration of the fact that the homatropine constituent alone suffices to produce the observed depth of cycloplegia

4 The use of a 5 per cent solution of homatropine hydrobromide and a 1 per cent solution of benzedrine sulfate in combination to produce cycloplegia for refraction has been an asset to the art of refraction and a great convenience to the patient. By the use of this drug combination, it has been demonstrated that cycloplegia satisfactory for refraction is possible of attainment with the use of a greatly reduced dose of homatropine. The use of this smaller dose has resulted in much more rapid recovery from cycloplegia. Rapid recovery follows the administration of a dose of homatropine which is just sufficient in amount to give after forty minutes a concentration in the ciliary muscle which merely suffices to produce complete cycloplegia. Since recovery from cycloplegic effect does not involve the elimination of any excess of drug, it is correspondingly rapid. A number of private patients have reported recovery from homatropine-benzedrine cycloplegia in from five to eight hours.

In conclusion, it may be emphasized that the use of a 5 per cent solution of homatropine hydrobromide combined with a 1 per cent solution of benzedrine sulfate for the production of cycloplegia in refraction can be recommended (1) because of the complete cycloplegia induced by 1 drop of a 5 per cent solution of homatropine hydrobromide, (2) because of the increased diameter of the pupil, which is dependent on the synergistic action between the two drugs to produce mydriasis, and which is an aid in the performance of retinoscopic examination, and (3) because of the short (five to eight hour) duration of the cycloplegia developed by such a minute dose of homatropine.

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A CATARACT OPERATION TO REDUCE THE INCIDENCE OF PROLAPSE OF THE IRIS

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Of the many pitfalls confronting one in the performance of operations for cataract, I dare say none gives more concern, especially in the simple extraction of cataract, than does prolapse of the iris. A prolapsed iris may be simply an annoyance, or it may be a grave source of danger to the well-being of the eye. Delayed healing of the wound, inflammatory reactions, keratitis, a kinked cornea with resultant haze and even sympathetic involvement of the other eye all seem to be lurking about to make the lot of the ophthalmic surgeon a not too happy one.

Every one at some time has the urge to try something foreign to his everyday methods, and while in such a mood about seven years ago I decided to perform some simple extractions, having in mind mental pictures of this procedure as done by my former chiefs, Drs Fisher, Berens, Schwenk, Risley, Harlan, Oliver, Thomson, Ziegler and others, during my term of internship at the Wills Hospital thirty-eight years ago. All these operators had their bad moments at some time or other, but much excellent work was done in those days with simple extraction of cataract. No conjunctival flaps were employed. The incision was made through a clear cornea, the knife coming out from 1.5 to 2 mm below the upper limbus. This permitted an easier exit for the cataractous lens, but a longer time was required for healing, with the result that an empty anterior chamber with leakage of the aqueous was accepted as a matter of course. However, the end results compared favorably with those of the complicated techniques used by so many operators today, employing hemstitching and large, bloody conjunctival flaps.

My opinion is that there is a tendency today to make operations for cataract more and more complicated instead of endeavoring to simplify them.

To return from this little digression, too many prolapses occurred during my season of simple extractions, and the procedure I was using was abandoned. However, when I was conning my results at a later date, the thought arose that there must be some potential cause for these prolapses and that possibly some of them could be blamed directly on

Read by invitation before the College of Physicians of Philadelphia, Section on Ophthalmology, April 21, 1938

the operative method or technic rather than on unforeseen circumstances. It is known that a restless patient always favors prolapse of the iris, but it is also known that some of the best behaved patients are the subject of this unfortunate occurrence.

In recalling some of these annoying prolapses, I was impressed by the fact that so many occurred up and in at about 11 o'clock in the left eye and up and in at approximately 1 o'clock in the right eye. All the prolapses did not occur here, but the great majority did. Why did not these prolapses take place directly above, as one would expect? Why did there seem to be a tendency for the majority of them to occur, or at least to start, approximately at the same spot? It certainly appeared to indicate that something was being done to favor this occur-

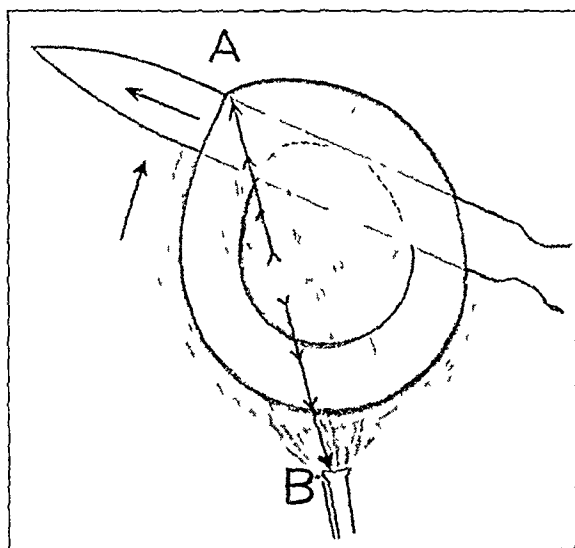


Fig 1—Counterpuncture has been performed, and the knife is being thrust upward and outward, dragging and stretching the cornea and iris peripherally. Tissues are being stretched in line from *A* to *B*, that is, between the point of fixation and the cutting edge of the knife.

rence at the time of operation. As a consequence, whenever an extraction was done an effort was made to determine just what was going on. However, a study of some motion picture films of cataract extraction which could be stopped at desired intervals for a still picture seemed to reveal a reason for the tendency of the iris to prolapse. There appeared to be little doubt but that the manner of making the incision plus possibly the use of a none too sharp knife had much to do with the resulting prolapse.

The customary incision is made by thrusting the knife through the corneal limbus, crossing the anterior chamber and then making the counterpuncture. Immediately afterward the point end of the knife is carried upward as it is pushed through, completing a sizable cut. As

the knife is withdrawn, the heel is raised, completing another cut at the opposite limbus. This is repeated until the section is finished. More sweeps are made before the knife finally emerges through the cornea above, depending on the operator and the sharpness of the knife. If one views the motion picture film and stops it at the proper place during projection, it will be found that the cornea while being cut after the counterpuncture is pulled and stretched into an oval shape, with the long axis of the oval at approximately 115 degrees as the knife is pushed along and at about 70 degrees as it is being withdrawn. The duller the knife, the more pronounced is this stretching.

One cannot help but deduct that there must be a severe stretch and pull on the cornea, and, in my opinion, on the associated structures as well, namely, the iris tissue and the base of the iris. It is difficult to

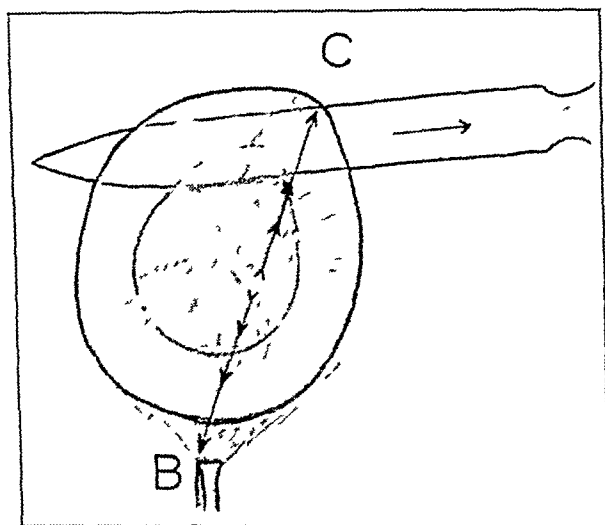


Fig 2—The knife is being withdrawn, causing stretching between *B* and *C*

imagine the cornea's being distorted at the limbus without some accompanying associated condition in the iris as well. When one considers that the eyeball is held by means of a fixation forceps below the lower limbus and that a strong pull is being exerted against this fixation by the engaged knife, there must, of necessity, be some stretching or tendency to stretching of the cornea and base of the iris while the section is being completed. The force of the cutting is upward and outward from the pupillary center as the knife is pushed through and pulled back. The structures are being stretched peripherally. The base of the iris as well as the iris is being dragged outward as well as upward and no doubt is weakened sufficiently in some cases, especially when low tonicity is present, to allow the iris to drift out through the open wound after it is bandaged if it does not emerge before. What better condition could one ask for to favor prolapse of the iris? This, to my mind, is one of the main reasons for its occurrence.

My opinions, of course are theoretic, but one may easily visualize what occurs when one sees a thoroughly, ovally distorted cornea during cataract extraction, with the fixation forceps pulling one way and the cataract knife opposing this traction, while between lie the stretched-out cornea and iris

One must not lose sight of the fact that as the cataractous lens emerges through the pupil, during a simple extraction, a certain amount of stretching of the iris tissues must of necessity take place, just as the perineum gives to permit passage of the head. This must cause more or less weakening of the contractile powers of the iris tissue, thereby favoring prolapse. The greater the tone of the iris, the less the chance of prolapse.

I have tried peripheral iridotomy and iridectomy but found these not to be the answer in preventing prolapse, although the push from

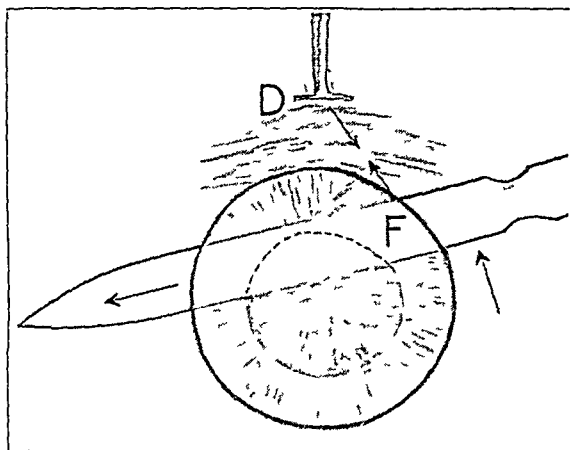


Fig 3—Counterpuncture is being made, and the heel of the knife is raised in cutting. The tissues are pushed inward. There is compression of the tissues between *F* and *D*. No stretching occurs below the knife. (Compare this technic with that shown in figure 1.)

the aqueous beneath must be lessened thereby. If the iris structure has been distended and distorted and has lost tone, my opinion is that it would require more than a peripheral iridectomy to keep this pupillary contour intact.

The diminishing of the contractile tone of the iris fibers is directly affected by the passage of the lens through the pupil and also by operative traumatism in making the section. It is manifestly impossible to do away with the stretching caused by the passage of the lens during simple extraction, but it is possible to reduce the resulting traumatism from a forced incision which drags the tissues outward at each push or pull of the cataract knife.

One must not ignore the previously mentioned excess drag caused by a dull knife, and any manner of incising a cornea must produce more traumatism and damage to the structures involved when a dull knife is used

As to drugs, physostigmine salicylate may be instilled immediately after extraction, but beneficial action from its use cannot be depended on. However, there is no harm in giving it the benefit of the doubt. A favorite method of Dr. Risley was to instill both atropine sulfate and physostigmine salicylate on completion of a simple extraction, in the hope of obtaining a contractile action from the latter drug and later a dilative action from the atropine.

Working on the observations as previously expressed, I had the idea that possibly some of the traumatism to the iris during the section might be lessened. I think this has been accomplished in a measure

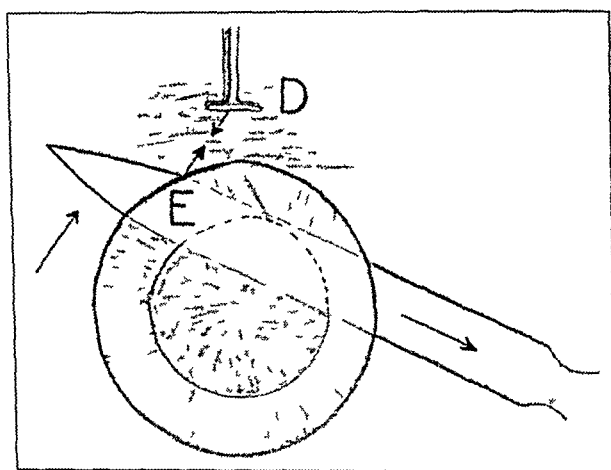


Fig. 4—The knife is being pulled back, with the point end raised. Tissues are drawn inward. There is compression between *E* and *D*. No drag occurs below the knife.

by making the section in reverse of the conventional manner and by changing the manner of fixation with the forceps.

To eliminate as much as possible the opposing pull between forceps and knife, the fixation is made about 5 mm above the upper limbus, the wide-jawed Luer forceps being used. The patient is asked to look down, and a deep bite is made down to the resistant sclera. With the eyeball fixed in this manner, any cutting that is subsequently done results in pushing or compressing the intervening tissues rather than in stretching them into a state of paresis, as partly occurs when fixation is made below the lower limbus. As the knife goes upward, the force is made against the point of fixation and not in opposition to it. This is one point. Now to reduce the peripheral stretching of the tissues as the incision is made.

This is partly accomplished by reversing the manner of making the incision. After the puncture is made in the regular way, in the same manner that counterpuncture is made, the point of the knife is carried through instead of being pushed upward and inward toward the nose (in case of the left eye), as in the customary manner, and at the same time the heel of the knife is raised, a cut being made upward through the limbus with the force directed inward toward the pupillary area. When the knife is being withdrawn, the point end is raised, and the cut is also made upward, the force still being toward the pupillary center. The succeeding sweeps are made in the same way, completing the section. In this manner the force of the cutting tends to push the tissues toward the pupil rather than dragging them peripherally.

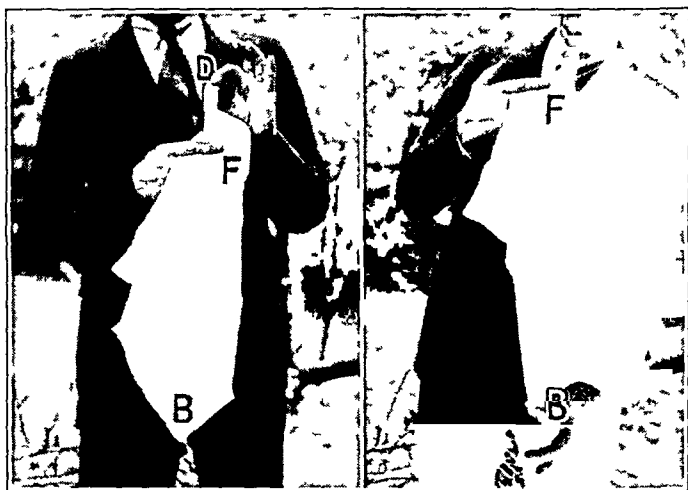


Fig 5—Illustration of the principle involved by fixation above and below. The handkerchief represents the vertical meridian of the cornea and the adjacent structures. On the left fixation is at *D* (above the upper limbus). The point of upward pressure of the knife is at *F*. Compression occurs between *D* and *F*, but there is no disturbance between *F* and *B*. On the right fixation is below at *B*. The knife push is at the same point *F*. Note the stretching of the whole structure between *B* and *F*.

This manner of making the incision will be found to be somewhat awkward at first, but one soon gets the knack of it, and it is just as easy to accomplish as the conventional section.

Grasping the globe above the upper limbus probably would not be greeted with delight by those who find it necessary to make large, bloody conjunctival flaps, but as I do not care for these embellishments, including the complicating sutures employed by many, the procedure is in no way a hindrance. My preference is for simply a small fringe of conjunctiva. I make no attempt to scalp the eyeball, so to speak, believing the less traumatism the better, and in my own work I have found no reason whatever to complicate my extractions by suturing.

One must not conclude from these remarks that prolapse of the iris has been done away with. It has not, as too many other factors enter into the situation. However, I think I can safely say that it is possible to reduce its incidence.

DISCUSSION

DR H. MAXWELL LANGDON. I wonder if Dr. Parker has considered the use of a bridge suture under the superior rectus muscle. It seems to me that it would not be so much in the way as a forceps in this position, and I think that it would give good counterpressure against the knife. I must confess that I do not see just how reversing the usual procedure in making the cut exerts less drag on the cornea. If the point or the heel of the knife is raised first, the other portion of the knife must make the next step in the procedure, and it seems to me that the drag would be about the same.

DR FRANCIS HEED ADLER. I should like to ask Dr. Parker if he has noticed any decrease in the amount of striate keratitis in his cases with the present method of making the section. I cannot understand how his method would diminish the incidence of prolapse of the iris, but I can believe that it might materially reduce the amount of striate keratitis.

DR FRANK C. PARKER. In answer to Dr. Langdon's question, I have never used this manner of fixation, as it requires a pull on the globe before fixation comes into play. With an open wound such as is present after extraction, this does not appeal to me. Again, fixation with a forceps does away with any "swaying" of the eyeball. Further, the placing of this suture adds to the complication of the procedure and the time consumed in performing it.

In answer to Dr. Adler's question, I have not seen any striate keratitis for some time. Whether or not this can be attributed to the lessened "drag" in this incision, I am not prepared to say. However, one might easily deduct that with diminished stretching of the cornea, striate keratitis would be kept at a minimum.

INDUCED SIZE EFFECT

I A NEW PHENOMENON IN BINOCULAR SPACE PERCEPTION ASSOCIATED WITH THE RELATIVE SIZES OF THE IMAGES OF THE TWO EYES

KENNETH N OGLE, PH D

HANOVER, N H

Probably one of the most important of the visual clues by which a person can estimate the relative distances and positions of objects in space is stereoscopic localization. These clues arise from the disparities between the two retinal images caused by the slightly different points of view of the two eyes. That stereopsis is the means for the most accurate discrimination of the relative distances of objects is, of course, universally appreciated. It has not been sufficiently recognized, however, that stereoscopic vision also plays an important role in the subjective orientation of all objects in the entire binocular visual field, in the sense of a rotation about the point of fixation. In the absence of strong perspective clues, this particular capacity may provide one of the important visual criteria by which a person can orient himself in the outside world of objects.

Certain experiments which demonstrate a relation between this type of subjective orientation and the relative sizes of the ocular images in the two eyes have been described by A. Ames Jr.¹ These experiments indicated that a false subjective orientation may result from increasing the size of the image in one eye. For example, if a person with good stereoscopic vision looks down on a grass lawn on which there are few perspective clues and a difference in the sizes of the retinal images is introduced by a size lens before one eye, he will see the lawn tipped abnormally, according to the nature of the size lens. In general, with any change in the relative sizes of the images, different stereoscopic depth values relative to the fixation point will be associated with the images of all object points seen binocularly. The result is that the whole of the binocular field is falsely oriented about the fixation point. This phenomenon is obviously related to the rotations of the apparent frontal

From the Division of Research in Physiological Optics, the Dartmouth Eye Institute of the Dartmouth Medical School

1 Ames, A., Jr. Aniseikonia. A Factor in the Functioning of Vision, *Am J Ophth* 18 1014, 1935

plane, the nonius horopter and the region of binocular single vision,² when differences are introduced between the sizes of the retinal images

In the early studies of the effects of size lenses, it was found that an apparent rotation of surfaces seen binocularly resulted not only from increasing the size of the image in one eye in the horizontal meridian but also from increasing it in the vertical meridian. The fact that an apparent rotation occurs as a result of a difference in the size of the images in the vertical meridian constitutes a new and unusual phenomenon, which will be described as the "induced size effect" and to which special emphasis will be given.

The apparent rotation that takes place about a vertical axis when the size of the image in one eye is increased in the horizontal meridian would be expected for the following reasons. In binocular vision the stereoscopic function, as far as is known, exists only in the horizontal meridian. Since objects occupying different positions in space are imaged on the retinas of the two eyes on horizontally disparate points, a change in the size of the retinal image of one eye in the horizontal meridian changes the relative stereoscopic disparities of all those images. Accordingly, the subjective values associated with all the fused images of the objects will be changed. The result is a new subjective orientation of the objects from their original positions, as though the whole binocular visual field had been rotated about a vertical axis through the fixation point, the objects on the side of the eye having the smaller image appearing nearer and those on the side of the eye having the larger image appearing farther away. Such an apparent rotation is a natural result of the particular geometric arrangement of the two eyes with respect to the positions of objects seen in space, and the effective changes of that arrangement caused by the size lens.³ On the basis of the analytic horopter theory, the angle of this apparent rotation can be computed from the equation $^{2b,d} \tan \psi' = - \frac{M-1}{M+1} \frac{b}{a}$, in which ψ' is the angle of apparent rotation, M is the magnification of the size lens placed before the left eye in the horizontal meridian, b is the visual

2 (a) Ames, A., Jr., Ogle, K. N., and Gliddon, G. H. Corresponding Retinal Points, Horopter and Size and Shape of Ocular Images, *J. Optic. Soc. America* **22** 619, 1932. (b) Ogle, K. N. Analytical Treatment of the Longitudinal Horopter. Its Measurement and Application to Related Phenomena, Especially to the Relative Size and Shape of Ocular Images, *ibid.* **22** 665, 1932. (c) Herzau, W., and Ogle, K. N. Ueber den Grossenunterschied der Bilder beider Augen bei asymmetrischer Konvergenz und seine Bedeutung für das zweiaugige Sehen, Ein Beitrag zur "Aniseikonia"—Forschung, *Arch. f. Ophth.* **137** 327, 1937. (d) Ogle, K. N. Die mathematische Analyse des Langshoropters, *Arch. f. d. ges. Physiol.* **239** 748, 1938.

3 Ogle, K. N. The Correction of Aniseikonia with Ophthalmic Lenses, *J. Optic. Soc. America* **26** 235, 1936.

distance of the fixation point from the midpoint of the interpupillary base line and a is one-half the interpupillary distance. This relation for the apparent frontal plane, the nonius horopter and the region of binocular single vision has been verified experimentally on the longitudinal horopter apparatus. It has been verified approximately for more complicated objects.⁴ The phenomenon, due to the difference in the sizes of the images introduced in the horizontal meridian only, is termed the "geometric effect," since it becomes primarily a problem in geometry.

On the other hand, that an apparent rotation about a vertical axis should result from increasing the size of the image of one eye in the vertical meridian is an entirely new and unexpected phenomenon⁵ and one that has no obvious connection with any known function of binocular vision. Since no stereoscopic depth function is known to result from a disparity in the vertical meridians of the retinal images of an object in space, the apparent rotation of the binocular visual field about the fixation point when a difference in sizes of the images of the two eyes is introduced in the vertical meridian cannot be explained by simple geometric means. Moreover, there is no obvious functional basis known that can account for such a phenomenon. The apparent rotation in this case, however, is in the direction opposite to that of the geometric effect, as though an increase in the size of the image in one eye in the vertical meridian induced an increase in the size of the image of the other eye in the horizontal meridian. For this reason this phenomenon has been designated the "induced size effect."

The influence of changes in the relative sizes and shapes of the images in the two eyes on these localization phenomena has been subject to an intensive research study over an extended period. The results of these studies will be presented in a series of papers. This paper, which is the first, will present that material which broadly describes the phenomena.

APPARATUS

The qualitative relation between the apparent rotation of surfaces and the relative sizes of the ocular images can be easily demonstrated, for example, by simply placing a meridional size lens in a trial frame before one eye and observing a field of objects that are more or less

4 Ames, A., Jr., and Ogle, K. N. Size and Shape of Ocular Images III. Visual Sensitivity to Differences in the Relative Size of Ocular Images of the Two Eyes, *Arch Ophth* 7:904 (June) 1932. This problem is considered further in the present paper.

5 The first evidence of the existence of this phenomenon was found in the failure of the apparent frontal plane (horopter) data to rotate to the full theoretic amount with overall size lenses placed before one eye when a small fusion object was substituted at the fixation point for the usual bare vertical wire or thread. The phenomenon was also described briefly by Ames.¹

free from perspective features. For this study, however, when quantitative results are desired for the apparent rotation which occurs with a given difference in size, a special instrument is necessary.

The apparatus used is schematically illustrated in figure 1, which is more or less self-explanatory. An observer, whose head is held by a suitable chin cup or wax bite and forehead rests, fixates an object plane at a given distance in symmetric convergence. After considerable experimentation, an object plane consisting of a sheet of plate glass 30 by 30 by 0.3 cm was selected. This is supported in a frame free to rotate about a vertical axis. A protractor mounted on the table, with an indicator fastened on the frame, permits the measurement of the angle of rotation from the "frontal" position. A suitable pattern of fusible figures or contours is drawn on the surface of the glass with india ink or black paint. A large sheet of white cardboard,

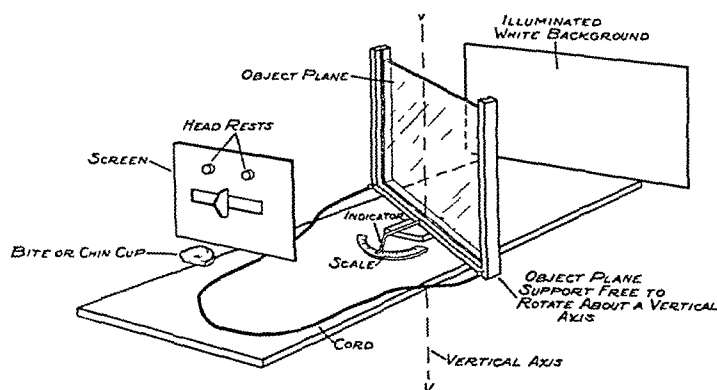


Fig 1—Schematic drawing of the apparatus used for a quantitative study of the geometric and induced size effects

uniformly illuminated, suspended behind the object plane for a background, silhouettes the pattern as seen by the observer. A white cardboard shield with an opening, which restricts the field of view to the plane, is usually placed in front of the eyes at a distance of about 10 cm. Suitable and adjustable lens holders (not shown in the figure) are also mounted before the eyes. By means of a cord, the observer can set the rotational position of the object plane.

EXPERIMENTAL TECHNIC WITH RESPECT TO CERTAIN ASPECTS OF THE PHENOMENA

Criteria for Setting the Object Plane—In obtaining the data for these experiments, the observer first adjusts the position of the plane according to a given criterion under normal binocular vision, and then, by the same criterion, when a difference in the sizes of the images has been introduced by a meridional size lens. The difference in the angular positions of the surface for the two adjustments is the desired

measurement It will be clear that this rotation of the object plane is in the direction opposite to the apparent rotation of a fixed plane or surface

Three criteria according to which the object plane can be adjusted have been used

1 The plane is set so that it appears "normal" to the subjective direction of the center of the plane from the observer, that is, the plane is set for an apparent "frontal" position⁶ For this criterion, the binocular visual field should be restricted to the object plane and other precautions should be taken to eliminate, so far as possible, the influence of empiric factors This is the criterion that has been used most

2 The plane is set so that it appears "normal" to a particular dimension of an object also seen in the field of view by binocular (or monocular) perspective Two types of such objects may be considered those whose positions through the perspective sense are uninfluenced by a size lens (for example, a string extending toward the object plane in a sagittal direction) and those whose positions through the perspective sense provide a good monocular criterion and whose perspective clues, as seen by the two eyes, are influenced only to a second order by the size lens (for example, a box in near observation) A pencil, mounted in the field of view and directed toward the center of the plane, establishes, by perspective factors, a subjective direction The observer may then adjust the object plane so that it appears "normal" to the apparent direction indicated by the pencil Here, reliance is placed on the perspective sense to orient the subjective directions in the binocular field

3 Not unlike the second criterion, the plane is set so that it appears parallel to a frame seen binocularly (or monocularly, if the visual field of one eye is restricted by a suitable aperture) This frame establishes by form and perspective factors a "surface" of reference⁷

Each of these criteria gave good results It was found, of course, that the primary, or naked eyes', adjustment might vary with the criterion, but the change in the apparent rotation of the plane caused by a given difference in the sizes of the images was more or less the same This statement, perhaps, would refer only to results of experi-

6 In these experiments the object plane is set for the "normal" position only in respect to a rotation about a vertical axis The problem relating to the other rotation about a horizontal axis is to be considered in another paper

7 A fourth criterion, for surfaces which are mounted in a more or less horizontal position free to rotate over a pivot, is to set the surface so that it appears "level" This criterion may be entirely subjective or may be referred to a fixed frame, plumb, etc

ments in which the particular apparatus described here was used, for, conceivably, the findings might vary with other types of apparatus.

Since these experiments have been concerned mainly with the phenomenon of the induced size effect, in which the change in the apparent rotation of the plane is of importance, the criterion of the apparent "normal" to the subjective direction of the center of the object plane (that is, of the apparent "frontal" position) has been used consistently in all cases.

Importance of Empiric Factors—In experiments of this type it is difficult to exclude all empiric factors which might tend to mask or inhibit the effects. Therefore, certain precautions are necessary. Among the most important of these are that movements of the object plane during an adjustment should be made quickly and that the judgment of "normalness" should be made while the plane is not moving. A compensating factor may be involved with continuous back and forth rotations that tend to influence the binocular setting of the plane. There is evidence that such a factor exists, in that with monocular vision the object plane can be adjusted for a "frontal" position with considerable accuracy⁸ by means of these back and forth rotations. However, it has been found that the stereoscopic function may so dominate that such monocular influences are negligible.

Among the more common factors which may tend to mask the apparent rotation of the plane when a difference in the sizes of the images is introduced is the form of the fusible pattern used on the object plane. Recognizable geometric forms or configurations that appear distorted in a definite manner when the plane is rotated may easily provide clues for ascertaining the "frontal" position. Patterns of this type (e. g., a circle or a square) on the plane must be avoided.

When there are many objects at varying distances in the field of view, it has been found that the parallax introduced by movements of the head may also tend to inhibit these size effects.

In general, an observer is more sensitive to the size effects if the object plane contains some stereoscopic device. For example, part of the pattern may be put on the back as well as on the front side of the glass plane, the thickness of the glass being sufficient to stimulate stereopsis at near visual distances. A stereoscopic stimulus apparently becomes more important for greater visual distances.⁹

⁸ That is, by a mean deviation of the order of 15 degrees for a visual distance of 40 cm.

⁹ Some persons when looking down on a grass lawn with a meridional size lens before one eye see no tipping of the ground until a twig or rod, which would serve to stimulate stereopsis, is stuck into the ground. Then a tipping appears suddenly.

Differences in the illumination apparently have little influence on the behavior of either the geometric effect or the induced size effect. As long as the fusible detail is seen readily, the rotation of the plane always appears with a difference in the sizes of the images. Even greatly different illuminations in the two eyes have little or no influence, as has been demonstrated by placing neutral tint filters before one of the eyes.

TABLE 1—*Typical Data for Four Observers Showing the Difference in Behavior of the Geometric and Induced Size Effects*

Magnification of Size Lens Used, %	Geometric Effect Meridional Size Lenses Placed at Axis 90°								Induced Effect Meridional Size Lenses Placed at Axis 180°							
	Observer								Observer							
	P D 61	P D 62	P D 68	P D 61									Observer			
	S C B	K N O	H M B	M B C									S C B	K N O	H M B	M B C
Before right eye																
6	-16.5 (0.5)								16.5 (0.6)							
4	-9.1 (0.6)	-13.4 (0.7)	-5.1 (0.4)	-10.0 (0.5)					16.3 (0.2)	11.9 (1.4)	10.2 (0.7)	10.5 (0.9)				
3		-10.7 (0.5)	-5.6 (0.4)							10.6 (0.9)	8.7 (0.6)					
2	-3.1 (0.1)	-6.7 (0.7)	-4.3 (0.4)	-6.3 (0.7)					10.4 (0.7)	6.8 (1.0)	5.8 (0.4)	4.7 (1.2)				
1		-3.4 (0.5)	-2.2 (0.3)							3.3 (0.4)	3.0 (0.6)					
Normal	4.9 (0.4)	0.6 (0.5)	1.2 (0.5)	-0.4 (0.7)					4.9 (0.4)	0.6 (0.5)	1.2 (0.5)	-0.4 (0.7)				
Before left eye																
1		4.3 (0.4)	2.8 (0.6)							-3.9 (1.0)	-1.1 (0.7)					
2	9.9 (0.4)	7.0 (0.4)	5.2 (0.3)	6.0 (0.4)					-3.3 (0.5)	-8.0 (1.0)	-3.7 (0.5)	-5.3 (0.9)				
3		10.9 (0.4)	10.2 (0.2)							-12.9 (0.8)	-6.0 (0.7)					
4	17.9 (1.0)	14.5 (0.5)	11.6 (0.4)	11.6 (0.8)					-8.8 (0.5)	-15.8 (0.9)	-7.4 (0.6)	-12.1 (0.8)				
6	20.9 (0.4)								-14.9 (1.2)							
Sensitivity, %																
I	-3.3	-3.6	-2.8	-3.0					3.2	3.7	2.5	2.7				
G	-3.7	-3.7	-3.3	-3.7												

* The data are the degrees of rotation of the plane about a vertical axis from a true frontal position. The plane was adjusted for an apparent "frontal" position for a given difference in the sizes of the images introduced by meridional size lenses in the horizontal and in the vertical meridian. The visual distance was 40 cm. A positive value indicates that the plane was rotated counterclockwise, i.e., the right side of the plane was moved away from the observer. The data in parentheses are mean deviations.

While the aforementioned factors tend to inhibit the effects, it appears that when strict attention is paid to the criterion demanded they may be ignored and their influences tend to disappear with experience. After considerable practice and provided the attention is confined strictly to the center of the object plane, almost the same results may be obtained regardless of whether the binocular visual field is restricted to the object plane alone or whether the whole plane, with supports and other objects having perspective forms, are visible at the same time. Of course, individual variations are found.

SENSITIVITY OF THE APPARENT ROTATION OF SURFACES

Preliminary Type of Data—The preliminary data to be reported here were taken with a pattern on the object plane consisting of a large number of relatively small spots of black india ink scattered indiscriminately on both sides of the glass sheet. Such a pattern provides fusion

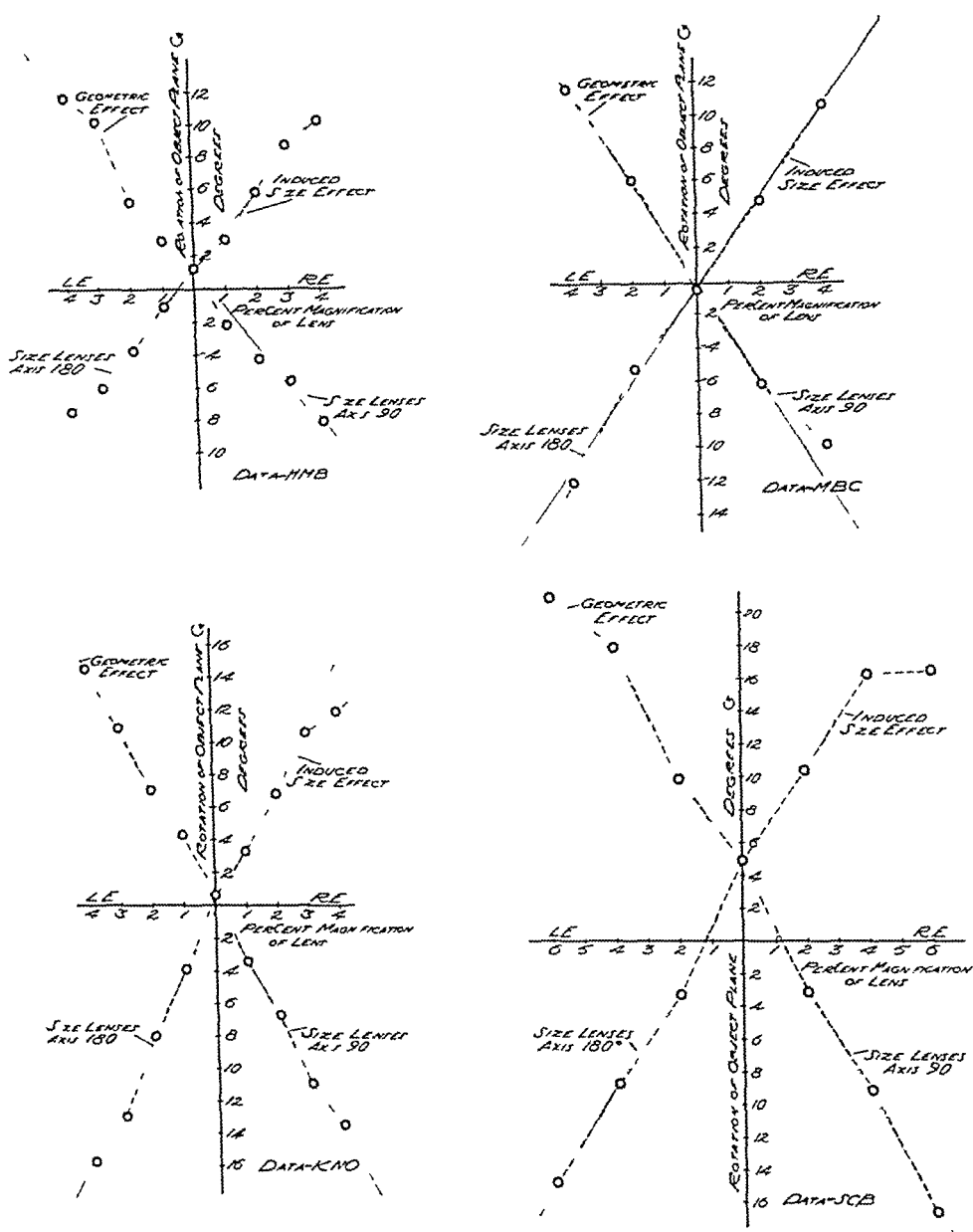


Fig 2—Graphic representation of the typical preliminary type data for several observers showing the geometric and induced size effects. The visual distance was 40 cm.

stimuli as well as adequate stereoscopic stimuli, over the whole of the restricted binocular visual field and yet presents no easily recognizable geometric configurations. After the observer's head had been adjusted in the apparatus, he was asked to set the plane, so far as the horizontal meridian was concerned, according to the criterion of the apparent

"frontal" position, that is, to set the plane so that it appeared "normal" to the subjective direction of the center of the plane. The angular positions of the plane for several adjustments (from three to five) were recorded. Then a meridional size lens, the magnification properties of which were known accurately for the particular visual distance, was inserted before one eye, and the observer was again asked to adjust the position of the plane for the same criterion. The plane's position was again recorded for several settings. With a series of size lenses, complete data were obtained for the corresponding apparent rotations.

Data taken in this manner have been obtained for a large number of persons¹⁰. Table 1 contains the typical results, with mean variations, for four more or less experienced observers, for both the geometric (size lenses axis 90 degrees) and the induced (size lenses axis 180 degrees) effects. These data are illustrated graphically in figure 2, in which the percentage of difference in the sizes of the images produced by the meridional lens is plotted on the abscissa axis and the amount of rotation of the plane in degrees (positive counterclockwise) is plotted on the ordinate axis.

These graphs show that, within the limits indicated, the distribution of the points representing the data is more or less linear. In general, with differences in the sizes of the images of less than 4 per cent, the amount of apparent rotation of the surface in both the geometric and the induced size effects may be considered proportional to the difference in the meridional sizes of the images. The magnitude of the proportionality, which can be determined from the slope of the line which best fits the data points, will be designated as the sensitivity of the effect. This is expressed in degrees of apparent rotation of the plane for a 1 per cent difference in the sizes of the images in the two eyes. The computed sensitivities of the geometric effect (designated by *G*) and of the induced size effect (designated by *I*) for the data in table 1 are given in the bottom row. In general, the sensitivity to the induced effect is of the same order as that of the geometric effect.

Inspection of table 1 shows that the sensitivity of these more or less experienced observers is about 3 to 3.5 degrees of rotation for a 1 per cent difference in the sizes of the images. Some variation in the sensitivity is found between observers (apart from being due to differences in the interpupillary distances) and to a smaller degree in the data for a given observer taken at various times. The latter variation tends to

¹⁰ As early as 1934 a technic was developed for a general study of the effect of size lenses on the subjective orientation of various types of tipping boards. Mr. Henry A. Imus, with the assistance of Mr. William F. Peck, Mr. Harold M. Fisher and others, obtained a great deal of important data. That work laid the foundation for the present investigations.

decrease with experience. Similar data obtained for a large group¹¹ (600) of inexperienced observers show a sensitivity for near vision (40 cm) to the geometric effect of -2.3 degrees for 1 per cent ($\sigma = 0.59$) and to the induced effect of 2.2 degrees for 1 per cent ($\sigma = 0.65$). These sensitivities are in general lower than those found for more experienced observers. For trained observers the sensitivity of the induced effect may, under certain conditions, exceed that of the geometric effect. Various factors, such as fatigue, may from time to time contribute to a variation in the sensitivity, especially in the induced size effect.

These general results are also found for visual distances other than 40 cm. For greater visual distances, i. e., 3 meters, more concentration and a definite response to stereoscopic stimuli on the part of the observer are necessary.

The induced size effect seems to occur immediately when a difference in the sizes of the images of the two eyes is introduced in the vertical meridian. There has been evidence that for less complex visual patterns on the object plane the induced size effect reaches a maximum after the lapse of a short interval during which the observer adjusts the surface. On the other hand, no easily demonstrable after-effect or lag persists when the difference in size is removed.

One of the striking aspects of such data is the accuracy with which the settings of the object plane are made. Some idea of this accuracy can be obtained from a study of the mean deviations of the mean settings for the data given in table 1. These average about ± 0.5 degree, and rarely exceed 1 degree. Since a 1 per cent difference in the sizes of the images causes an apparent rotation of the plane of about 3 degrees, such a mean deviation corresponds roughly to ± 0.2 per cent of difference in the sizes of the images. This figure agrees with the order of sensitivity of the eyes to differences⁴ in the sizes of the images as determined by other methods.

Overall Size Lenses, Rotated Meridional Size Lenses—Since the sensitivity of the induced size effect for such a complex fusion pattern has about a one to one relationship with that of the geometric effect, though in the opposite direction, it would be expected that the use of overall size lenses would cause no marked apparent rotation of the object plane. Qualitatively, this is true, though considerable variations may occur. This phase of the problem, however, will be left for consideration at a later time.

¹¹ From a visual survey of the class of 1940 at Dartmouth College, a report of which will be published.

Another instance in which the geometric and the induced effects may also act more or less at the same time may be found in determining the apparent rotation of the object plane when the meridional size lens before one eye is rotated to various angular positions (in the sense of rotating an astigmatic lens). A typical set of data,¹² as illustrated graphically in figure 3, shows the usual cosine type of curve. When the magnifying effect of the lens is in the horizontal meridian (axis 90 degrees), the apparent rotation is a geometric one, when it is in the vertical meridian (axis 180 degrees), the apparent rotation is due solely to the induced size effect. At the oblique meridians of 45 and 135 degrees, the two effects more or less offset each other, since the magnification components of the lens in the vertical and horizontal meridians are equal. At all other oblique meridians, one effect or the other dominates.

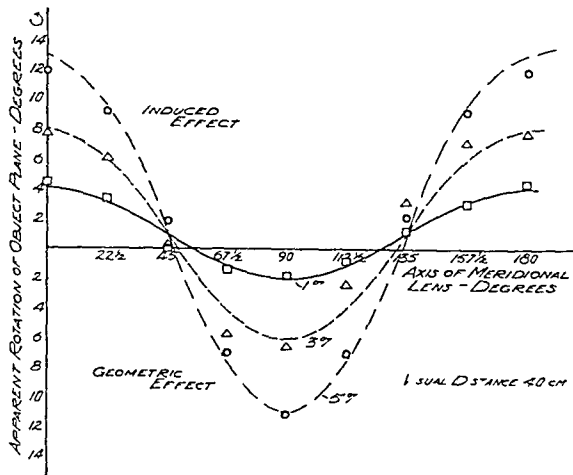


Fig 3—Graphic representation of typical data (for H F) showing the magnitude of the apparent rotation of a surface seen binocularly about a vertical axis caused by the rotation of a meridional size lens before the right eye to different angular positions

Rotational Deficiency of the Geometric Effect—On the basis of the horopter theory, the magnitude of the apparent rotation of the object plane for a given difference in sizes of the images introduced in the horizontal meridian can be computed from the equation $\tan \psi' = -\frac{M-1}{M+1} \frac{b}{a}$. The sensitivity due to the geometric effect would be, therefore, $G = -0.28 \frac{b}{a}$ degrees of rotation for a 1 per cent difference in the sizes of the images. In this equation, also, b is the visual distance and a is one-half the interpupillary distance. In general, it has been found that the measured sensitivity is less than the theoretic by about 20 per cent. This deviation of the measured sensitivity from the theoretic

12 These data were obtained by Mr H A Imus and Mr H M Fisher

sensitivity will, for convenience, be designated as a deficiency of the geometric sensitivity. While the exact cause of this deficiency is as yet unknown, it is probably due to special empiric factors that cannot be eliminated easily in this type of apparatus. The magnitude of the deficiency varies, to a certain extent, with the observer. In this respect, inexperience in binocular observation plays a part, though the greatest deficiencies are usually found in persons with weak fusion powers. For a given trained observer, the deficiency is remarkably constant for a given apparatus.

MAXIMUM INDUCED SIZE EFFECT PHENOMENON

When a meridional size lens is placed before one eye which magnifies the retinal image in the horizontal meridian (lens at axis 90 degrees), the change in the apparent rotation of the binocular visual space is in accordance with purely geometric causes. Apart from certain unavoidable empiric factors, therefore, this apparent rotation would agree with the longitudinal horopter theory. Hence, as successive lenses of increasing magnification are used before one eye, the magnitude of the apparent rotation of the surface seen binocularly increases nearly proportionally with the difference in the sizes of the images.

This is not true in the case of the induced size effect. As the difference in the sizes of the images of the eyes in the vertical meridian is increased, the apparent rotation of the surface seen binocularly increases up to a limit beyond which no further increase, but usually a decrease, is found. This phenomenon will be evident from typical complete data for several observers taken with the same pattern of scattered dots on both sides of the object plane. These data are given in table 2 and are illustrated graphically in figure 4, in which the percentage of difference in the sizes of the images introduced by meridional size lenses is plotted on the abscissa axis and the amount of apparent rotation of the object plane about the vertical axis (positive counter-clockwise) is plotted on the ordinate axis.

It will be clear from an inspection of these graphs that in the geometric effect the apparent rotation of the plane is directly proportional to the difference in the sizes of the images in the horizontal meridian. This is indicated by the fact that a straight line describes adequately the distribution of the data points throughout the wide range of differences in the sizes of the images. This is not true for the induced size effect, for the smooth curve drawn through the points is a typical S-shaped curve. Here the apparent rotation of the plane for a given difference in the sizes of the images in the vertical meridian is in the direction opposite to that of the geometric effect, and hence the curve lies in the opposite quadrants of the graph.

Previously, the sensitivity of either of the effects was defined as the magnitude of the apparent rotation of the plane caused by a 1 per cent difference in the sizes of the images. These results would show that for the induced size effect the sensitivity varies over the range of differences in the sizes of the images in the vertical meridian. In the central regions of these curves the sensitivity is more or less constant,

TABLE 2—*Typical Data for Four Observers Showing the Difference in Behavior of the Geometric and Induced Size Effects**

Magnification of Size Lens Used, %	Observer							
	H A W		H M B		G S N		R H D	
	Geometric Effect	Induced Effect	Geometric Effect	Induced Effect	Geometric Effect	Induced Effect	Geometric Effect	Induced Effect
	Lenses Axis 90°	Lenses Axis 180°	Lenses Axis 90°	Lenses Axis 180°	Lenses Axis 90°	Lenses Axis 180°	Lenses Axis 90°	Lenses Axis 180°
Before right eye								
12.5		8.8 (0.8)				14.3 (0.8)		
10.3		8.1 (0.2)				17.7 (0.4)		
8.2		9.0 (0.2)	—24.2 (0.6)	11.6 (0.9)	—30.0 (0.7)	18.8 (0.5)	—30.3 (0.6)	23.1 (0.3)
6.6							—26.1 (0.3)	23.0 (0.8)
6.1	—16.7 (1.1)	7.1 (0.4)	—14.0 (0.6)	10.8 (0.8)	—22.6 (1.4)	16.6 (0.6)	—20.2 (0.9)	18.8 (0.3)
4.5							—18.3 (0.1)	17.5 (0.4)
4.0	—10.5 (1.3)	6.0 (0.5)			—15.4 (0.3)	11.1 (0.9)		
3.0			—10.9 (0.2)	8.5 (0.8)				
2.0	—6.1 (0.3)	4.3 (0.3)			—7.0 (0.4)	7.7 (0.3)	—11.2 (0.2)	11.3 (0.3)
No lenses	—0.5 (0.4)	—0.5 (0.4)	—2.3 (0.5)	—2.3 (0.5)	0.4 (0.3)	1.0 (0.2)	—0.2 (0.3)	—0.2 (0.3)
Before left eye								
2.0	4.2 (0.4)	—4.8 (0.6)			7.5 (0.4)	—7.7 (0.3)	11.1 (0.8)	—11.2 (0.3)
3.0			8.9 (0.4)	—10.4 (0.6)				
4.0	13.8 (0.3)	—7.9 (1.4)			15.0 (0.7)	—13.9 (0.7)		
4.5							19.1 (0.6)	—15.1 (0.1)
6.1	16.4 (0.2)	—7.9 (0.8)	14.9 (0.9)	—17.5 (0.9)	21.7 (1.0)	—18.8 (0.6)	19.8 (0.3)	—18.5 (0.4)
6.6							26.5 (0.8)	—19.5 (1.0)
8.2		—11.0 (1.0)	22.2 (0.9)	—21.1 (0.5)	29.2 (0.4)	—22.6 (1.0)	30.4 (0.1)	—20.0 (1.0)
10.3		—9.8 (1.2)				—21.7 (2.7)		
12.5		—9.2 (1.2)				—18.9 (1.9)		
Maximum sensitivity, %	—2.84	2.03	—2.87	2.75	3.72	3.36	—3.86	3.90

* The data are the degrees of rotation of the plane about a vertical axis from a true frontal position. The plane was adjusted for an apparent "frontal" position for a given difference in the sizes of the images introduced by meridional size lenses in the horizontal and in the vertical meridians. The visual distance was 40 cm. The data in parentheses are the mean deviations.

that is, the apparent rotation of the plane is approximately proportional to the difference in the sizes of the images. These portions of the curves are usually within a range of ± 4 per cent difference and correspond to the data described in the last section of this paper as the preliminary data. These sensitivities at the central part of the curves will obviously be the maximum sensitivities of the effect. With increasing differences in the sizes of the images in the vertical meridian, the sensitivity of the apparent rotation decreases, until a maximum

apparent rotation is reached. These maxima correspond to the tops and bottoms of the S-shaped curves. With still increasing differences in the sizes beyond these maxima, the sensitivity increases negatively, that is, the apparent rotation of the plane with increasing differences in the sizes of the images in the vertical meridian actually tends to decrease. The maximum sensitivity of the geometric and the induced

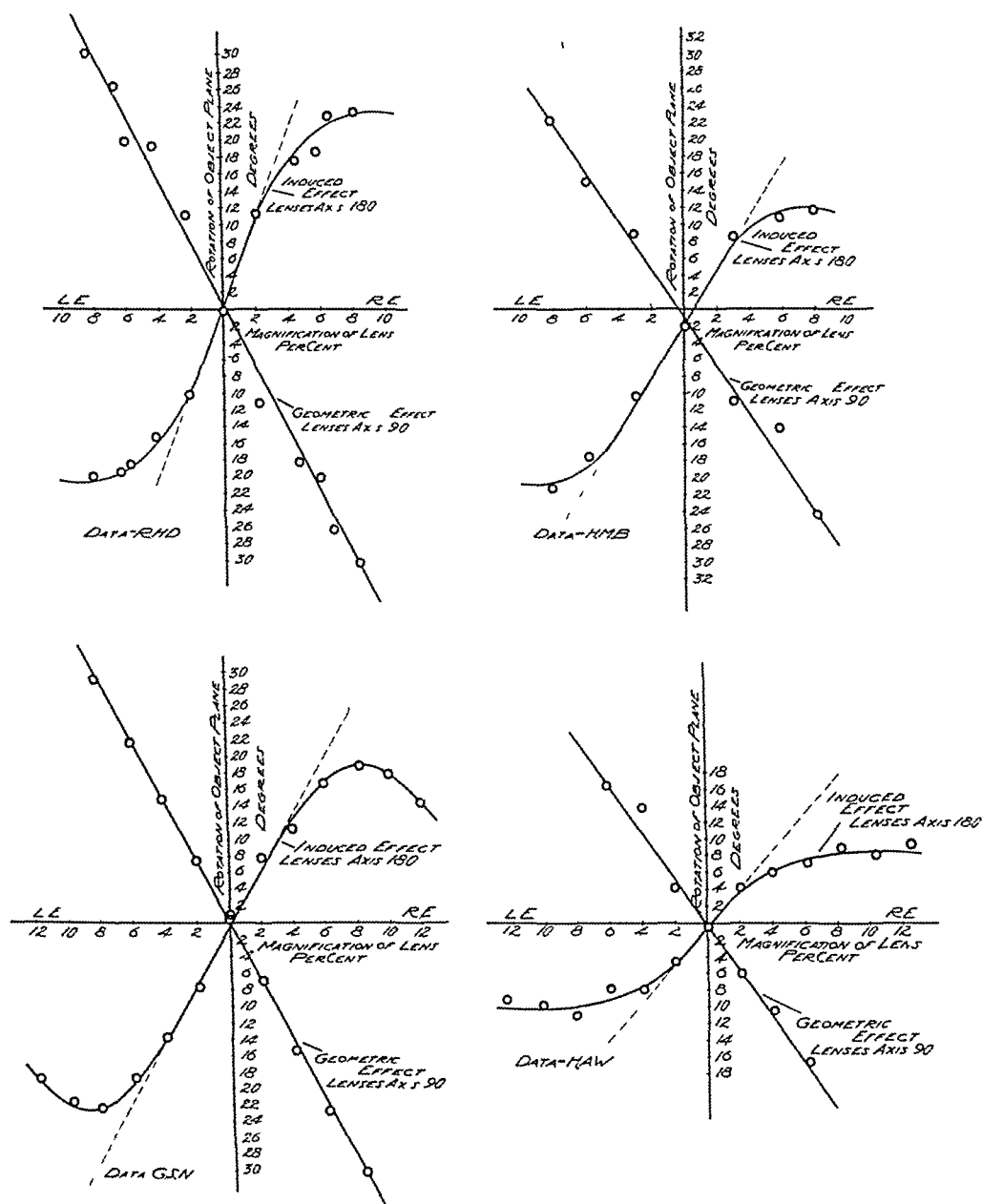


Fig 4—Graphic representation of typical data showing the difference in behavior of the geometric and induced size effects for greater differences in the sizes of images. The data are the amount of apparent rotations (degrees) of the object plane caused by differences in the sizes of the images (percentage) introduced by meridional size lenses before one or the other of the eyes. The visual distance was 40 cm.

effects is given in the lower row of table 2. Inspection of these figures shows, again, nearly a one to one relationship.

While there are certain objections to such a pattern for this experiment, the data obtained are useful to demonstrate that even with such a complex pattern the phenomenon of the maximum induced effect exists.

TABLE 3—*Typical Data for K N O Showing the Induced Size Effect Obtained with an Object Plane Pattern Consisting of a Ring of Scattered Dots**

Meridional Size Lenses Used at Axis 180° Before Right Eye, %	Rotation of Plane, Degrees	Meridional Size Lenses Used at Axis 180° Before Left Eye, %	Rotation of Plane, Degrees
No lenses	-2.1 (0.7)	No lenses	-2.1 (0.7)
0.5	-1.8 (0.7)	0.5	-5.9 (0.4)
1.0	0.2 (0.4)	1.0	-7.9 (0.1)
1.5	-0.5 (0.6)	1.5	-9.3 (0.9)
2.0	1.4 (0.4)	2.0	-11.3 (1.0)
2.2	-0.1 (0.8)	2.2	-10.3 (1.2)
2.5	1.1 (1.2)	2.5	-13.3 (0.9)
3.0	4.3 (0.7)	3.0	-16.0 (1.1)
3.5	3.2 (1.3)	3.5	-17.2 (0.8)
4.0	3.1 (0.9)	4.0	-16.2 (0.7)
5.0	1.7 (1.4)	5.0	-16.7 (2.1)
6.0	2.3 (0.8)	6.0	-19.2 (0.8)
7.0	-0.6 (0.8)	7.0	-20.7 (1.2)
8.2	-0.5 (0.4)	8.2	-20.8 (2.2)
		9.0	-18.4 (2.1)
		12.5	-16.9 (2.0)

* The data are the degrees of rotation of the plane about a vertical axis from the true frontal position, when the plane is adjusted for an apparent "frontal" position for a given difference in the size of the images in the vertical meridian. The visual distance was 40 cm., the angular size of the ring, 5 degrees. The numbers in parentheses are the mean deviations.

TABLE 4—*Typical Data for R H D Showing the Induced Size Effect Obtained with an Object Plane Pattern Consisting of Two Separated Horizontal Bands of Scattered Dots**

Lenses Before	Meridional Size Lenses Used at Axis 180°					
	None	2%	4%	6%	8%	10%
Right eye	1.1 (0.1)	8.6 (0.3)	14.1 (0.5)	15.7 (0.6)	13.3 (0.9)	13.5 (?)
Left eye		-5.5 (0.4)	-11.1 (0.1)	-12.9 (1.5)	-10.8 (0.2)	-11.5 (1.0)

* The data are the degrees of rotation of the plane about a vertical axis from the true frontal position, when the plane is adjusted for an apparent "frontal" position for a given difference in the size of the images in the vertical meridian. The visual distance was 40 cm., the angular separation of bands, 10 degrees. The numbers in parentheses are the mean deviations of the settings.

With such a complex type of pattern on the object plane, some variations may be expected between different observers.

The average difference in the sizes of the images and the vertical meridian at which the maximum effect occurs (and also at which the induced effect curve begins to depart from the straight line portion of the curve) for the particular pattern on the object plane used for these data varies somewhat with the observer. Lower values are usually found for the more inexperienced or untrained observers. The average

difference in the sizes of the images in the vertical meridian for the maximum effect for the data given here is between 7 and 9 per cent. For one highly trained observer, it was questionable whether the effect had begun to leave the straight line portion of the curve even at an 8 per cent

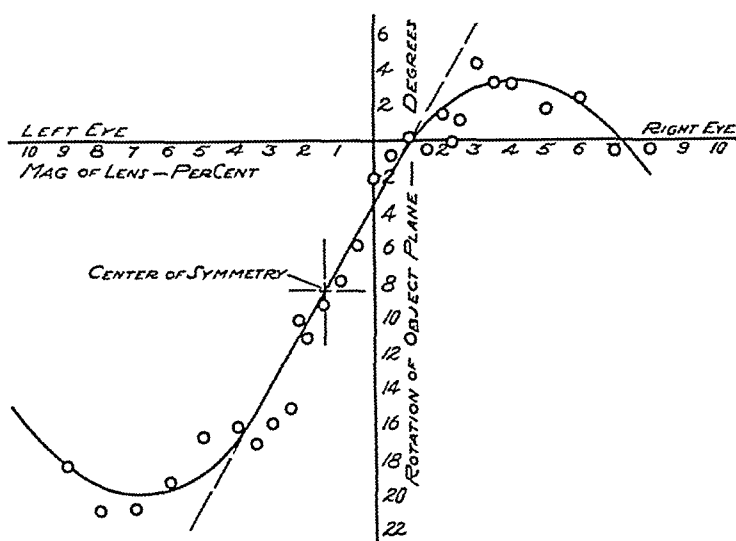


Fig 5—Graphic representation of typical data (for K N O) showing the induced size effect for a surface having fusion stimuli unrestricted at a fixed visual angle. The visual distance was 40 cm.

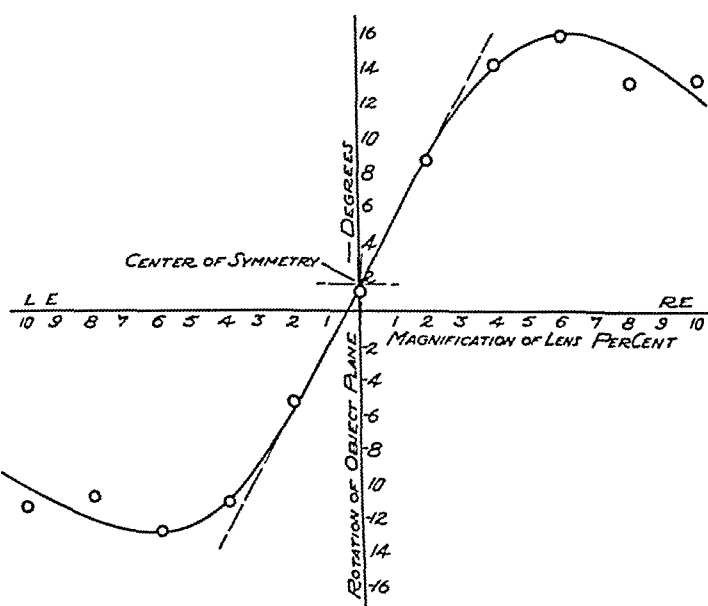


Fig 6—Graphic representation of typical data (for R H D) showing the induced size effect for a surface having fusion stimuli restricted to a fixed visual angle. The visual distance was 40 cm.

difference in the sizes of the images. This observer, however, was conscious of a disturbed sense of fusion and a definite sensation that the criterion by which the object plane could be adjusted was confined more and more to the central regions of the plane as the difference in

the sizes of the images in the vertical meridian was increased. This would, of course, be consistent with the fact that the linear retinal disparity due to a given difference in the sizes of the images is greater in the peripheral parts of the retinas than in the parafoveal parts,⁴ and hence fusion would tend to be more disturbed in the peripheral regions. Certainly, it was clear to some of the more experienced observers that as the difference in the sizes of the images in the vertical meridian was increased the peripheral regions of the visual field played a less and less important part.

When the central portions were eliminated entirely, as, for example, in a pattern on the object plane consisting of an approximate ring of

TABLE 5—*Typical Data for R H D Showing the Geometric and Induced Size Effects for Visual Distance of 20 and 75 Cm*

Meridional Size Lenses Used, %	Visual Distance, 75 Cm		Visual Distance, 20 Cm	
	Geometric Effect Lenses at Axis 90°	Induced Size Effect Lenses at Axis 180°	Geometric Effect Lenses at Axis 90°	Induced Size Effect Lenses at Axis 180°
Right eye 10		25.5 (1.0)		8.7 (0.2)
8		28.4 (1.5)	-13.0 (0.8)	7.7 (0.9)
6	*	27.0 (1.0)	-10.2 (0.2)	8.0 (0.4)
4	-24.6 (0.4)	22.9 (0.5)	-7.8 (0.7)	6.2 (0.2)
2	-12.8 (0.8)	14.1 (0.2)	-3.3 (0.6)	3.2 (1.0)
None	0.2 (0.5)	1.2 (0.3)	-0.2 (0.8)	0.7 (0.8)
Left eye 2	16.0 (0.3)	-8.8 (0.7)	3.7 (0.3)	-1.9 (0.6)
4	28.7 (0.6)	-14.2 (0.5)	8.4 (0.3)	-5.0 (0.4)
6	*	-20.1 (0.1)	12.7 (0.5)	-8.5 (0.8)
8		-19.1 (2.0)	16.1 (0.6)	-8.9 (0.5)
10		-15.1 (2.1)		-9.1 (0.4)
12				-9.6 (0.6)
14				-7.7 (0.8)
Maximum sensitivity	6.7°/°	6.0°/°	-2.0	1.4°/°
Theoretic sensitivity	7.2°/°		-2.0	

The asterisk indicates that mechanical limitations prevented these data from being obtained.

scattered dots, the points of maximum induced effect became more definite for all observers. Typical results for one observer, when using this type of pattern in which the mean radius of the ring subtended a vertical visual angle of 5 degrees, are shown in table 3 and are illustrated graphically in figure 5. Likewise, the results for an observer using a pattern on the object plane consisting of two separated horizontal bands of scattered dots are given in table 4 and illustrated in figure 6. It will be clear from an inspection of these curves that the maximum points of the induced effect are much more pronounced than those for a more complex pattern on the object plane. On the basis of considerable experience it is believed that the data points are best represented by a curve which is symmetric in shape.

That the same phenomenon exists also for other visual distances is shown by the data for one observer given in table 5 and illustrated in figure 7.

These experiments on the induced size effect seemed regularly to cause some eyestrain, and if they were continued for any length of time, headaches and other ocular disturbances resulted. Especially was this discomfort felt when the differences in the sizes of the images were greater than 5 or 6 per cent. During each observation, the observer felt a nervous tension and an ocular "pulling" sensation, usually accompanied by a strong desire to finish the observation quickly. In some persons these ocular disturbances persisted for several hours after taking the data.

The patterns on the object plane described here are complex, and so for the present a discussion concerning the significance of the maxima found in the induced effect will be necessarily postponed.

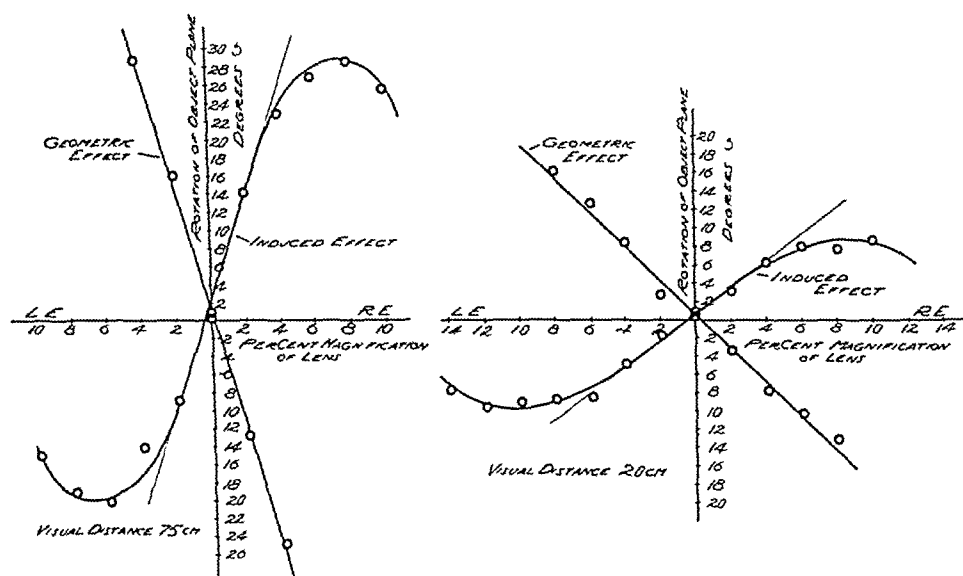


Fig 7—Graphic representation of typical data (for R H D) showing the induced size effect for the visual distances of 75 and 20 cm

COMMENT AND CONCLUSIONS

For geometric reasons, together with the fact that the stereoscopic function exists in the horizontal meridian, one might anticipate a change in the apparent rotational position of a surface seen binocularly when a change is introduced in the relative sizes of the images of the eyes in the horizontal meridian. Apart from a small deficiency, the results with a complex pattern verify this theoretic expectation.

The induced effect, however, which is the phenomenon of the apparent rotation of the binocular visual field caused by a difference in the sizes of the images in the vertical meridian, is a new and unexpected phenomenon that could not have been anticipated. Certainly, no geometric explanation is obvious. Moreover, its functional basis is not clear since no stereoscopic depth perception is known to originate from

disparities of retinal images in the vertical meridian. Even if the phenomenon were due to some other type of physiologic or interpretative cause, a functional basis would be expected.

Three facts, based on the preliminary data given in this paper, are of importance in regard to this phenomenon. First, a one to one ratio of the maximum sensitivities of the induced to the geometric effects exists, though the two effects are opposite in sign, second, the effect reaches a maximum value for differences in the sizes of the images in the vertical meridian greater than 5 or 6 per cent, and third, above this difference the induced effect decreases slowly.

On the basis of the data presented, only a conjecture as to the nature of this phenomenon is possible. The one to one relationship between the sensitivity of the induced and the geometric effect for a difference in the sizes of the images of less than 4 per cent certainly suggests that the induced effect is an image size phenomenon. Whatever mechanism may be responsible, it appears that so far as binocular depth perception is concerned, the introduction of a difference in the sizes of the images in the vertical meridian effectively causes a difference in the sizes of the images in the horizontal meridian in direct proportion to that in the vertical meridian. One might argue for a compensation theory, in which the ocular system, through some mechanism, compensates for the difference in the sizes of the images in the vertical meridian but can only do so by an overall change in the relative sizes of the ocular images. Thus, while compensating for this difference, a residual difference in the sizes of the images, which will be proportional to the compensation, remains in the horizontal meridian. Such a hypothesis avoids the necessity of trying to explain the effect on a known functional basis.

The fact that a maximum induced effect occurs for differences in the sizes of the images in the vertical meridian of 5 or 6 per cent is significant. Irrespective of the nature of the mechanism which causes the effect, this fact indicates a definite limitation in the extent to which it can operate. In this connection, it should be noted that seldom have differences greater than 5 per cent been found in the clinic for patients whose fusion powers were normal.¹³

It is of further significance that if the difference in the sizes of the images in the vertical meridian is further increased after the maximum apparent rotation of the object plane has been reached, the induced effect still persists and decreases slowly. Thus, on the basis of the foregoing theory, the compensation process is maintained only

13 Carleton, E. H., and Madigan, L. F. Size and Shape of Ocular Images II. Clinical Significance, *Arch. Ophth.* 7:729 (May) 1932.

to a slightly less extent than the limiting amount for high differences in the sizes of the images in the vertical meridian

The only known allied phenomenon is the apparent change in the sizes of the ocular images found when the eyes are turned in asymmetric convergence^{2c} Apparently, a compensatory process occurs there which offsets the retinal difference in the sizes of the images that would arise because of the difference in the distances from the object to the two eyes Whether or not the induced size effect could be a part of the same phenomenon is merely a matter of conjecture It is not the purpose of this paper to attempt an exhaustive discussion of the effect but merely to present evidence which will establish its existence This phenomenon must be considered, however, one of the most important phases of the study of the relation between binocular vision and depth perception and the relative sizes and shapes of the ocular images of the two eyes Moreover, it is believed that the induced effect may prove an important tool in determining other facts regarding the nature of the binocular visual processes Of special weight in this respect is the extreme accuracy with which the eyes respond to these various binocular phenomena

This work was done under the supervision of Prof A Ames Jr , some of the data used were supplied by various members of the research group

EXPERIMENTAL IONTOPHORESIS OF RABBITS' CORNEAS

REPORT OF TWO CASES OF CORNEAL DYSTROPHY WITH TREATMENT
BY IONIC MEDICATION

STEPHEN G. SEECH, M.D.

AND

WILLIAM LEGRANDE COOPER, M.D.

LOS ANGELES

Leduc in 1900 was the first to describe methods of therapeutic ionization and in 1903 he published the first report on the application of zinc ions in the treatment of rodent ulcers. In 1904 he used quinine in the treatment of an acute attack of tic, claiming a favorable result.

Ramsden in 1908 reported a case of ophthalmia neonatorum in which cure was effected in two days with ionization.

Traquair¹ in 1911 treated corneal ulcers with zinc iontophoresis. His best results were achieved in cases of moderately severe involvement and also in those cases in the advanced stage in which the condition of the cornea contraindicated cauterization. Scar formation was less extensive than with the use of cautery. The application of a small electrode was found most advantageously effective, a current of 0.5 milliamperes being used for one and a half minutes.

Birkhauser² in 1921 experimented on the corneas of rabbits and then on old corneal opacities. He found that the usual methods of treatment did not result in any improvement, while chloride iontophoresis increased the visual acuity. He recommended the selection of cases and expressed the belief that the opacities which blend into the normal tissue, without sharp borders, are more amenable to treatment.

Fietta³ in 1924 applied atropine ions to break down adhesions and found them only moderately satisfactory. In 1932 he⁴ reviewed the subject and concluded that only the anterior segment of the eyeball lent

Read before the Los Angeles County Medical Association, Section of Ophthalmology, March 28, 1938.

1 Traquair, H. M. The Treatment of Purulent Keratitis by Zinc Iontophoresis, *Ophth. Rev.* **30** 1 (Jan.) 1911.

2 Birkhauser, R. Iontophoretic Treatment of Corneal Opacities with Tube Electrode, *Klin. Monatsbl. f. Augenh.* **66** 536, 1921.

3 Fietta, P. Quelques essais d'iontophorèse à l'atropine, *Rev. gen. d'opht.* **38** 317 (Aug.) 1924.

4 Fietta, P. The Value of Iontophoresis in Ophthalmology, *Ann. d'ocul.* **169** 613 (Aug.) 1932.

itself to ionization, the course was long, and many treatments were necessary

Mairi⁵ in 1927 reported 1 case of albuminuric retinitis in which general treatment did not bring about any improvement. After twenty-eight treatments with ionization the edema disappeared and vision improved to 7/10.

Smith⁶ in 1927 expressed the belief that zinc ionization is a potent therapeutic weapon. He obtained striking results with corneal lesions and emphasized that a current of not more than 0.5 to 1 milliamperes should be used for not more than a few seconds. For other conditions he permitted the duration of the treatment to be increased to two minutes with a current of 2 milliamperes.

Simon⁷ in 1927 used a 1 per cent solution of potassium iodide in rabbits' eyes for from one to thirty minutes with a current of from 1 to 5 milliamperes. The aqueous showed penetration of the iodide in all the eyes, but none was found in the lenses.

Steindorff⁸ in 1928 made quantitative estimations of the iodine content of the aqueous, vitreous and lens substance of rabbits' eyes. He exposed the eyes to a current of 10 milliamperes for thirty minutes. The aqueous showed a considerable amount of iodine, but not a trace was found in the lens.

Cantonnet⁹ in 1928 found that ionization would temporarily reduce tension in cases of glaucoma. He employed solutions of calcium chloride and sodium iodide for from twenty to thirty minutes. He did not advocate ionization as a substitute for operation but only when the operation is refused or contraindicated.

Protopopoff¹⁰ and Malkin¹¹ in 1929 both reported concurrently on the treatment of cataract with a 1 per cent solution of sodium iodide. They did not find any improvement in the visual acuity, nor could they observe any retardation in the progress of the cataract.^{11a}

5 Mairi, E. A Case of Albuminuric Retinitis Cured by Iontophoresis, *Arch di ottal* **34** 20 (Jan) 1927

6 Smith, E. T. Zinc Ionization in Ophthalmic Work, *M. J. Australia* **2** 115 (July 23) 1927

7 Simon de Guilleuma, J. M. Ionization of the Eye, *Clin. opht* **16** 483 (Sept) 1927

8 Steindorff, K. Experiments in Iontophoresis, *Arch. f. Ophth* **120** 175, 1928

9 Cantonnet, A. L'ionisation dans le glaucome, abstracted, *Brit. J. Ophth* **14** 376 (July) 1930

10 Protopopoff, B. V. Iontophoresis in Cataracts, *Arch. oftal* **6** 378 (Oct) 1929

11 Malkin, B. Experiences with Iontophoresis in Corneal Ulcers. Preliminary Report, *Klin. Monatsbl. f. Augenh.* **83** 502 (Oct-Nov) 1929

11a Malkin, B. Iodine Iontophoresis in Senile Cataract, *Ztschr. f. Augenh.* **78** 259 (Sept) 1932

Ptaschnick¹² in 1930 applied the Birkhauser technic, his experience was encouraging, even in those cases in which other methods of treatment failed. At least thirty or forty treatments were necessary before improvement could be seen.

Airuga¹³ in 1930 applied a solution of ethylhydrocupreine hydrochloride in a dilution of 1:100 in 21 cases of corneal ulcer. He used a current of 2 milliamperes for from two to four minutes. At the most, three applications were necessary. The principal advantage of this type of treatment was the thinness of the scar.

Jusefova¹⁴ in 1931 applied atropine iontophoresis in 23 cases in which mydriatics were ineffective. In most cases he accomplished partial or complete dilation and found that the ionic medication of atropine had a definite analgesic action.

Donin¹⁵ in 1931 and Malkin¹⁶ in 1931 used a 1 per cent solution of copper sulfate in the treatment of trachoma and found it an acceptable adjunct but ineffective if used alone.

Ossimin¹⁷ in 1931 obtained good results in the treatment of corneal ulcer with the use of Cantonnet's electrode, applying a current of from 0.2 to 2 milliamperes for from fifteen to thirty minutes. The treatment stopped the progress of the disease and led to regeneration of the tissue with clearing of the opacities. He recommended ionization as a favorable aiding factor in the treatment of opacities of the vitreous and recent leukoma of the cornea. The effect was not lasting in cases of glaucoma. He used solutions of zinc, sodium iodide, sodium salicylate and potassium chloride in dilutions of 1:1,000 as electrolytes.

Simon¹⁸ in 1916 used salicylic acid and lithium ions in the treatment of episcleritis, Erlanger¹⁹ in 1932 applied histamine in strengths of

12 Ptaschnick, D. B. Iontophoresis and Its Use in Corneal Lesions, *Arch ophthalmol* **7** 210, 1930.

13 Arruga, R. Treatment of Serpiginous Ulcer of the Cornea by Means of Iontophoresis with Ethyl Hydrocupreine Hydrochloride, *Arch de oftal hispano-am* **30** 327 (June) 1930.

14 Jusefova, F. J. The Use of Atropine Iontophoresis on Ophthalmic Practice, *Arch oftal* **8** 296, 1931.

15 Donin, J. H. Experience with Iontophoresis in Trachoma, *Russk oftal j* **14** 396 (April) 1931.

16 Malkin, B. Iontophoresis in the Treatment of Trachoma, *Russk oftal j* **14** 387 (April) 1931.

17 Ossimin, S. D. Iontophoresis in Ocular Diseases, *Russk oftal j* **14** 301 (Oct-Nov) 1931.

18 Simon de Guilleuma, J. M. Modele nouveau d'oeillere porta-electrode pour le traitement electro-ionique du globe de l'oeil, *Clin opht* **21** 463, 1916.

19 Erlanger, G., and Erlanger, A. Dilatation of Pupil Produced by Iontophoresis with Epinephrine, *Klin Monatsbl f Augenh* **88** 86 (Jan) 1932, Treatment of Scleritis with Calcium-Adrenaline, *ibid* **88** 93 (Jan) 1932.

from 1 5,000 to 1 10,000, he also obtained localized mydriasis with a solution of epinephrine chloride in a dilution of 1 1,000 and a 4 per cent solution of calcium chloride. He had frequently seen in dogs that prolapse of the iris retracted promptly with this form of treatment.

Bielsky²⁰ in 1934 presented the results of a thorough study of the contemporary standing of iontophoresis in the field of ophthalmology. He and his co-workers established experimentally that the reflectory action of iontophoresis consisted in saturating the skin with ions which irritate the "receptor" nerve endings in the dermis. This irritation is transmitted to the vegetative nervous system, resulting in the con-

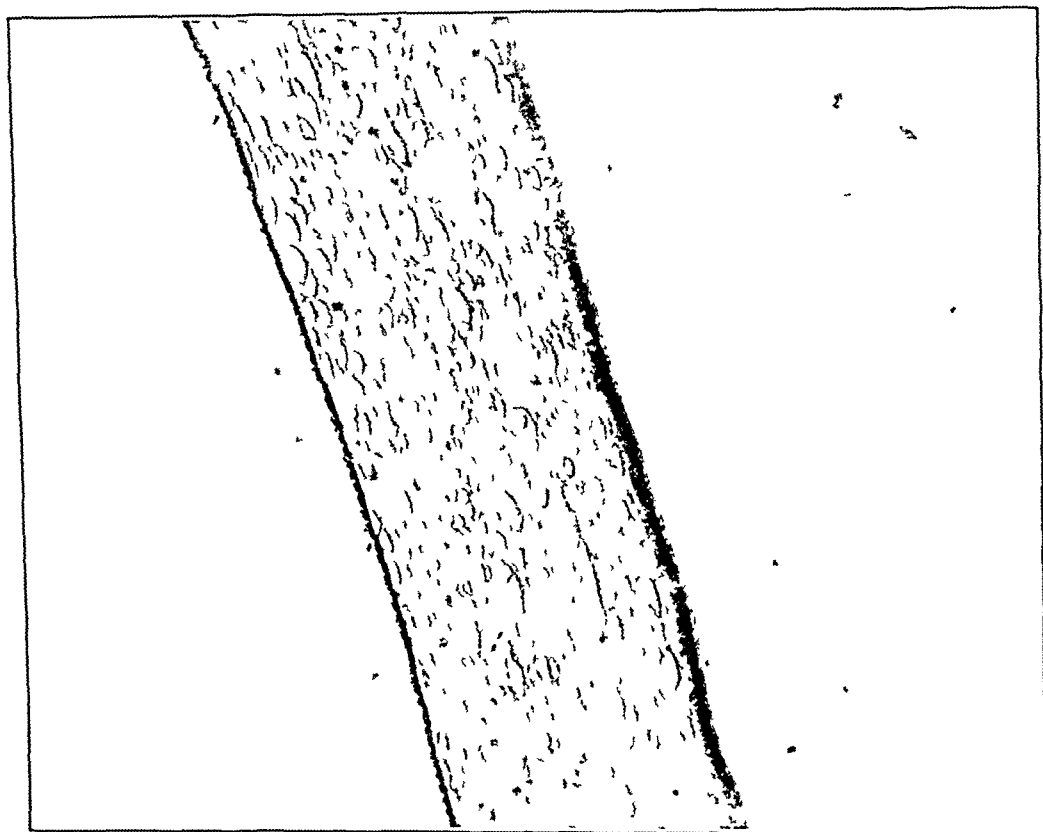


Fig 1—Reparative changes, second stage, dissemination of ions in epithelial layers, $\times 70$

densatory excitability of the voluntary muscles. If iontophoresis is used on cocaineized skin, no effect will be produced, which is one of the proofs in support of this theory. Since the vegetative nervous system most likely regulates all trophic processes in the tissues and in the metabolism of each cell, there is the possibility that the vegetative tonus will be increased or decreased, thereby influencing the physiologic functions and the pathologic deviations in the life of the tissues. Bielsky recommended more experimentation and a better knowledge of the

²⁰ Bielsky, A. Iontophoresis in Ophthalmology, *Sovet vestnik oftal* 4 123, 1934.

chemical and biologic processes of the tissues in order to obtain better results with iontophoresis

METHOD OF TREATMENT

Iontophoresis is a method of treatment by which drugs are introduced into the body by an electric current. For this purpose the galvanic current is used, which is uniformly constant, in one direction and at low tension, the rate of flow is from 1 to 30 milliamperes at a voltage up to 70. The direct current that we used was derived from a series of dry cell batteries. It was preferred to a current supplied by a dynamo

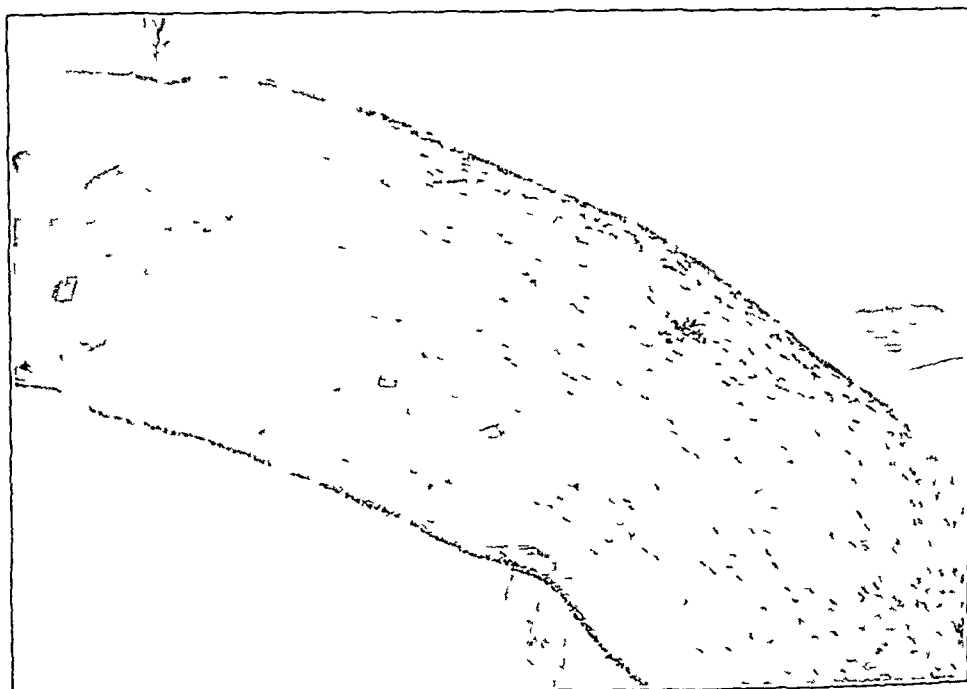


Fig 2—Reparative changes, third stage, edema of the corneal corpuscles, $\times 65$

or motor generator because the flow is always constant. The advantage of this simple instrument made up of dry cells is its dependability of smooth current, the ease of handling, the convenience of portability and the lack of danger from the electric current. The only and slight disadvantage is the necessary replacement of the dry cell when it deteriorates, but this is an inexpensive and simple procedure.

The instrument is equipped with a milliampere meter to measure the dosage, it also indicates the decline of the strength of the battery. There is a dial regulator to control the strength of the current and two poles plainly marked for their respective polarity. The conducting cords are of flexible copper wire covered with rubber insulation, the electrodes may be made of different metals. We chose a small block of tin plate

covered with a layer of felt. The purpose of the felt is to diffuse the products of electrical decomposition that takes place around the metal electrode and thus prevent injuries. The pad is covered each time with a clean piece of sterile gauze in order to have a clean surface for the treatment.

The principle of ionic treatment consists in the transportation of ions of a certain chemical composition by means of the electric current so that they are brought into contact with the tissue ions and intracellular organisms. The theory of ionic medication is rational. Failures are due to improper technic, imperfect penetration of ions, formation of

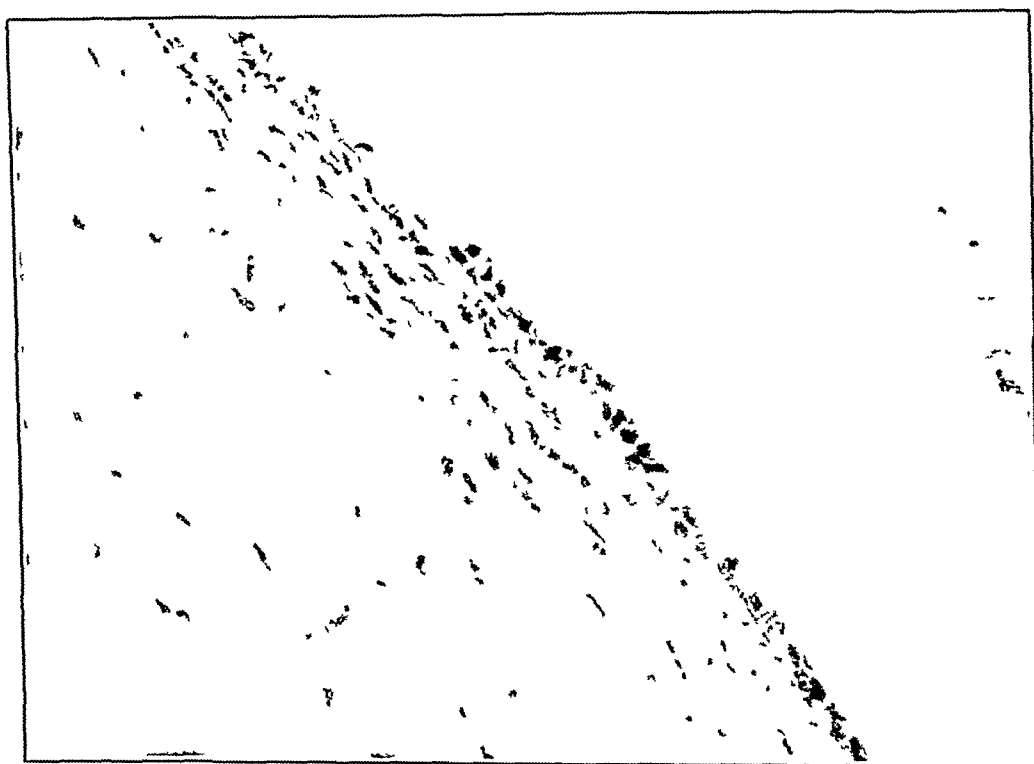


Fig 3—Reparative changes, third stage, edema of the corneal corpuscles, $\times 260$

insoluble compounds with the plasma and the neglect of the safety rules in the use of the galvanic current. Best results follow the application of a current of from 2 to 3 milliamperes per square centimeter, with a duration of the treatment from two to four minutes. The current should be turned on and off gradually. If the metallic part of the electrode touches any part of the treated surface, the caustic product formed under the metal electrode will cause a tissue damage varying from erythema to erosion.

In order to avoid damage to the tissues, we examine the electrode for defects in the covering and for uniform saturation before each treatment. The electrode should never be applied over a denuded area.

It is more difficult to treat diseases of the eye with ionic medication than diseases of any other part of the body, for two reasons, first, the anatomic position of the eye makes it difficult to apply the electrode properly, second, a strong current cannot be applied to force the penetration of the ions into the deeper tissues on account of the sensitivity of the cells of the eyes

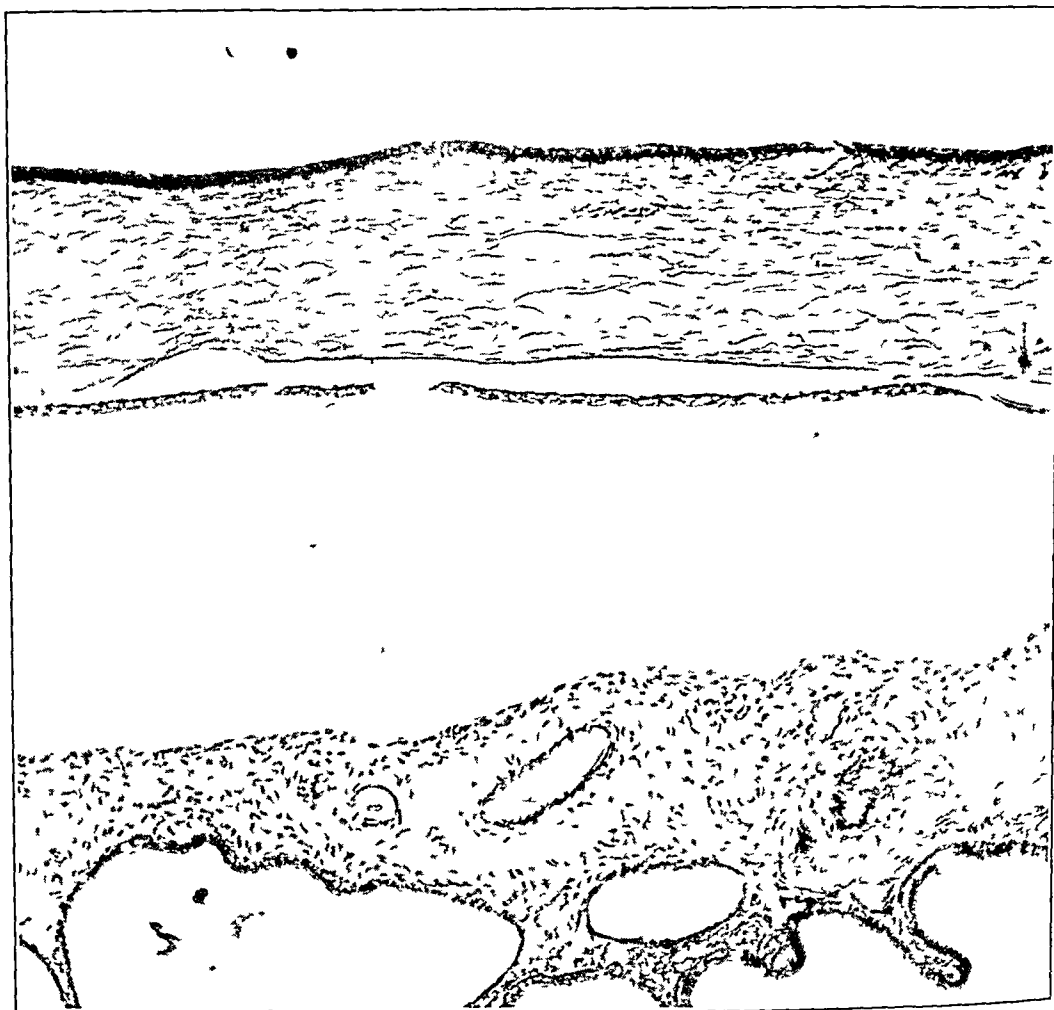


Fig 4—Borderline changes, first stage, mild vacuolation, $\times 100$

EFFECTS OF IONIZATION ON CORNEA

To find out what effects ionization would have on the cornea, we treated healthy corneas of rabbits with various solutions. It became necessary to record the experimental effects of ionization clinically as well as histologically. Rabbits were used exclusively, and all corneal ionizations were done with the animals under ether anesthesia. The histologic sections herewith described were prepared by the paraffin

method and routinely stained with hematoxylin and eosin, except in special instances in which selective methods of staining were indicated. The contralateral untreated eye of every animal received the same technical handling from enucleation to staining to serve as a control, misinterpretation of technical artefacts thus being avoided.

We employed solutions of zinc sulfate, zinc chloride, barium chloride, sodium chloride, colloidal sulfur and quinine bisulfate. Examinations by histologic methods have shown that ordinarily the chemical salt solution will enter the corneal tissues and be disseminated through them for variable depths, as seen by the tissue reactions in the stained preparations. The distance of penetration may be increased even to

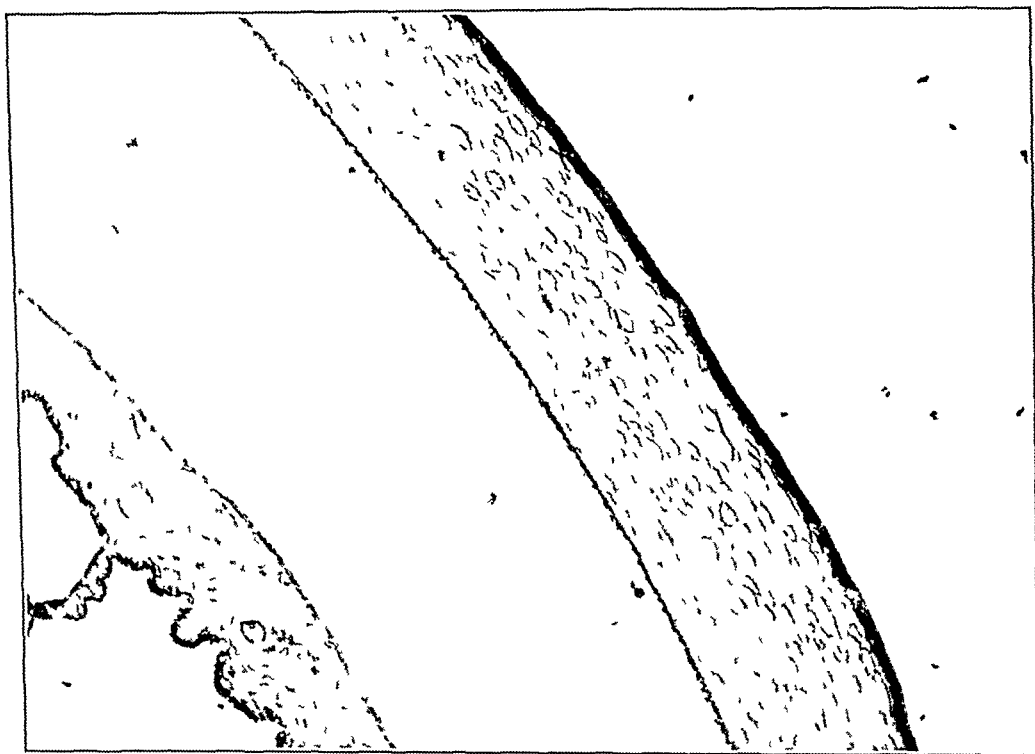


Fig 5—Borderline changes, third stage, distortion and displacement of fibrillae, $\times 65$

the posterior pole of the eyeball, which invariably will result in a permanent pathologic change. Although our object and primary intention was to observe the advantageous effects of ionic medication, it would also be well to mention some of the pathologic manifestations which were encountered.

The effects obtained in the cornea with ionization with different solutions follow:

Zinc Sulfate—Various grades of pathologic change can be induced with ionization with zinc sulfate, depending on the strength of the solution. It appears that only the 0.25 per cent solution is satisfactory, providing a current of not more than 1 milliampere is applied for not

over three minutes. In our experiments the 0.5 and 1 per cent solutions consistently showed a tendency to produce vacuolation in the substantia propria. A 1 per cent solution applied for a period of over five minutes always showed rupture of Descemet's membrane.

The healing process with this salt is considerably retarded, which makes it necessary to increase the time interval between treatments. Enough time is given to insure the return of the tissues to their absolute normal resting state. If this is not permitted, it has been found that the tissue changes will continue from that point toward a pathologic change, in other words, the ionization will be carried past a safety

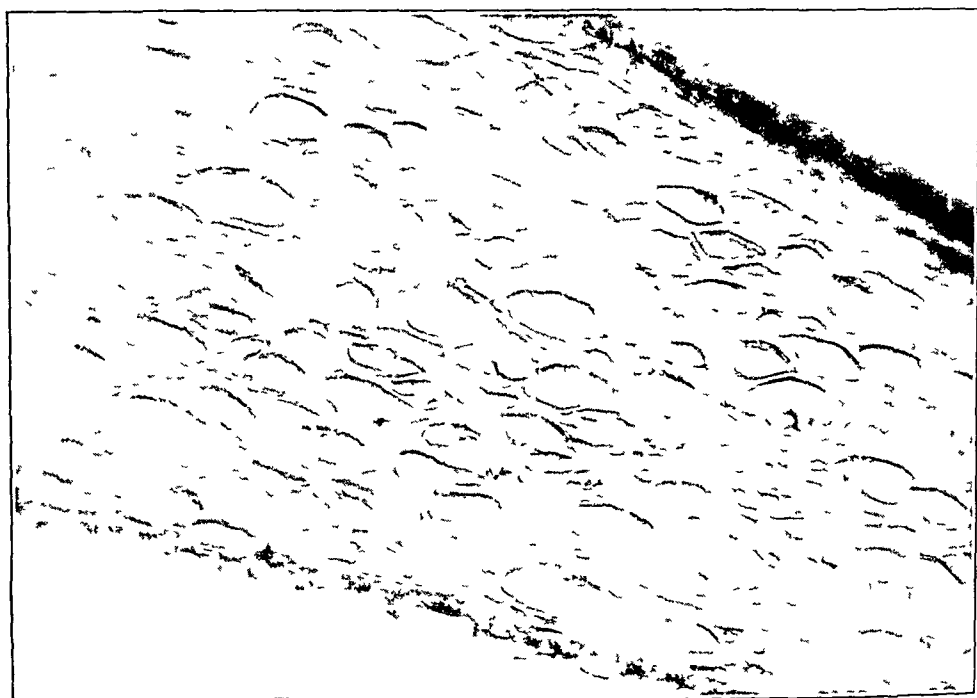


Fig. 6—Borderline changes, third stage, distortion and displacement of fibrillae, $\times 260$

point, since the next treatment is presumably given while the tissue is supposed to have returned to normal.

Zinc Chloride—Ionization with zinc chloride was carried out in only a small percentage of our experiments since pathologic changes were always found in varying grades of severity, even when high dilutions were used. Marked edema resulting in bulging of the cornea or ulceration was produced. Microscopically, inflammatory hyperplasia of the epithelial elements and exudative inflammatory infiltration of the stroma were found. Occasionally rupture of Descemet's membrane was seen.

Physiologic Solution of Sodium Chloride—The tissue changes found on ionization with physiologic solution of sodium chloride were

so slight that the treatment was adjudged to be of no therapeutic value. Since it is necessary to produce a temporary alteration in the tissues, of which this solution was incapable, we saw no advantage in continuing with it.

Colloidal Sulfur—Ionization with colloidal sulfur is not without danger, since particles of sulfur tend to polarize and form minute particles of highly charged irritants in the tissue. Microscopic sections showed peppered areas of erosion in the corneal epithelial surface.

Quinine Bisulfate—For ionization with quinine bisulfate, a dilution of 1:200 appeared to be the solution of choice. The only changes

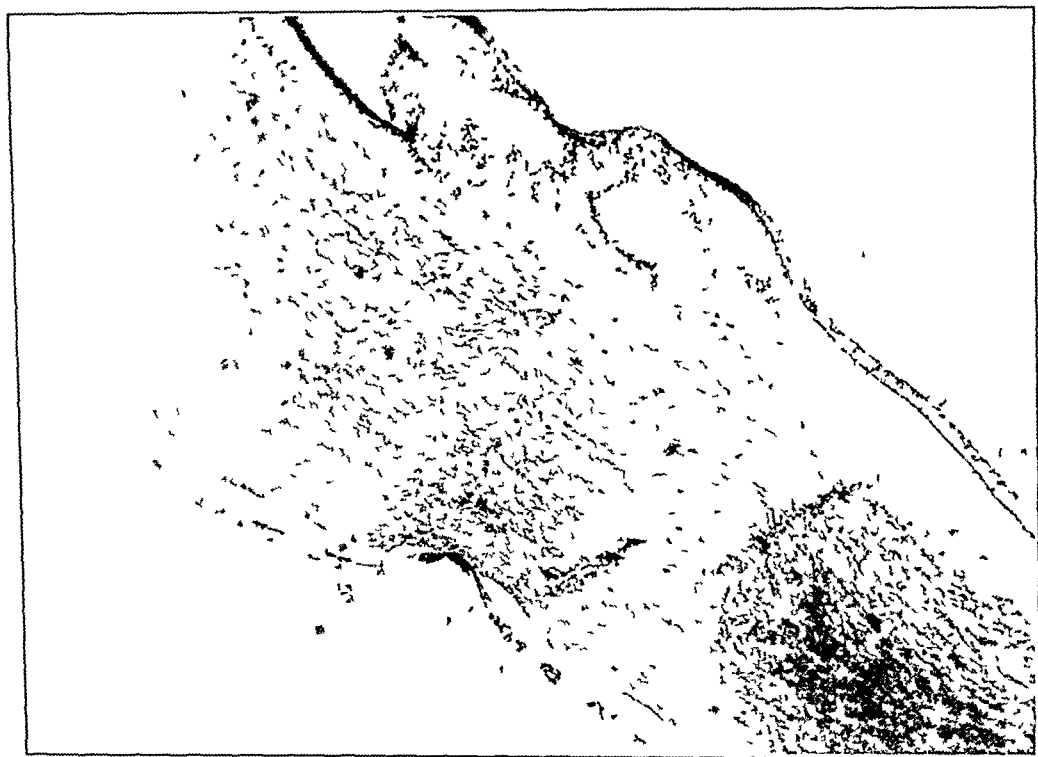


Fig 7—Pathologic changes, first stage, diffuse inflammatory infiltration, $\times 65$

observed were of a temporary nature, such as edema with a return of the tissue to normal, mild coarctation of the lamellae and swelling of the corneal corpuscles. None of the animals suffered any untoward effects, even after the use of as high a concentration as a 10 per cent solution for as long as ten minutes with a current of 25 milliamperes.

CLASSIFICATION OF TISSUE CHANGES

There is a definite sequence of tissue changes in corneas subjected to ionization. The entire process may be divided into three classes: reparative or therapeutic changes, borderline changes and pathologic or destructive changes. Each type of change is seen in several stages.

Reparative or Therapeutic Changes—Such changes are only transiently pathologic, and the tissues will return to normal after the response to ionic stimulation ceases

First Stage This stage consists of ionic irritation and is a superficial reaction which does not extend past the anterior lamina elastica. It is seen clinically as haziness of the cornea.

Second Stage Dissemination of the ions in the stratified pavement epithelial layer make up the second stage. The clinical appearance is that of a light powder-like sprayed area with hazy margins.

Third Stage This stage consists of edema of the corneal corpuscles subjacent to Bowman's membrane, they are distorted but do

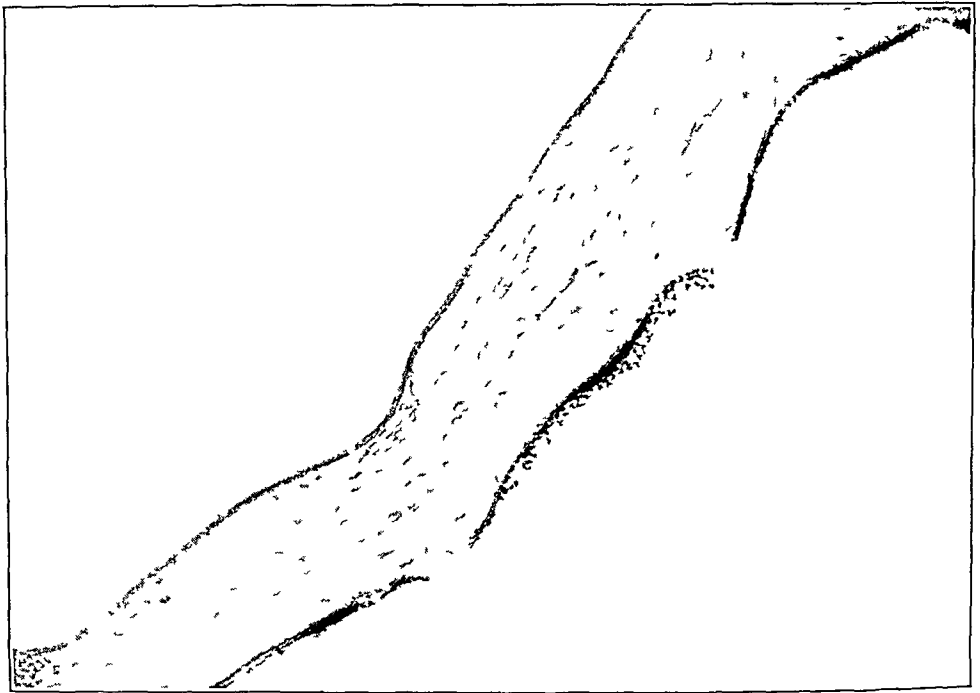


Fig 8—Pathologic changes, second stage, hyperplasia of epithelial elements of the posterior lamina elastica, $\times 65$

not lose their stellate forms. The lacunar spaces are swollen. Clinically, the appearance is that of a moderate degree of opacity.

Borderline Changes—These changes are of an indefinite nature.

First Stage This stage consists of mild vacuolation which tends to crush or displace the lamellae, the tissue is probably reclaimable. Clinically, diffuse haziness is to be seen.

Second Stage At this stage distortion and rupture of the corneal corpuscles occur. It is speculative whether the tissue is reclaimable. Clinically, a mild interstitial keratitis is present.

Third Stage This stage consists of marked distortion and displacement of the fibrillae with edematous fluid.

Pathologic or Destructive Changes—When ionic stimulation is carried past a point at which the return of the tissue to normal resting state is impossible, resulting in a permanent pathologic injury, the changes are considered pathologic or destructive.

First Stage This stage consists of diffuse inflammatory infiltration with polymorphonuclear leukocytes, plasma cells and collagenous histiocytes, later these cells are mainly replaced by lymphocytes. Newly formed capillaries may be seen. The cornea bulges forward, and a deep keratitis is present.

Second Stage Inflammatory hyperplasia of the epithelial elements of the posterior lamina elastica occurs during the second stage, and a



Fig 9—Pathologic changes, third stage, ulcerative necrosis, with a ruptured Descemet's membrane protruding into the anterior chamber, $\times 65$

thickened tail-like tag of Descemet's membrane protrudes into the anterior chamber following rupture.

Third Stage This stage consists of dehydration, subsequent coagulation or ulcerative necrosis. Clinically, the appearance is that of ulcerative keratitis with inflammatory exudation.

Fourth Stage At this stage reparative fibrosis or scar tissue formation occurs.

EXPERIMENTAL STUDY OF EFFICACY OF IONIZATION

It is the reparative or therapeutic changes with which we are concerned. The rationale of ionic medication appears to be in promoting

temporary tissue changes, on the return of the tissue to a resting state, the rehabilitation of the surrounding normal tissue will commence regeneration in the adjacent diseased tissue. In the cornea with the normal absence of blood vessels the response to ionic medication can naturally be expected to be different. Stimulation with an electric current causes fluid to be brought and interdispersed in the lamellae. The corneal corpuscles at first respond by becoming mildly edematous. As this

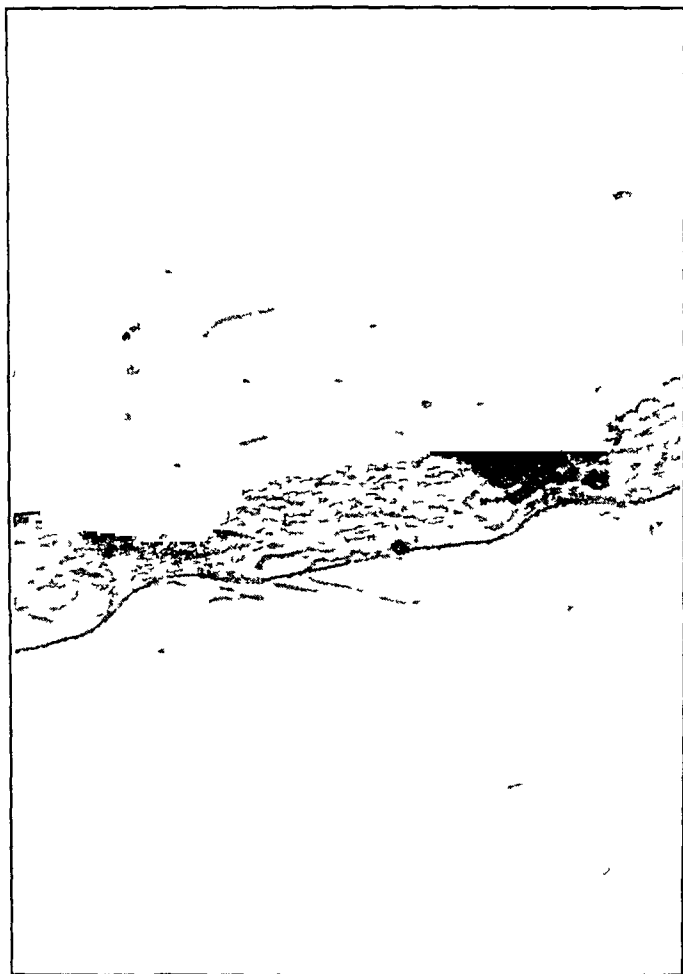


Fig 10—Pathologic changes, fourth stage, reparative fibroses (scar tissue) formation, $\times 65$

process continues, the accumulation of fluid is also carried into the lacunae, and there is intracellular pressure on the individual lamellae fibers and corpuscles of the substantia propria. It is this modified type of cellular coarctation which is the first step toward regenerative irritation. By repeating this process at periodic intervals, the alternating irritations and relaxations stimulate the tissue in continuing with its reparative function.

In order to prove the participation of the quinine bisulfate in the medicating process after ionization, we have decided on microchemical tests, since we failed to demonstrate the quinine salt in the sectioned cornea. These tests are strictly comparative and should be attempted only in the presence of a normal control, e. g., the contralateral untreated cornea of the same animal. Hence comparison is drawn between the epithelial scrapings of an ionized cornea and an untreated corneal surface from the same animal. At first we used the thalleoquin reaction, thalleoquin being the resultant bromine compound of quinine bisulfate. By this method a treated cornea is scraped with a blunt scalpel, and the epithelial elements are fixed on a clean slide. The preparation is treated with a drop of full strength ammonia water, and then a drop of liquid bromine is added. A cover slip on the resulting solution will make the material ready for microscopic examination with a high dry objective. The corneal scrapings which have been treated with ionization with quinine bisulfate will take on an emerald green color due to the formation of thalleoquin. The objection to this method, of course, is the irritating effects of the fumes from the ammonia and bromine reagents on the examiner's eyes.

When a number of examinations are to be made, we favor the herapathite test, since it is more convenient to perform and permanent mounts can be made for reference. Briefly, the method consists of scraping the treated cornea while the animal is still under anesthesia and mounting the material on a slide. Scrapings from the untreated eye should always be obtained and placed along side the previous smear on the same slide, but the two specimens should not be permitted to become confluent. If the material should be clumpy, a little saline solution may be used to emulsify the preparations, and thin smears can then be made. The slide is carefully fixed by heat after being permitted to dry at room temperature. We have previously used egg albumin slide fixation but found this step unnecessary. The slide is flooded for two minutes with 10 per cent ammonia water and washed off with a solution of iodine and sulfuric acid. (The reagent consists of 50 cc of 95 per cent alcohol, 2 drops of concentrated sulfuric acid and 2 drops of tincture of iodine.)

The preparation is gently heated by passing it through a flame. When it is dry and still warm, it is mounted in cedar oil, or for permanent mounts, in neutral Canada balsam. A large no. 1 cover slip is used to include both fields of scrapings. The new substance, herapathite, which is an iodine compound of quinine bisulfate, is seen in the ionized epithelium as an iridescent, greenish, refractile countenance, as compared with the light, reddish brown, opaque epithelial cells of the untreated cornea. Old preparations will fail to give this reaction, hence solutions should be in readiness for use as soon as the scrapings are obtained.

CORNEAL DYSTROPHY

Near the termination of experiments of the type described we had the good fortune to encounter 2 instances of corneal dystrophy, which are reported here. In general, certain consistent clinical findings are presented by this condition. Changes of a degenerative nature which have an obscure origin are frequently found in the corneal epithelium. They have a preference for the exposed area of the palpebral fissure, they appear as a rule bilaterally, mostly at puberty, and progress slowly. They have been described by different names, but apparently all belong to the same clinical group, in different stages of the disease. Essential changes appear to be hyaline deposits between the corneal lamellae, later, the lamellae and corneal corpuscles separate, swell up and disintegrate, flattening the epithelium and finally destroying Bowman's membrane.

The causation of corneal dystrophy is unknown. Several theories were advanced, the condition being blamed on impaired nutrition, endocrine insufficiency, dystrophy of the nerve fibers and sequela of corneal tuberculosis and consanguinity. Fischer has demonstrated a close relation between the transparency of the cornea and its water content. If the cornea loses or takes up more than about 20 to 30 per cent of its water content, it becomes semitranslucent. He has also shown that the imbibition of water is related to the integrity of the epithelium and endothelium and that if either layer is injured the cornea becomes permeable to water in both directions.

Most of the patients have myopia or myopic astigmatism with only slight improvement with correcting glasses. According to all authors, treatment has been unsatisfactory.

CASE 1—Mr W. A. S., aged 50, an American, a barber, was referred for examination on May 12, 1937. The family history revealed nothing noteworthy, and there was no history of intermarriage. The eyes of his two children who were over 20 years of age, were in good condition. The patient had had scarlet fever, mumps and measles during childhood. Ten years before examination he had had a gonorrheal infection. He had never had any ocular trouble. Three years before examination he was given a presbyopic correction, the eyes were found in good condition at that time. He was a moderate smoker. He ate plenty of fruits and vegetables and had meat daily. He first noticed foggy vision in the right eye in the early part of May 1937, while reading and cutting hair. There had never been any pain or redness of the right eye.

External examination of the right eye showed the lids to close well, the palpebral opening was of normal width. The eyeball moved well in every direction. The palpebral and bulbar conjunctivae were smooth and pale. In the lower half of the pupillary area the cornea showed a diffuse opacity, about 3.5 by 4 mm in cross diameters. There was a wide arcus senilis, and a clear corneal space was seen between the opacity and the arcus senilis. The corneal sensitivity diminished to touch. The pupil was round and reacted well. Vision was 20/50, and tension was 20 mm (Schiotz).

The left eye was normal in every respect Vision was 20/20, and tension was 18 mm (Schiotz)

Ophthalmoscopic examination showed clear lenses and vitreous Both disks were well defined and vascularized The vessels were normal in size and in their course Macular areas appeared normal

Biomicroscopic examination of the right eye revealed a grayish diffuse opacity in the lower half of the pupillary area, somewhat circular and extending beyond the lower border of the pupil It consisted of numerous small dots, lines and confluent patches, irregular in shape and not well defined The covering epithelium was uneven and edematous and bulged forward in the formation of small blebs The opacities extended downward to the middle of the stroma The corneal nerves were not enlarged No vessels were visible in the cornea Descemet's membrane showed wrinkling Many floating cells were seen in the anterior chamber The iris pattern was well defined, and a few blood vessels were visible There was no staining with fluorescein

General physical examination did not reveal any abnormal condition Complement fixation of the blood for syphilis was 4 plus

The patient was ordered to apply hot applications and use drops of ethylmorphine hydrochloride and of carotene in oil as preliminary treatment, before examination was completed On June 1 the patient started antisyphilitic treatment, and at the same time we began ionization of the cornea with a solution of quinine bisulfate On June 13 there was noticeable a clearing of the opacity The improvement continued, and vision on August 31 was found to be 20/30 At the last examination, made on Jan 4, 1938, after twenty-four ionization treatments, the following picture was observed Below the lower pupillary border, in Bowman's membrane, there was a small grayish patch the size of a pinhead, with pigment in the epithelium A wavy pigment line was seen in the pupillary epithelium The posterior corneal surface was studded with dustlike pigment cells, an occasional floater was seen in the anterior chamber The fundus was normal The corrected vision with a plus 0.75 cylinder, axis 165 was 20/20, and the patient was able to read Jaeger test type 1

CASE 2—Mrs P C, aged 44, a housewife, Russian by birth, was referred for examination on Aug 28, 1937 The family history was essentially unimportant There had been no intermarriages She had had two children, one was myopic and wore correcting lenses The past history disclosed that the patient had had typhoid thirty years previously and hysterectomy eleven years previously for fibroid tumors She had never had any inflammation of the eyes She was given reading glasses three years previously The eyes did not show any defect She did not smoke or drink and ate mostly vegetables and fruits and occasionally meat The present complaint consisted of blurred vision of the right eye of several months' duration The eyes were never red or painful

External examination of the right eye showed the lids to be normal The palpebral opening was of normal width, and the movements of the eyeball were in normal limits The bulbar conjunctiva was slightly injected In the pupillary area of the cornea there was an irregularly outlined, roundish, opaque area, 4 by 4 mm in size, which did not stain with fluorescein or mercurochrome The periphery of the cornea was clear There was diminished corneal sensitivity The pupil was round and reacted well Vision was 20/100, and the tension ranged from 15 to 18 mm (Schiotz)

The left eye was normal Vision was 20/20, and the tension ranged from 15 to 18 mm (Schiotz)

Biomicroscopic examination of the right eye showed the pupillary area of the cornea to be occupied by an irregularly outlined, grayish opacity, consisting of numerous areas the size of a pinpoint, originating in Bowman's membrane and extending toward the center as far as the anterior third of the parenchymal stroma. The epithelium was smooth. There were no enlarged corneal nerves and no corneal vascularization. The cornea did not stain. Two small precipitates were seen on the posterior corneal surface, a few floaters were observed in the anterior chamber. The iris was normal.

The patient was referred again for a general examination which did not show any deviation from the normal. Examination of the blood revealed 5,260,000 red blood cells with a hemoglobin content of 68 per cent, and 12,600 white blood cells. The sugar content of the blood was 62 mg per hundred cubic centimeters of blood. Complement fixation of the blood for syphilis was negative. Urinalysis gave negative results.

Preliminary treatment consisted in the use of hot fomentations and drops of ethylmorphine hydrochloride and the application of an ointment containing 2 per cent quinine bisulfate. On September 16 the patient began treatments with iontophoresis, a 1:200 solution of quinine bisulfate being used. On October 30 only a small opacity could be seen below the pupillary border. The last examination, on December 14, after fourteen treatments, showed the following picture. The bulbar conjunctiva was clear. At the lower pupillary border of the cornea, at 6 o'clock, irregularly outlined, two faint subepithelial opacities the size of a pinhead were seen. In the pupillary area there were several faint small opacities the size of a pinhead. There was a pigment line in the epithelium, the anterior chamber was clear and the fundus was normal. Uncorrected vision was 20/20 + 3, and with a +0.25 cylinder, axis 165 vision was 20/20. The patient was able to read Jaeger test type 1.

SUMMARY AND CONCLUSIONS

A review of ionic medication as used in the practice of ophthalmology is presented.

Animal experimentation for the purpose of investigating the efficacy of various medicaments and of establishing the different effects of ionic medication has been systematically carried out.

Two patients with corneal dystrophy have been successfully treated by iontophoresis.

We believe that this method of treatment has a definite place in ophthalmic practice.

INFERIOR IRIDOTOMY IN OPERATIONS FOR CATARACT ON EYES WITH POSTERIOR SYNECHIAE OR PUPILLARY MEMBRANE

VALUE OF OPERATION

PAUL A CHANDLER, M D
BOSTON

The operative procedure to be discussed in this article may not be new to some ophthalmologists. I have not seen it described, however, in the literature or in textbooks on ophthalmology and believe it to be of sufficient value to deserve emphasis.

Not infrequently extraction of the lens must be performed when, as the result of preceding iritis or iridocyclitis, the iris is adherent to the lens and there is more or less pupillary membrane. The need for removing the lens may be due to senile cataract, to complicated cataract or to the impossibility of obtaining an effective pupil without sacrificing a clear lens. In some cases extraction of the lens is required to relieve intractable secondary glaucoma resulting from iridocyclitis. In such cases operation for cataract should not be undertaken until the inflammatory process has long been quiescent, but in spite of this precaution surgical intervention often causes a flare-up of the inflammation. Even after intracapsular extraction one may see a small drawn-up pupil blocked by an inflammatory membrane. Further operation is then necessary to obtain satisfactory vision.

When the inflammatory process is again quiescent after extraction of the lens, one may be able with a Ziegler knife or a small narrow cataract knife to divide the membrane and iris and obtain a good pupillary opening. In some cases, however, the membrane may be so tough that it is impossible to obtain a good opening without undue trauma to the eye. A more effective procedure, although usually involving more risk, is iridotomy with one of the various types of scissors through a keratome incision. Either of these operations, especially the first, may prove ineffective if hemorrhage occurs, as it not infrequently does, from dilated vessels of the iris. Furthermore, both operations require cutting

Read at the meeting of the American Ophthalmological Society, San Francisco, June 10, 1938

into the vitreous, and secondary glaucoma of a serious nature may result from the escape of vitreous into the anterior chamber. They may thus prove to be more serious than the original cataract extraction.

Another type of case in which the extraction of a cataract may result in a drawn-up, ineffective pupil is that in which a previous filtering operation has been done for chronic glaucoma. There is almost always a certain amount of postoperative uveitis or iridocyclitis after such an operation. In spite of vigorous use of a mydriatic, posterior synechiae not infrequently occur, since it is seldom possible to obtain a wide pupillary dilatation postoperatively. Cataract extraction in these cases will be followed by a much higher percentage of drawn-up pupils and pupillary membranes than in a comparable series of cases in which there are no complications.

It occurred to me that one might be able to forestall the postoperative complications just discussed by performing iridotomy below at the time of extraction of the lens. The first person on whom I tried this operation was a young woman with severe iridocyclitis. The eye showed extensive posterior synechiae and pupillary membrane. Eventually the lens became opaque, and vision was reduced to the perception of shadows. Secondary glaucoma supervened, which was not satisfactorily controlled by the ordinary palliative medical and surgical procedures, the latter including iridectomy. When the inflammatory process eventually quieted down, it was decided to remove the lens in the hope that this might restore vision and also have a favorable influence on the glaucoma. At operation, after the usual incision was made, the synechiae were freed with a spatula. The Noyes scissors were then introduced, and the iris was split from the lower pupillary border nearly down to the root. Capsulotomy was done with a toothed forceps, and the lens was expressed. In spite of a severe postoperative flare-up of the inflammatory process, a wide pupil remained, and the visual result was good. Incidentally, the glaucoma was relieved.

Since then I have employed inferior iridotomy as a routine during cataract extraction in all cases in which there has been previous uveitis or iridocyclitis or in which a decompression operation for glaucoma has been performed. The extent of the iridotomy is graded according to the severity of the previous inflammation. When the inflammation has been mild, the iris may be split for only 1 or 2 mm, whereas when the inflammation has been severe the iris is always split nearly down to the root. The objection might be made that this will leave a large unsightly pupil, which would cause the patient to suffer from glare. This is, indeed, sometimes the case when a larger iridotomy has been performed than the subsequent behavior of the eye proved necessary. In most cases in which the previous inflammation has been severe, however,

even when iridotomy has been performed as far down as possible, the strong tendency of the pupil to be drawn up results in a nearly normal pupil, so that one would not suspect that iridotomy had been done. Even when a more or less unsightly pupil results, this disadvantage is more than offset by the excellent visual results and the usual avoidance of further operation.

During the past six or seven years, I have performed inferior iridotomy, as described here, on approximately 20 eyes. In only 1 case has further operation been necessary. In this case, in spite of the performance of iridotomy nearly down to the root of the iris and intracapsular extraction, a pupillary membrane and drawn-up pupil occurred, and poor vision resulted. Subsequently, the lower border of the membrane was easily freed from the iris with a knife needle, and the pupil immediately went down, leaving a good opening, which has remained.

The reports of 2 illustrative cases follow.

CASE 1—Mrs C H P, aged 68, had an ocular condition diagnosed chronic simple glaucoma. Both eyes were trephined in September 1931. There was moderate postoperative iritis, with the formation of posterior synechiae. Subsequently, gradual clouding of the lenses took place, until in December 1932 vision in each eye was reduced to 20/70. It was decided at this time to remove the left lens. At operation, after placing a suture on either side of the bleb, the usual incision was made, the section being completed in the cornea so as not to disturb the filtering cicatrix. Iridectomy was done above. The adhesions were freed with a spatula. The lens was removed in the capsule by the Verhoeff method. There was considerable postoperative iritis, and the pupil was drawn up. Since the bleb and the scar of the corneal section encroached on the pupillary area above, only a narrow slitlike pupillary opening remained. The final corrected vision equaled 20/30. Reading vision was satisfactory, but the patient had difficulty in getting around. In 1937 cataract extraction was done on the left eye. This time the operation was done exactly as on the other eye, except that iridotomy of about 4 mm was done below. There was moderate postoperative iritis, the pupil was drawn up to about its normal position. Corrected vision was 20/30.

CASE 2—Mrs M K, aged 50, had previously undergone iridencleisis on the right eye for chronic simple glaucoma. There were many posterior synechiae, a partial pupillary membrane and immature cataract. The visual acuity was 20/200. Combined cataract extraction had been performed elsewhere on the left eye, some of the capsule being left. There had been severe postoperative iridocyclitis, the pupil had become drawn up and blocked by a dense membrane, partly capsular and partly inflammatory. Three attempts had been made to make a pupil, once with a Ziegler knife and twice with the Noyes scissors. Each attempt was unsuccessful on account of bleeding of the iris. It was decided not to attempt further operation on this eye on account of the marked vascularization of the iris and membrane. Cataract extraction on the other eye was therefore decided on. After the usual section, posterior synechiae were freed with a spatula, and iridotomy 4 or 5 mm long was done below. The lens was removed in capsule, Verhoeff's technic being used. There was marked postoperative cyclitis, persisting for a number of weeks. Although the pupil was drawn up to about the normal level, a good opening remained, and the visual result was excellent.

CONCLUSIONS

Inferior iridotomy in cataract extraction on eyes that have been affected with iritis or cyclitis or that have been operated on for glaucoma is recommended to insure a permanent pupillary opening. It is simple to perform and perfectly safe, since the lens is still in place to protect the vitreous. In nearly all cases the resulting pupillary opening is adequate and permanent. No harm is done by the procedure, even if in spite of it the pupil closes up.

RESULTS OF AUTOTRANSPLANTATION OF CORNEA INTO ANTERIOR CHAMBER

THEIR SIGNIFICANCE REGARDING CORNEAL NUTRITION

TRYGVE GUNDERSEN, M D

BOSTON

The exact source of nourishment of the cornea and especially of its various layers, the epithelium, the stroma and the "endothelium" (mesenchymal epithelium), is not known. It is generally supposed that the cornea is a relatively inactive tissue with a low metabolism in which the nutritive requirements are not great, that under normal conditions it derives metabolites partly from the pericorneal blood vessels by a process of diffusion and partly from the aqueous humor. A more complete discussion of the subject of corneal nutrition and of the experimental evidence for the existing theories is given in another communication.¹ Direct evidence more conclusive than that heretofore produced as to the part played by the aqueous in corneal nutrition has been obtained by transferring a piece of normal cornea from one eye into the anterior chamber of the opposite eye and ascertaining its fate by clinical and histologic observations.

It is not a rare accident in corneoscleral trephining to force the trephine disk into the anterior chamber. Elliot² stated that it occurs in 16 per cent of cases in which trephining is done. Unless the disk carries infection into the anterior chamber or obstructs the trephine opening, no harm results. He quoted Elschnig as believing that "the aqueous has tissue-dissolving qualities which, in his opinion, is proved by the fact that the loose scleral disk, fallen into the anterior chamber during trephining, has completely disappeared in a relatively short time." Elliot stated that in his experience the disk becomes hidden by the sclera so that it cannot be observed.

It was my fortune to see such a piece of sclera in the anterior chamber of a patient on whom the late Dr. George Deiby had done

Read at the meeting of the American Ophthalmological Society, San Francisco, June 10, 1938

From the Howe Laboratory of Ophthalmology, Harvard University, and the Massachusetts Eye and Ear Infirmary

1 Gundersen, T. Vascular Obliteration for Various Types of Keratitis. Its Significance Regarding Corneal Nutrition, Arch. Ophth., to be published

2 Elliot, R. H. A Treatise on Glaucoma, New York, Oxford University Press, 1922, p. 573

the trephining operation of Elliot eight years previously. The disk was adherent to the surface of the iris 4 mm below the lower pupillary border and held in position by a delicate white membrane. There had been little if any shrinkage, and the color was that of normal scleral tissue. With the slit lamp, no foreign body reaction was visible and no blood vessels could be seen entering the disk. This clinical observation indicates that scleral tissue does not dissolve in aqueous but that it may live or be well tolerated as a foreign body. However, it throws no light on the effect of aqueous on corneal epithelium, since there is no epithelium on these scleral disks.

The viability of all corneal layers in aqueous humor was studied by introducing full thicknesses of cornea into the anterior chamber. Experiments were done on 17 cats and 10 rabbits.

In the majority of the experiments a large disk of cornea was removed from one eye of the animal with the mechanical trephine of von Hippel and immediately introduced into the anterior chamber of the opposite eye through a small incision made with a keratome. Except in 1 instance the disk became adherent to the iris, from which blood vessels soon entered the foreign tissue. Preliminary iridectomies were done on the recipient eyes of 2 cats and 2 rabbits in a fruitless effort to obviate this difficulty. In 2 animals attempts were made to keep the disk from adhering to the iris by dislodging it with the point of a dissection needle, but adhesions always reformed. In 3 instances full thickness strips of cornea 3 mm wide and 10 mm long were cut from one eye and threaded across the anterior chamber of the opposite eye through a transfixing incision made with a cataract knife. These experiments were no more successful than the others in preventing vascularization of the tissue introduced.

REPORT OF EXPERIMENT

The following report concerns the single instance in which the corneal disk did remain free in the anterior chamber.

On April 15, 1936, a male cat 5.2 Kg in weight and approximately 5 years old, was anesthetized by means of an intraperitoneal injection of 3 cc of dial. A disk of cornea was removed from the left eye with a mechanical trephine (3.5 mm blade) and immediately introduced into the anterior chamber of the right eye through a small corneal incision near the upper temporal limbus. There was no blood in the anterior chamber after the procedure. The eye was atropinized in order to give the transplant a large pupillary space in which to lodge. Immediately afterward the disk appeared slightly opalescent, it drifted about in the semiviscid new aqueous and showed a tendency to remain in the pupillary space.

The following day the implant was on the lens and surrounded by a delicate exudate, apparently fibrin, but was well separated from the free margin of the dilated pupil. No appreciable change occurred until the fourteenth day after operation, when the fibrin had completely disappeared and the corneal disk moved

about freely in the anterior chamber. The disk never again became adherent. With oblique illumination it appeared somewhat grayish, with the ophthalmoscope a bright fundus reflex could be seen through it, and with the slit lamp the beam of light traversed it quite like it does the normal cornea. The disk was always found in the lower filtration angle, but if the cat was turned over, making the upper angle dependent, the disk slowly sank across the pupil, to disappear under the sclera.

There seemed to be slight shrinkage in the size of the disk during the first six months of its presence in the anterior chamber. During this period it also became globular and slightly more opaque, simulating the appearance of a kernel of sago. No further changes were observed until its removal, on Dec 14, 1937, twenty months after its insertion into the eye. The disk was examined histologically by Dr Verhoeff, who submitted the following report

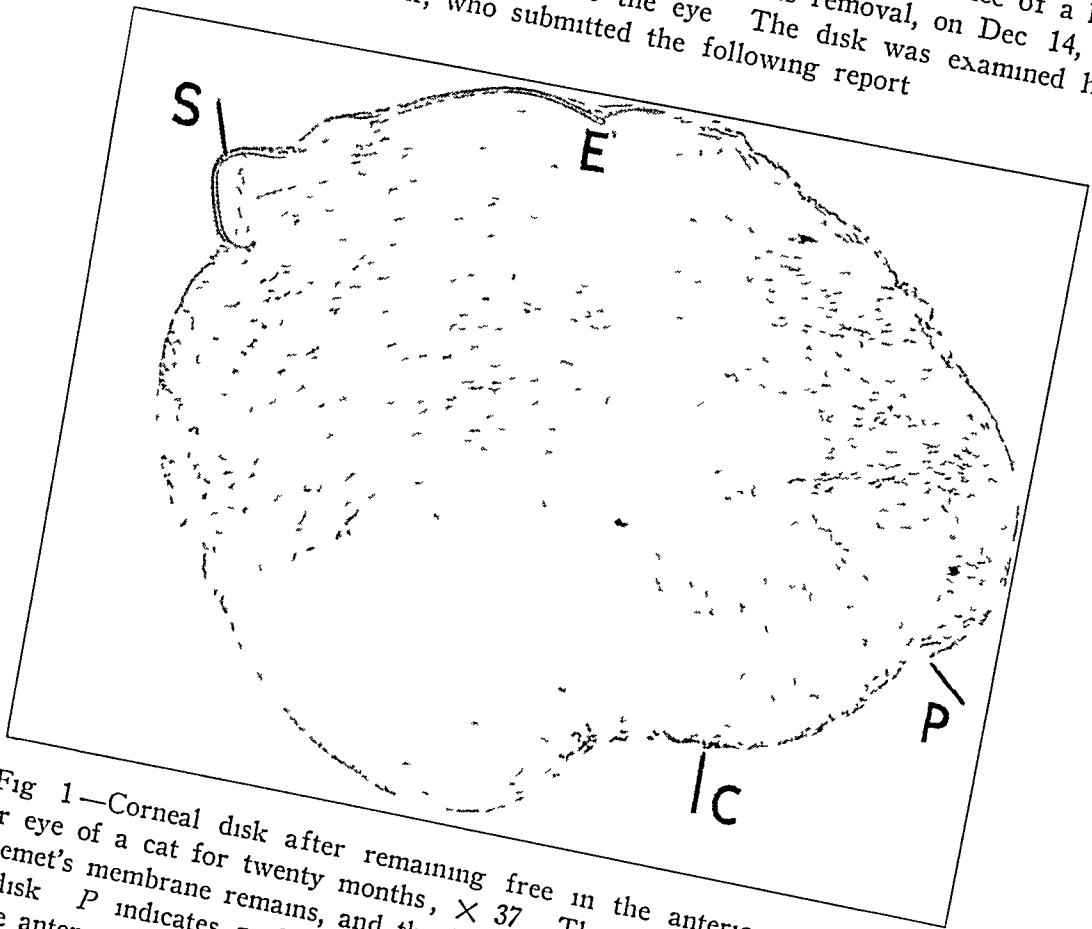


Fig 1—Corneal disk after remaining free in the anterior chamber of the other eye of a cat for twenty months, $\times 37$. The corneal epithelium is absent. Descemet's membrane remains, and the "endothelium" has grown entirely around the disk. *P* indicates proliferation of the corneal corpuscles in a small area on the anterior surface, *C*, chromatophores from the iris on the anterior surface, *S*, the space beneath Descemet's membrane, filled with delicate newly formed tissue, *E*, proliferation of endothelium in a furrow.

Histologic Examination of Free Implant—After fixation of the eye in Zenker's fluid, the disk was removed from the anterior chamber, embedded in pyroxylin and cut in serial cross sections. Staining was done with hematoxylin and eosin. A median anteroposterior section showed the disk to be roughly elliptic, with axes of 2.4 mm and 1.8 mm (fig 1). No corneal epithelium could be found on it in any sections. The stroma had an extremely sharp margin all around, showed no evidence of loss of substance or necrosis and was everywhere free from

infiltrating cells. Occasionally a few small vacuoles could be seen just beneath the surface. Except for some distortion due to change in the shape of the disk, the lamellae appeared normal. They stained with eosin about as uniformly as, although somewhat less intensely than, the lamellae of a normal cornea. The

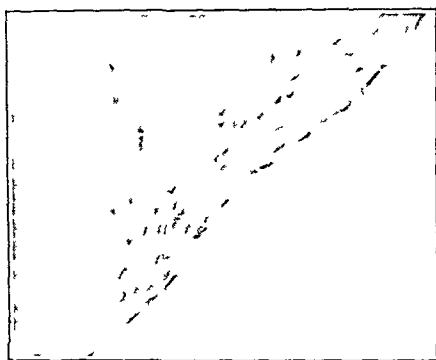


Fig 2—Area indicated by *P* in figure 1 under higher magnification

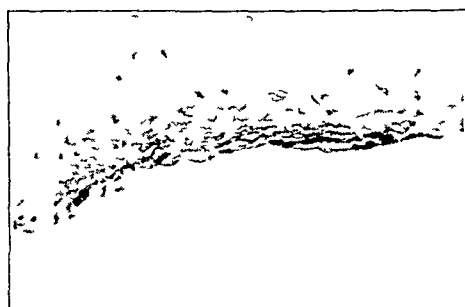


Fig 3—Area indicated by *C* in figure 1 under higher magnification, showing chromatophores. There is slight vacuolation of the stroma here



Fig 4—Area indicated by *S* in figure 1 under higher magnification, showing delicate tissue and stellate cells in space beneath Descemet's membrane

corneal corpuscles, however, were greatly reduced in number, especially in the central portion of the disk. In a small area just beneath the anterior surface they had proliferated as they do at the site of a corneal incision, but much less markedly (fig 2). At the cut edge of the disk, however, such proliferation was

absent In another small area on the anterior surface, evidently where the disk had been in contact with the iris for a time, there were a few chromatophores and several large swollen cells (fig 3) With the exception of a group of eight plasma cells seen in one section, no cellular exudate was found on the disk

On the posterior surface of the disk, Descemet's membrane remained, but was more or less wrinkled Small spaces caused by the membrane separating from the stroma were filled by extremely delicate tissue containing a few stellate cells (fig 4) The so-called endothelium (mesenchymal epithelium) not only was present over Descemet's membrane but had grown entirely around the disk, even over its anterior surface, from which the epithelium had disappeared In furrows resulting from the wrinkling of Descemet's membrane, the epithelium had proliferated into several layers, but elsewhere it consisted of a single layer Nowhere had it formed "warts" or a new layer of Descemet's membrane

Histologic Examination of Adherent Implants—Of the adherent implants, only five were examined histologically Two were adherent to the cornea, two others to the iris and one to both the cornea and the iris Each implant was sectioned in situ In each case the epithelium was entirely absent from the surface of the implant The corneal corpuscles were more numerous, and the corneal lamellae stained somewhat more intensely than in the free implant On the free surfaces of the implants "endothelium" was present In 1 instance a small epithelial cyst had formed where the disk had folded on itself, enclosing the original epithelial surface, which was thus completely removed from the aqueous The stroma was slightly vascularized

COMMENT

The clinical and histologic observations in these autotransplantation experiments show what changes occurred in full thickness pieces of cat's cornea which remained in the anterior chamber of the opposite eye for periods up to twenty months

The fact that the epithelium on the surface of each implant had entirely disappeared clearly indicates that corneal epithelium cannot subsist when nourished by aqueous alone It is well known that corneal epithelium can grow into the anterior chamber through perforating wounds, traumatic or surgical But this does not necessarily mean that the epithelium subsists on nutrients which it derives from the aqueous In every case of epithelization of the anterior chamber there are blood vessels in close approximation to the epithelium, which presumably feed it As a matter of fact, demonstration of new blood vessels is often necessary to establish the clinical diagnosis of this condition It is true that the epithelium sometimes forms cysts in the anterior chamber, but in these instances the conditions are not analogous to those of my experiment The cysts are invariably attached to the iris, the ciliary body or the posterior surface of the cornea and contain serum derived from blood vessels,

On the other hand, it is evident that the other corneal tissues can live almost unaltered in aqueous for a prolonged period For the stroma showed little change, although in the center of the free disk,

the region farthest removed from aqueous, the corneal corpuscles were greatly reduced in number. There was no hyalinization or evidence of inflammation in any of the implants. Descemet's membrane remained, and the endothelium had grown entirely around the free implant.

There was no definite reduction in volume of the implanted pieces of cornea. The free implant changed its shape from that of a disk 3.5 mm in diameter and 0.6 mm in thickness to that of an ellipsoid with a major axis of 2.4 mm and a minor axis of 1.8 mm. The calculated reduction in volume, based on the assumption that the disk had become a perfect ellipsoid, was no more than 0.21 cu mm. The free corneal disk, although gradually diminishing in transparency for six months after its insertion, never became completely opaque. Incidentally, these experiments indicate, contrary to the opinion of Elschnig, that a disk lost in the anterior chamber as a result of trephining is not dissolved by the aqueous.

CONCLUSIONS

It is evident from these direct experiments that corneal epithelium does not live when nourished only by the aqueous and excluded from its tear supply, that under these conditions the corneal stroma does live for a prolonged period, although its nourishment may be somewhat inadequate, and that the so-called endothelium not only lives but proliferates.

AN INTERN'S EXPERIENCES WITH THE VERHOEFF METHOD OF CATARACT EXTRACTION

PAUL H CASE, M D

PHOENIX, ARIZ

At the Massachusetts Eye and Ear Infirmary the most popular method for the intracapsular extraction of cataract is the "sliding method" of Verhoeff. As an intern at this institution I have had the privilege of assisting Dr. Verhoeff in many of his operations and of being taught by him the technic of his method. My experiences as a beginner with this method may be of interest to operators who have not yet employed it or who have not attempted intracapsular extraction by any method. I shall first describe the technic of the operation, then briefly give the results that I have personally obtained by its use and finally analyze the few of my cases in which the capsule ruptured.

TECHNIC

For anesthesia, 5 drops of a solution containing 4 per cent cocaine hydrochloride and 1 per cent pilocarpine nitrate are instilled at suitable intervals into the conjunctival sac, then 0.5 cc. of a 2 per cent solution of procaine hydrochloride with epinephrine is injected subconjunctivally at the insertion of the superior rectus muscle, and the same amount is injected into the lower bulbar conjunctiva and fornix. The purpose of the pilocarpine is to prevent the dilation of the pupil by the cocaine. A solution of procaine hydrochloride of the same strength is used to obtain paralysis of the muscles of the lid by the Van Lint method. The Murdock speculum is used, and during delivery of the cataract it is carefully supported by an assistant to avoid pressure on the eye. If the eye is prominent or the palpebral orifice small, canthotomy is done.

A suture through the tendon of the superior rectus muscle is used routinely as a bridle suture. The tendon, with the conjunctiva, is grasped at the scleral insertion, and a no. 6 white silk suture is carried through the tendon. The suture is then carried through the towel covering the head so as to be anchored when tied. If the patient is obviously a "good actor," the bridle suture is not tied. In any case it is not tied until after the speculum has been properly supported, and then not tightly. Owing to the subconjunctival injection of procaine hydrochloride, little or no discomfort is experienced when the suture is carried through the tendon.

Two corneoscleral "track" sutures are used routinely. The first step is to sever the conjunctiva from the limbus from 9:30 to 2:30 o'clock and then to undermine the conjunctiva sufficiently to permit its being easily pulled over the wound. Two sutures (no. 1 black silk) are inserted through the limbus, one at about 11 and the other at about 1 o'clock. Each suture is entered 1 mm. from the limbus and carried through the cornea and sclera for a distance of

about 2 mm at approximately one-third the depth of these tissues. The needle employed is a Berbecker needle (no 6, $\frac{3}{8}$ circle). A two-fifths section is made at or close to the limbus, so as to cut the original sutures about in half. A no 1 black silk, double-armed suture is passed through each of the suture tracks, the original black silk sutures serving as guides. After the tracks are rethreaded, the cut pieces of the original sutures are removed, leaving a single continuous suture in each track. This suture is carried through the conjunctival flap above, the suture end from the cornea being placed 1 mm from the free end of the conjunctiva and the suture end from the sclera being placed 2 mm farther back. Thus after the removal of the lens the conjunctival flap may be pulled down so as to cover the wound well when the sutures are tied and bring the edges of the wound closely and firmly together. At this stage Dr. Verhoeff performs a radial iridotomy upward by means of straight scissors. In some cases I have modified this by tearing each pillar from its base for a distance of about 2 mm on each side, after the iridotomy has been completed. This procedure gives a wider coloboma than does simple iridotomy but has the advantage of permitting better exposure of the lens. Possibly it may minimize the danger of secondary glaucoma. The coloboma is narrower than that obtained by iridectomy. The lens is exposed by prolapsing the pillars of the coloboma on each side or by pushing them aside within the eye by means of a spatula.

The lens is grasped at or just below its equator at 12 o'clock with the Verhoeff new capsule forceps. To bring the lens so that it bulges slightly above and can be more easily grasped, a slight amount of pressure is first exerted on the cornea at or near the limbus at 6 o'clock with a Verhoeff lens expresser. After the lens is grasped, pressure is continued with the expresser until the lens is dislocated, and at the same time moderate traction is exerted on the capsule with the forceps. The amount of traction that can safely be used is judged by the tentlike appearance of the capsule as well as by the feeling of resistance. The lens is removed by a continued combination of traction and pressure. The point of application of the expresser is varied according to the effect produced. If it is found advantageous, a rotary effect is produced during traction. It is extremely important to keep the lens against the scleral lip to prevent presentation and loss of vitreous between the lens and the sclera. If this precaution is carefully observed, the danger of loss of vitreous is small. Too often this point is neglected because the operator's attention is focused on manipulating the expresser.

As soon as the lens is delivered, the corneoscleral sutures are made snug but are not yet tied, care being taken that the iris is not drawn up into the suture tracks. If the suture through the tendon of the superior rectus muscle has been tied, it is cut. The iris is then replaced. The sutures are finally tied firmly, so that the lips of the wound are brought closely together, but not so tight as to cause wrinkling of the cornea. The final step in the operation is the toilet of the wound, which includes placing gentle traction with the capsule forceps at various points on the free margin of the conjunctiva so as to be sure that the conjunctiva smoothly covers the wound. No medication is instilled into the conjunctival sac after operation. Boric acid ointment is applied to the closed lids. A double ocular bandage is put on, and the patient is sent back to his room.

In discussing the technic of this operation with other ophthalmologists, frequent comment is made regarding the sutures to this effect: "They sound good, but they take too much time and are too much trouble to put in and rethread." The extra time required is often advantageous

During the first few minutes of the operation the patient is fearful and likely to be restless, but later he becomes more accustomed to the manipulations of the operator. Thus during the later part of the operation, when the lens is being delivered, he gives better cooperation. Rethreading the sutures is not difficult if done properly. The needle holder should be held as one does a pen or pencil to give better relaxation of the hand. Resting the hand on the side of the patient's face also allows relaxation. When one is rethreading a track, particular care should be taken to see that the needle is exactly parallel to the direction of the track. After a corneal track is rethreaded, the wound is sponged and the opening of the scleral track can be seen as a small black dot—the cut end of the track suture—and can then be easily entered. Some of the advantages of the corneoscleroconjunctival sutures are: 1. The lips of the wound are tightly held together. 2. There is less astigmatism after the operation because of the more exact approximation of the edges of the wound. 3. The danger of prolapse of the iris is greatly reduced. 4. There are fewer postoperative hyphemas. Postoperative hyphema most often comes from bleeding of the wound. The sutures prevent such bleeding by effecting perfect apposition of the edges of the wound and firm closure of the wound. In the experience of the operators who have adopted the track sutures, these advantages have been manifest. An additional advantage is that the patient may be allowed to sit up the day after operation, or in case of necessity he may be allowed to be propped up immediately after operation without breaking open the wound. The sutures should be allowed to remain two weeks and may be safely left in the wound until nearly ready to slough out. In removing them, adequate anesthesia is obtained by applying to their sites a solution of 2 per cent pontocaine hydrochloride or 10 per cent cocaine hydrochloride on a cotton-tipped toothpick.

I have employed the technic described in a series of 57 routine cases. In 51 of the cases the lens was removed in its capsule. There was a loss of vitreous in 3 of the 51 cases. In 6, the capsule was broken, and there was loss of vitreous in 2 of these. In this series the youngest patient was 43 years of age and the oldest 77. The average age was 58 years. Unfortunately the amount of prominence of the eyes was not determined for statistical study. In 1 of the 3 cases in which there was loss of vitreous the eye was quite prominent, but from several equally prominent eyes the lenses were removed intracapsularly without this complication. The lenses in 10 of the 51 cases were mature and in 41, immature. Postoperative hyphema occurred in 2 of the cases. A brief report is given of the cases in which the capsule ruptured, with a discussion as to the apparent cause of the rupture in each.

REPORT OF CASES

CASE 1—H B, a 69 year old man, was admitted to the hospital with an almost mature cataract in the left eye and an immature cataract in the right eye. He was found on general examination to have a blood pressure of 174 systolic and 100 diastolic and severe bronchial asthma with emphysema. The liver was enlarged to 3 fingerbreadths below the margin of the rib. Preoperatively, he was given ephedrine for the asthmatic condition and 3 grains (0.19 Gm) of pentobarbital sodium to quiet him generally. A good hold on the capsule of the lens was obtained, and the lens was about one half way out of the wound when the patient coughed, lifted his head off the table and tried to squeeze his eye. The result was rupture of the capsule and extrusion of the nucleus of the lens with a considerable amount of vitreous. The sutures were tied, and no attempt made to remove the remaining capsule. That the akinesia produced by the method of Van Lint was sufficient was evidenced by the fact that the patient could not close the eye at the end of the operation.

The cause of the ruptured capsule and loss of vitreous in this case probably was the lack of proper preoperative medication. Since the general condition of the patient and his inability to cooperate at the operation were known, he should have been given different sedation. The problem was talked over with several physicians, who stated that the barbiturates are poorly tolerated by patients of this class. As preoperative medication, they advised instead 30 grains (1.94 Gm) each of chloral hydrate and triple bromides in addition to epinephrine. Postoperatively, they advised 20 grains (1.29 Gm) of chloral hydrate every three hours in addition to epinephrine or ephedrine.

CASE 2—S M, a 63 year old man, entered the hospital with nearly mature cataracts in each eye. Slit lamp examination before the operation gave negative results. The pupils dilated only fairly well, but as complete dilatation is rarely obtained in elderly persons, this finding, unfortunately, was passed over lightly. The left eye was operated on first, and the lens came out easily in capsule. One week later the right eye was operated on. The operation progressed satisfactorily until an attempt was made to dislocate the lens. The temporal side dislocated easily with practically no effort, but the nasal side could not be dislocated, even after considerable pressure, and vitreous presented in the wound. The lens was then scooped, with a slight loss of vitreous. The capsule broke during this procedure, and a small amount remained stuck to the lower nasal margin of the iris. It was not until then that it was realized that there was a posterior synechia which prevented the lens from coming out in capsule.

Failure in this case to remove the lens in capsule may be considered due to carelessness in the performance of the operation. Since it was known that the pupil would not dilate fully before operation, a spatula should have been swept between the iris and the lens before an attempt was made to deliver the lens. For this purpose, Dr. Allen Greenwood has made a convenient spatula which conforms to the contour of the

surface of the lens. Fortunately there was a clear central opening, and the patient finally obtained vision of 20/30 in the eye.

CASE 3 (two operations)—P W, a 47 year old woman, two years before admission to the hospital began taking dinitrophenol (100 capsules) to reduce weight. As there was no loss of weight, the medication was stopped. About six to seven months previous to admission the vision in both eyes began to fail, and when she entered the hospital it was reduced to about 4/200 in each eye. Slit lamp examination showed haziness of the entire lenses with posterior cortical changes of the "mincemeat" type. The nuclei were but little sclerosed. Both operations progressed satisfactorily until an attempt was made to dislocate the lens. An effort was made for thirty-five minutes on the right lens and twenty minutes on the left lens before the capsule broke. There was no loss of vitreous.

The obvious explanation for the breaking of the capsules in this case was the age of the patient, 47 years. Lenses have been removed in capsule in still younger persons, but this age is about the borderline at which the zonular fibers may be expected to be fragile enough for intracapsular extraction. Intracapsular extraction should not have been attempted on the second eye.

CASE 4—A H, a 65 year old white woman, had a mature cataract in one eye and a normal second eye with vision of 20/20. Slit lamp examination gave negative results. The operation progressed satisfactorily until the lens was about one-third out of the wound, when the patient moved the eye and the capsule broke, considerable milky cortical material spilling into the anterior chamber. Most of the cortical material was removed by irrigation. The patient was discharged ten days after operation with vision of 20/50. She returned with acute iritis, large keratic precipitates, fibrin in the anterior chamber and secondary glaucoma. The iritis subsided in six weeks, and the final vision was 20/30.

It is possible, especially in view of the fact that the cataract was unilateral, that there had been a previous low grade uveitis which had caused the cataract and which was lighted up by the operation. The lens was swollen and tense, and the capsule was difficult to grasp. Therefore, it required but a slight movement of the eye to break the capsule. The larger the hold on the capsule, the more tense it becomes and the more easily it ruptures. Therefore especial care should be exercised when putting forceps on a swollen cataract. Incidentally, it is to be noted that a swollen cataract usually has to mold through the wound and thus tends to obstruct loss of vitreous.

CASE 5—F B, a white man aged 64, had high myopia in both eyes, a nearly mature cataract in the right eye and an immature cataract in the left eye. The operation on the right eye was successful in the delivery of the lens in its capsule. One week later the left eye was operated on. The vision in this eye before operation was 20/200 through a pinhole. It was expected that the zonular fibers would be extremely weak, as they usually are in a patient 64 years of age. The operation progressed satisfactorily until the capsule was grasped and an

attempt was made to dislocate the lens. Then pressure and traction were applied for fifteen minutes, when the capsule broke and the nucleus had to be expressed.

The explanation for the breaking of the capsule in this case seemed to be that the zonular fibers were unusually resistant.

SUMMARY

In a first series of 57 attempted intracapsular cataract extractions by the Verhoeff method, 51 lenses were removed in capsule. Loss of vitreous occurred in 3 of the cases in which the capsule was removed intact and in 2 of the 6 cases in which the capsule ruptured. The apparent causes of the rupture of the capsule were poor cooperation of the patient due to improper preoperative medication, 1 case, posterior synechiae which were not freed before extraction of the lens, 1 case, dinitrophenol cataract and the age of the patient (too young for intracapsular extraction), 2 cases, intumescent cataract, 1 case, high myopia and unusually resistant zonule, 1 case.

Clinical Notes

A COBALT BLUE FILTER FOR OBSERVATION OF THE FIT OF CONTACT LENSES

THEODORE E OBRIG, A B, NEW YORK

Pressure at the limbus or inside the limbus on the cornea itself is without doubt the reason for discomfort from contact lenses in at least 90 per cent of the patients who complain of inability to wear them except for short periods. Any contact lens which rests on any portion of the cornea cannot be worn for more than one hour and often for a much shorter time.

Many contact lenses which have been judged as clearing the entire cornea have been proved, with a new method of observation, to rest on or inside the limbus. This is not surprising in view of the decided difficulty of observing the actual clearance as the edges of the cornea are approached. Part of this difficulty is due to the assumption that the corneal portion of the trial contact lens completely covers the cornea.

Recent observations from castings of 200 eyes, reported in the ARCHIVES,¹ proved that the cornea was actually wider horizontally than the corneal portion of the contact lens in 95 per cent of the eyes observed and wider vertically in 30 per cent of the same group. It is therefore most necessary to check the clearance outside of the corneal portion of the trial contact lens.

It is easy to measure, with fair accuracy, the actual clearance of a contact lens from the cornea with a slit lamp over the area included by an imaginary line 2 mm inside of the sulcus. Observation from this point to 1 mm outside of the sulcus has remained practically impossible until recently. The intense white beam of a slit lamp or spotlight illuminates to the surface of the contact lens, the sclera and the iris to an extent which makes accurate observation of clearance most difficult.

I have found that a fairly dense cobalt blue filter placed between the source of illumination and the eye under examination is invaluable for observing the accuracy of the fit of a contact lens to which has been added before insertion a buffer solution containing 1 drop of fluorescein. Slight staining of the cornea by fluorescein in minute abrasions is also easily observable. In each instance the fluorescein will glow brilliantly in the blue light and at the same time the anterior chamber, the cornea itself and the sclera are not brilliantly illuminated. This does away with brilliant surface reflections, secondary illumination from reflections from the sclera, iris or corneal substance. A brilliant yellow-green will be observed wherever the contact lens is not in contact with the cornea or conjunctiva, and a dark area will be observed where the cornea touches the lens. Contact on the conjunctiva is indicated merely by the absence of fluorescein on the portion in contact.

1 Obrig, T E. Molded Contact Lenses, Arch Ophth 19:735 (May) 1938

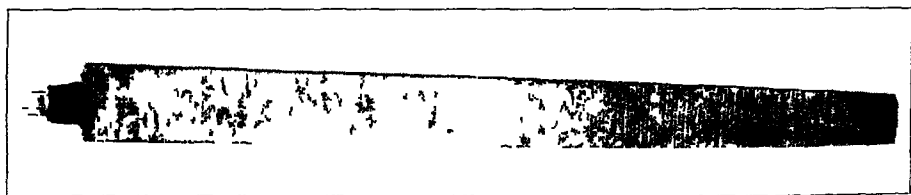
I have been using a new hand slit lamp² for making complete examination of the fit of contact lenses. The lamp is designed to give a brilliant white spotlight, a finely defined slit of light for oblique illumination and, most important, a cobalt blue filtered spotlight, originally intended for examination of the scleral and conjunctival blood vessels. It is compact, light and easily handled. It is an ideal instrument for this type of work.

2 This lamp is manufactured by Fryxell and Hill, of New York.

BLEPHARITIS COMB

JAMES W. SMITH, M.D., NEW YORK

In cases of blepharitis, particularly of the ulcerative type, great difficulty is experienced in removing the densely adherent scales. In many instances, even after the prolonged use of ointment, shampooing of the lashes with bland soap and manual manipulation with cotton or



Blepharitis comb

gauze, a great number of scales and fine scabs are still found firmly attached to the skin of the borders of the lids and enmeshed between the base of the cilia.

The comb¹ shown in the accompanying illustration consists of eight metallic needles, 3 mm long, covering a width of 3 mm. With this instrument the branlike scales of blepharitis squamosa are removed from the eyelashes in one treatment. In cases of stubborn blepharitis ulcerosa if the scabs are first made oily by the use of petrolatum or boric acid ointment it is possible to remove them without bleeding or discomfort. The edge of the skin of the lids is then accessible to medication with preparations such as iodine, silver nitrate or brilliant green.

The points of the needles are not long enough to injure the cornea, but in the interest of absolute safety the following technic is employed. The patient's gaze is directed inferiorly, the lid is raised off the globe and the instrument is held at a slightly oblique angle. The instrument can be sterilized, and debris collecting at the base of the needles can be removed with ordinary sewing thread.

Presented at the Eye Staff Conference, Hospital for Joint Diseases, Feb 24, 1938.

1 The comb is manufactured by E. B. Meyrowitz, Inc.

Abstracts from Current Literature

EDITED BY DR WILLIAM ZENTMAYER

Bacteriology and Serology

ATTEMPTS TO DEVELOP IN VITRO OCULOTROPISM OF THE AVIRULENT ANTHRAX BACILLUS AND OF *PROTEUS VULGARIS* V SPADAVECCHIA, Ann di ottal e clin ocul 65: 641 (Sept) 1937

After growing *Bacillus anthracis* and *Proteus vulgaris* in mediums containing lenticular, retinal and uveal beef tissue, the author studied changes in their power to invade the eyes of animals. Previous experiments concerning the growth of various organisms in the ocular mediums are reviewed. The author found that an avirulent strain of *B. anthracis* grew well in broth containing aqueous, vitreous, crystalline lens and a mixture of retina and choroid as well as in pure vitreous and aqueous. Organisms grown in such mediums remained avirulent when injected subcutaneously into guinea pigs. Six day cultures in such mediums were injected into the inoculated tissues were made of a series of rabbits, and cultures of the presence of living organisms six days after inoculation to determine the presence of living organisms. The signs of inflammation produced were limited to slight reaction at the site of inoculation, which disappeared after two or three days. Cultures of the tissues after six days were negative except for 1 animal inoculated in the choroid. For another animal smears showed organisms in the vitreous, but cultures were negative.

Proteus vulgaris was grown in a suspension of uveal and retinal tissue. After six days intravenous injections of the material were given to rabbits, and after another six days some of the animals were given an additional injection of the same material. There was no evidence of ocular inflammation, and cultures of the ocular tissues were negative. Thus there seems to be no evidence that oculotropism can be developed under these conditions, at least for the two organisms studied.

S R GIFFORD

Biochemistry

THE RELATION OF AGE AND PHOSPHORIC ACID ESTERS CONTENT OF THE LENS H K MULLER, Arch f Augenh 110:128, 1936

With the methods of hydrolysis devised by Lohman (*Biochem. Ztsch* 194. 306, 1928), Muller showed that lenses of animals of different age groups contained varying amounts of phosphoric acid esters. The phosphoric acid esters which are difficult to hydrolyze disappear from the lenses of older animals, while those esters which are easily hydrolyzable are still present. It is possible that vitamin C

is formed from these phosphoric acid esters which are hydrolyzed with difficulty, and this may be of importance in maintaining the transparency of the lens

F H ADLER

Conjunctiva

CHRONIC CONJUNCTIVITIS IS PROVOKED BY UNDETECTED MYCELIAN CONCRETIONS IN THE LACRIMAL CANALICULUS VAILERE-VIALEIX, Bull Soc d'opht de Paris, May 1937, p 298

In a previous report in the *Annales d'oculistique* of December 1933 (page 1030), the author reported a case of this condition Two additional cases are included in the present paper The outstanding symptoms are (1) the localization of the inflammation and secretion at the internal angle of the eye, (2) an intense sensation of continuous itching at the inner canthus and (3) entire absence of all local signs in the region of the canaliculus with no interference with the drainage of the tears

Additional factors of importance are the hyperemia and infiltration of the conjunctiva with development of follicles and granulations under the conjunctiva of the upper lid Ordinary therapeutic measures for acute conjunctivitis cause no change in the condition, and only after the concretions are removed from the canaliculi is there a cessation of symptoms

L L MAYER

SPOROTHRIX GRANULOMA OF THE LIMBUS G CAVALLACCI, Arch di ottal 44: 247 (July-Aug) 1937

A 25 year old farmer presented himself at the clinic, stating that two months previously he had been struck in the left eye by a willow branch He experienced only slight discomfort at the time, but later recalled rubbing his eye repeatedly with his fingers

Fifteen days later he noted a small gray spot at the limbus In a short time the area grew larger, and the surrounding conjunctiva became quite red For a few days before coming to the clinic he had experienced a moderate foreign body sensation, lacrimation and photophobia

The results of the physical and laboratory examinations were negative Ocular examination revealed vision of 10 in each eye In the left eye at the limbus between 4 and 5 o'clock was a rounded granuloma the size of a millet seed The mass was firmly fixed to the underlying tissue, and its surface was irregularly eroded, with slight central dimpling The conjunctiva surrounding it was slightly red, with a few new vessels Adjacent to the lesion was moderate corneal infiltration

Cultures taken after careful cleansing of the area were positive, and *Sporotrichum beurmanni* was isolated The virulence of the organism was proved by injection into the rat, mouse and rabbit

Treatment was carried out in the following manner Potassium iodide was given by mouth in increasingly larger doses up to a total of 3 Gm per day This was poorly tolerated, so that intramuscular injections of an iodine preparation were substituted A 1 per cent solution of potassium iodide was used locally After one month no sign of the original lesion could be found

F P GUIDA

THE USE OF STRONG SOLUTIONS OF ZINC SULFATE IN MORAX-AXENFELD CONJUNCTIVITIS L D MITZKEWITZ, Vestnik oftal 11: 554, 1937

Mitzkewitz used from a 3 to 5 per cent solution of zinc sulfate in the treatment of chronic and fresh angular conjunctivitis caused by the diplobacillus of Morax and Axenfeld. From four to six swabbings of the conjunctiva, without irrigation of the conjunctival sac, were sufficient to cause the subjective symptoms, the redness of the angle and maceration of the skin of the lids to abate. Instillation of 1 drop of cocaine hydrochloride prevented the burning sensation caused by the zinc sulfate. This treatment was also effective in cases of trachoma.

The bacterioscopic examination showed no parallelism between the clinical picture of the disease and the number of the bacilli present, so that zinc sulfate evidently possesses a neutralizing property toward the toxins of the micro-organism.

O SITCHEVSKA

Comparative Ophthalmology

THE VISUAL CELLS OF THE PLATYPUS (ORNITHORHYNCHUS) K O'DAY, Brit J Ophth 22: 321 (June) 1938

The author finds a striking difference in the structure of the visual cells of the monotreme *Platypus* (*Ornithorhynchus*) from that of the visual cells of the marsupial and higher mammals, and a close resemblance to that of the visual cells of the sauropsidea. In addition to single cones with oil droplets, double cones are present, the chief member only containing the oil droplet. The rods are surprisingly massive and do not seem to outnumber the cones.

The comparative sparsity of the rods is remarkable in view of the commonly held opinion that the animal is markedly nocturnal in its habits. However, it hunts and feeds freely in brilliant sunlight.

The finding of double cones in the retina of the monotreme and of twin cones in that of the marsupial indicates that the marsupial and higher mammals are more nearly related to each other than to the monotreme.

The article is illustrated

W ZENTMAYER

Cornea and Sclera

THREE CASES OF ANTERIOR SCLEROUVEITIS, TWO (ONE BILATERAL) OF THE MALIGNANT TYPE (PROGRESSIVE SCLEROPERIKERATITIS OF VON SZILY) CLINICAL, HISTOPATHOLOGIC AND BACTERIOLOGIC STUDY OF THREE ENUCLEATED EYES G BOSSALINO, Arch di ottal 44: 313 (Nov) 1937

Three cases of progressive scleroperikeratitis of von Szily are described by Bossalino. In 1 of these he was able to produce a cure, but in the other 2 cases, in 1 of which the condition was unilateral and in the other of which it was bilateral, the disease was so severe as to necessitate enucleation. Clinical and histologic investigation lead Bossalino to believe that the disease has its beginning in the sclera and is of tuberculous origin. Some evidence of tuberculosis was found in all 3

patients, 1 had a strongly positive Pirquet reaction, 1 healed apical lesions and a third calcified hilar nodes. Inoculation of an animal with material from one eye produced typical tuberculous lesions, and acid-fast bacilli were grown that were undoubtedly tubercle bacilli.

F P GUIDA

INFECTION OF THE CORNEA IN IRRITATION OF THE PERIPHERAL NERVES

P MIKAEIAN and B ZARCHI, *Vestnik oftal* 11:611, 1937

A review of the literature on the subject is given, particularly of the work done by Metalnikoff at the Pasteur Institute in Paris. Mikaelian and Zarchi based their experimental work on Speransky's theory of the relation between the trophicity of the nerves and pathologic processes.

Strains of *Bacillus pyocyaneus*, *Bacterium coli* and *Staphylococcus haemolyticus* were tried in a dilution of one loop to 1, 4 and 10 cc of physiologic solution of sodium chloride. *Staph. haemolyticus* in the last dilution was found to be the most suitable, as it produced a prolonged experimental keratitis of medium severity.

Two series of experiments were conducted with 8 pairs of rabbits in each series. The rabbits were anesthetized and either the second branch of the trigeminal nerve or the sciatic nerve was exposed, and a drop of castor oil was injected into the nerve, which was severed below the site of the injection. In some rabbits the nerve was crushed between the prongs of a forceps instead of injecting the castor oil. The two series of the experiment differed from each other in that in one the infection of the cornea with 0.05 cc of culture of the *staphylococcus* was produced ten days after the traumatization of the nerve, while in the second series the infection of the cornea and the irritation of the nerve were done simultaneously. In those rabbits in which the nerves were irritated ten days previously, the course of the infection of the cornea was milder than in the control group, while in the second series the reverse was observed. The results of this experiment indicate that the factor of time plays an important role in the relation between the irritation of the peripheral nerves and the infection of the cornea. Four tables illustrate the article.

O SITCHEVSKA

Experimental Pathology

EXPERIMENTS ON PAPILLARY STASIS R CAMPOS, *Arch di ottal* 44 211 (July-Aug), 267 (Sept-Oct) 1937

Campos ligated the optic nerve in 16 rabbits and 2 monkeys at various levels, i. e., from just behind the sclera, including the central vessels, to a point some distance behind the entrance of the vessels. In a few cases the effect of opening the sheath of the optic nerve distal to the ligature was observed. Campos makes the following observations:

1 When the ligature contains the central vessels, venous stasis makes its appearance immediately, followed shortly by lymphatic stasis.

2 Ligation just central to the entrance of the vessels into the nerve causes marked papillary lymphatic stasis, with only slight venous stasis

3 The more central the ligature, the less severe the edema, it may even be so slight as not to form true papillary stasis

4 Opening the sheath distal to the ligature has no effect on the appearance of papillary stasis

Microscopic examination of the nerves and eyes revealed the following changes

1 There was a rapidly forming interstitial neuritis near the ligature, intense edema of the nerve trunk, extending more peripherally than centrally with respect to the ligature, and dilatation of the distal portion of the nerve

2 The papillary stasis was not accompanied by an inflammatory phenomenon if the nerve had not been too severely traumatized

3 The spaces of the sheath were extensively obliterated distal to the ligature and dilated centrally

Campos' conclusions are as follows

1 It is probable that in the nerve tract, central to the entrance of the retinal vessels, there is a lymphatic current directed centrally on the inside and peripherally in the sheath

2 In the more distal portion of the nerve containing the central vessels and in the papilla there is a lymphatic current which is directed to the exterior following the lymphatic spaces around the vessels

3 Papillary stasis is probably secondary to compression of the central vessels, at their exit from the nerve, by the edema produced by the ligature. The evidence is inconclusive as to whether the interference is with the venous flow or with the lymphatic flow. There is some evidence to support the latter assumption

F P GUIDA

General

THE TOOTH AND THE EYE D A FRIEDMAN, Harefuah 13:77
(Nov) 1937

In this paper the author reviews briefly the history of focal infection with special reference to the influence of dental conditions on the eye and orbit. He presents 3 cases as classic examples, 1 of which is of rather unusual occurrence. It was that of an infant 2 weeks old who exhibited marked protrusion and lateral displacement of the right eye caused by subperiosteal abscess in the orbit. Other symptoms were edema of the lid and conjunctiva and a suppurative discharge from the nose and from an opening near the caruncle, which was much enlarged. Pressure over the superior maxillary region and on the roof of the nose brought forth pus from the upper jaw which escaped through the baby's mouth.

The author believes that this widespread infection originated from the embryonic teeth of the child which became infected from an abscess on the mother's nipple. This infection, according to his theory, traveled up the superior maxillary bone entered the ethmoid sinus

and caused osteomyelitis in the orbit and superior maxilla. After the sequester from the alveolar processes of the superior maxilla was removed and ample drainage was established, the baby quickly recovered.

The other 2 examples were cases of hemorrhagic neuroretinitis, the condition in 1 case being due to remaining roots of two superior molars and in the other to pyorrhea of the alveolar processes. Recovery occurred in both cases as soon as the causes were removed. The author is of the opinion that any dental disease or infection may affect the eye and orbit either by the contiguity of tissues or by the way of the blood and lymph channels.

Dr. Friedman believes that dental diseases responsible for ocular trouble are, in the order of their frequency, (1) abscess, (2) granuloma, (3) pyorrhea, (4) caries and (5) periodontitis, those due to diseased teeth, in the order of frequency, are (1) papillitis, (2) uveitis, (3) iridocyclitis, (4) opacities of the vitreous, (5) keratitis, (6) palpebral and blepharal edema, (7) conjunctivitis, (8) exophthalmos, (9) cellulitis of the orbit and (10) meningitis.

B. L. GORDON

General Diseases

RETROBULBAR NEURITIS IN PELLAGRA M. FINE and G. S. LACHMAN,
Am J Ophth 20:708 (July) 1937

Fine and Lachman report retrobulbar neuritis in 3 cases of pellagra in which the cutaneous lesions led to the correct diagnosis. None of the patients exhibited the cecentral scotomas said to be characteristic of tobacco-alcohol amblyopia. The authors speculate as to the relation of the pellagrous syndrome to the visual disturbances and the effect of alcohol. According to them, "It does not seem improbable that a relationship such as exists between vitamin-B₁ deficiency and the peripheral nervous system may also exist between vitamin-B₂ (G) deficiency and the central nervous system, of which the optic nerve is a part, and that in each case the alcoholism plays only an indirect role. Such a quantitative relationship would offer an explanation of the fact that some severe alcoholics never suffer from amblyopia, while other relatively moderate drinkers suffer serious insult to the visual fibers.

"One wonders, also, whether many cases of 'alcohol and tobacco' amblyopia are not complicated by a deficiency of vitamin G."

W. S. REESE

Glaucoma

A NOTE ON DIASTOLIC PRESSURE AND GLAUCOMA I. HARTSHORNE,
Am J Ophth 20:724 (July) 1937

Hartshorne calls attention to the fact that in cases of glaucoma in which the diastolic blood pressure is low a subnormal retinal blood supply may result, followed by atrophy of the optic nerve, as shown by concentric contraction of the field of vision. In these cases an effort should be made to increase the diastolic pressure and to decrease the

ocular tension, as the glaucoma is not dominating and not the cause of this type of contraction of the field of vision. The author cites 2 cases in which operation was avoided by treatment along such lines

W S REESE

JUVENILE GLAUCOMA WITH ANGIOMATOSIS OF THE RETINA L
MARUCCI, *Arch di ottal* 44:163 (May-June) 1937

Both eyes of a 30 year old woman with bilateral glaucoma showed a vascular anomaly which Marucci calls angiomatosis of the papillary region. The malformation was probably an atypical division of the central retinal artery with a disorganized arrangement of its branches. It seemed possible to Marucci that the glaucoma could have been due to a hypersecretion of aqueous and a congestion associated with a similar vascular anomaly in the ciliary body

F P GUIDA

Injuries

DIMETHYL-SULPHATE POISONING IN RELATION TO OPHTHALMOLOGY
S DE GROSZ, *Am J Ophth* 20:700 (July) 1937

De Grosz describes dimethylsulfate and its toxicology, particularly as to its effect on the eye. It is an extremely strong poison and was used in the World War together with chlosulfonic acid as a tear gas, though it is really irritating and corrosive, experiments showing that in the tissues it slowly hydrolyzes into sulfuric acid and methyl alcohol. De Grosz presents the following summary:

"The chemical and pharmacologic properties of dimethyl sulphate have been discussed and its toxic action on the experimental animal and on man described. Four cases are presented with analysis of symptoms and methods of treatment."

W S REESE

Instruments

THE SOUTER TONOMETER F H VERHOEFF, *Am J Ophth* 20:720
(July) 1937

Verhoeff recommends the Souter tonometer and describes his method of using it. He regards it as much more accurate and less dangerous than the Schiotz tonometer, its one disadvantage being the difficulty of determining the amount of indentation of the cornea at which the instrument is to be read.

W S REESE

A MODIFIED BRIGGS' RETRACTOR FOR DACRYOCYSTORHINOSTOMY H B
STALLARD, *Brit J Ophth* 22:361 (June) 1938

The modification of Briggs's retractor reported in this article consists of a broad blade-like termination of the shank on the temporal side. This shank is armed with three blunt, curved claws, and in addition there is a slot for the purpose of inserting a curved guard. The article is illustrated.

W ZENTMAYER

Lens

CALCIUM CONTENT OF THE CRYSTALLINE LENS IN EXPERIMENTAL
PARATHYROPRIVAL CATARACT S RINALDI, *Ann di ottal e clin
ocul* 65: 667 (Sept) 1937

The literature on cataract in parathyroidectomized animals and on the inorganic constituents of the normal and cataractous lens is reviewed. Fourteen rabbits were submitted to parathyroidectomy. Definite cortical opacities developed in the lenses of 6 animals, but these remained limited to the subcapsular cortex. In a seventh animal complete cataracts developed. One eye of each animal was removed before operation, and the second eye was removed from five to sixty-five days after operation. Eight normal beef lenses, 8 human lenses with cortical cataracts (senile), 1 human lens with sclerotic cataract and 1 human lens with cataracta brunescens were studied for comparison. The normal beef lenses showed an average calcium content of 0.085 mg per hundred cubic centimeters. In 3 animals which died with tetanic seizures before opacities of the lens developed, calcium was reduced in the second eye to approximately half the amount found in the eye removed before operation. The amounts were 0.20 mg per hundred cubic centimeters in the first eye and 0.10 mg in the second eye of 2 of these animals. In those in which cataract developed, however, the amount of calcium in the second eye was increased, often to twice the amount present in the first eye (0.15 mg per hundred cubic centimeters in the first eye and 0.36 mg in the second eye of 1 animal). In the animal with total cataract the amount in the cataractous lens was 2.12 mg. This last figure compares somewhat closely with the findings for human cataract, in which 2.94 mg was found in the cortical type, 3.03 mg in the sclerotic type and 2.92 mg in the cataracta brunescens. Since the amount of calcium in the aqueous and vitreous of parathyroidectomized animals has been found to be below normal, the author believes that his findings point to an increased permeability of the capsule of the lens in such animals. An increase in the weight of the lenses showing an increased calcium content is also in favor of increased permeability of the capsule, resulting in increased inhibition of water. Other evidence indicates that the p_H of the blood and aqueous increases in such animals, which may also be a factor in the chemical changes within the lens and the resulting precipitation of the lens proteins.

S R GIFFORD

PARATHYROID GLANDS AND THE EYE G LoCASCIO, *Ann di ottal e clin
ocul* 65: 801 (Nov) 1937

This paper is a resumé of the author's report at the Fifteenth Ophthalmological Congress. The author reviews the clinical picture of cataract in cases of parathyroprival tetany. Aside from the cases seen after thyroidectomy, of which some 70 have been reported, a number of cases of stratified opacities of the lens developing in infancy in association with tetany and low blood calcium and the cases developing in young mothers are considered as due to parathyroid deficiency. A few cases seen in adults not associated with any of the foregoing conditions, but with various symptoms of tetany, are probably of similar nature. A

study of experimental parathyroidoprival tetany in rabbits made at the University of Naples by a group of observers brought out the following facts

1 After parathyroidectomy opacities of the lens developed in 60 per cent of animals

2 There was no definite relation between the appearance of the opacities and tetany. While in many animals tetany preceded the opacities of the lens, in others this order was reversed, while in still others opacities of the lens developed without any signs of tetany being observed

3 The first opacities were seen from ten days to four weeks after operation. In only 3 animals did the lenses become completely opaque

4 Chemical study of the blood and aqueous of the animals showed (a) a slight but definite increase in the p_H of serum, aqueous and lens, (b) no changes in the chlorides or electrical conductivity, (c) no significant change in the ascorbic acid of the aqueous, (d) no change in the respiration (oxygen consumption) of the lens in partial cataract but a marked reduction in complete cataract, (e) no definite changes in ascorbic acid or glutathione in the lens, (f) constant reduction of the glycolysis and entire absence of glycolysis in complete cataract, (g) an increase of the amino nitrogen of the lens, (h) a constant increase in the calcium in the lens, the increase amounting to fourteen times the normal in complete cataract, (i) slight displacement of the isoelectric point of the lens to the acid side, (j) an increase of the water content of the lens, (k) no change in the inorganic phosphorus of the vitreous, and (l) changes in the capsular epithelium similar to those seen in other types of experimental cataract and in senile cataract

The author believes that the more alkaline reaction of aqueous and lens, by increasing inhibition of water, the decrease in calcium in the aqueous, by making the lens capsule more permeable, and the increase in calcium in the lens, by favoring precipitation of the lens proteins, are the most important chemical changes responsible for the opacities of the lens

S R GIFFORD

Methods of Examination

THE OPHTHALMODYNAMOMETER AND ITS DIAGNOSTIC VALUE E
HORNIKER, *Ann di ottal e clin ocul* 65:832 (Nov) 1937

The author employed the ophthalmodynamometers of Bailliant and of Sobański. He found the diastolic pressure of the retinal arteries in a series of normal persons to be from 30 to 35 mm of mercury and to be half that of the diastolic brachial pressure

In a series of 200 patients at a psychiatric hospital, he found an increase in retinal blood pressure with normal systemic pressure to be frequent in those with post-traumatic epilepsy and cephalalgia and also in postencephalitic patients. In a series of patients with tumor of the brain the correlation between retinal blood pressure and cerebrospinal pressure as determined at lumbar puncture was by no means constant, although some parallelism was observed. The relation between pressure in the central vein and intracranial pressure would seem to be more constant

S R GIFFORD

Ocular Muscles

ANATOMIC AND CLINICAL STUDY OF THE INSERTION OF THE OBLIQUE MUSCLES M SMALTINO, *Ann di ottal e clin ocul* 65 585 (Aug) 1937

The author made exact measurements on 13 eyes operated on for retinal detachment and on the eyes of 15 cadavers. The anterior extremity of the insertion of the superior oblique muscle was usually from 15 to 17 mm from the limbus. It was usually situated on a meridian making an angle of from 14 to 20 degrees with the meridian bisecting the superior rectus muscle. The anterior extremity of the insertion of the inferior oblique muscle was in most cases from 15 to 18 mm from the limbus and was located along a meridian making an angle of from 14 to 15 degrees with a line bisecting the external rectus muscle. For the frequency of various slight variations from these locations, the original article must be consulted.

S R GIFFORD

Operations

PLASTIC OPERATIONS ON THE LOWER LID, WITH ESPECIAL CONSIDERATION TO THE PROBLEM OF CARCINOMA T SCHORNSTEIN, *Arch f Augenh* 110: 549, 1937

The literature on the treatment of carcinoma of the lower lid reveals two main points of view. One group believes that operation is indicated because of the recurrences after intensive radiation and the failure in many cases of roentgen rays or radium to stop the spread of the growth. This group also points out the dangers attendant on roentgen treatment of cataract and destructive nodocyclitis. Those who favor radiation therapy point to the marked disfiguration caused by surgical removal of these growths.

Schornstein believes that surgical removal is on the whole the most satisfactory and sets forth in this paper his methods whereby good cosmetic results can be secured in the majority of cases by carefully planned plastic operations. In brief, these are as follows:

- 1 The tumor is removed by a wedge-shaped incision, the base corresponding to the site of the tumor on the lower lid and the apex extending down onto the cheek.

- 2 The conjunctiva is freed from the margin of the lid from a point at the middle of the upper lid, extending out to the temporal canthus and down to within 2 mm of the temporal side of the growth on the lower lid.

- 3 A curved incision is now made from the outer canthus up to the level of the brow and outward over the temple, curving down in front of the ear to about the level of the tragus.

- 4 The wedge-shaped piece of skin, including the tumor, is removed, and the whole temporal flap is swung down into position, where it can be stitched into place, filling the defect left by the removal of the wedge-shaped piece. By undermining above, the temporal defect can be closed.

Pictures of 3 patients so treated are presented.

F H ADLER

Orbit, Eyeball and Accessory Sinuses

SMOOTH MUSCLE OF THE PERIORBITA AND THE MECHANISM OF EXOPHTHALMOS C E BRUNTON, Brit J Ophth 22:257 (May) 1938

Brunton reviews the work of anatomists in reference to "Muller's orbital muscle" and gives a translation in full of Muller's article published in 1858. The following summary is presented:

"(1) The whole orbital region was removed in one piece from the heads of cats and dogs. After fixation, decalcification and celloidin embedding, sections were stained by different methods.

"(2) Sections at various planes show how smooth muscle and elastic tissue joint with collagenous fibres to form the periorbital membrane known as Muller's orbital membrane or muscle. This is a funnel-shaped structure, having its apex round the optic foramen and attached in front to the orbital margin. In planes behind the eyeball it contains much smooth muscle. Its contraction increases pressure behind the globe and forces the globe forward. Its relations to the investing fascia of the extrinsic muscles of the eye and to the secreting glands of the orbital region are considered.

"(3) Muller's orbital muscle in the lower animals is the final mechanism in them for proptosis. Muller's orbital muscle in man cannot by itself produce proptosis.

"(4) Photomicrographs show smooth muscle behind the eyeball in the upper and outer periorbita of human subjects, whether normal or suffering from Graves' disease. This muscle may be the functional analogue of the periorbital membrane of the lower animals and be a mechanism for the production of exophthalmos in man.

"(5) The nomenclature of the smooth muscle in different situations is discussed."

Brunton believes that if Muller's alternative name of "orbital membrane" were reserved for the structure in the lower animals and the name "orbital muscle" for the muscle in the region of the inferior orbital fissure in man, a distinction in the function of the two would be suggested.

Numerous illustrations accompany the article.

W ZENTMAYER.

The Pupil

TONIC PUPIL AND ITS RELATIONSHIP TO ADIE'S SYNDROME T ALAJOUANINE and PIERRE-V MORAX, Ann d'ocul 175:205 (March) 1938

Since the publication by Adie in 1931 and 1932 on the syndrome which now bears his name and which associates certain pupillary disturbances with tendinous areflexia, numerous reports of a similar nature have appeared. The authors consider many of the reported disturbances atypical and not to be included in this group.

Adie's syndrome, when complete, is recognized by (1) certain pupillary disturbances, usually unilateral, consisting of, to use the authors' expression, the tonic pupil, the features of which are sum-

marized as apparent absence of photomotor reflex and a special slowness of the synkinetic contraction in convergence and also of the dilatation which follows, (2) absence of some or even all tendinous reflexes of the extremities, (3) various sympathetic disturbances, and (4) an unknown etiology

A historical review is given, showing that before Adie's description in 1932, the tonic pupil was already well known, being first described by Piltz in 1900. Then follows a clinical study of the condition based on 8 personal cases and 92 reported in the literature. The study is divided into (1) onset, and (2) details of the syndrome. Seventy-five per cent of the cases occurred in females, and 85 per cent of the patients were young. The different types of pupillary disturbances, the ocular disturbances outside the iris and the neurologic and general symptoms are described. The article is to be continued.

S H MCKEE

Physiologic Optics

OPTICAL DECENTRATION OF THE EYE. A I DASHEVSKIY and D G BUSHMICH, *Am J Ophth* 21 125 (Feb) 1938

This article does not lend itself to abstracting. The authors present the following conclusions:

"1 The angle kappa does not lie precisely in the horizontal plane

"2 The angle of the plane's incline of the angle kappa to the horizontal does not exceed 25 degrees

"3 The measurement of the angle kappa by our method of a double localization of the light reflex appears to be the most precise of all proposed methods

"4 In most cases the visual line passes through the cornea inwards and upwards from the corneal center

"5 The slight degree of error in our method permits its application for clinical practice and theoretical use and appears to be the smallest as regards the other methods"

W S REESE

CONSTITUTION OF THE REFRACTED LIGHT BEAM IN THE ACCOMMODATED EYE. P VITO, *Ann di ottal e clin ocul* 65. 525 (July) 1937

This article is difficult to abstract. The author believes that displacement of the pupil to the nasal side during accommodation aids in neutralizing the usual horizontal asymmetry of the eye, bringing about increased symmetry of the resulting image.

R GIFFORD

Physiology

BEHAVIOR OF THE INTRAOCULAR PRESSURE IN ACUTE INTRAOCULAR VASCULAR REACTIONS. F POOS, *Arch f Augenh* 110 499, 1937

Subconjunctival injections of various drugs were given to a series of rabbits, and the intraocular pressure was measured by a tonometer over a period of twelve hours. When sympathomimetic drugs were injected such as epinephrine hydrochloride, there was a slight transitory fall in the intraocular pressure, which was soon followed by a rapid

and more marked rise. After this, during the twelve hours there occurred a series of rises and falls in pressure of diminishing extent, until the pressure gradually returned to the normal level.

Subconjunctival injections of parasympathetic drugs produced the same picture, with the exception that in place of the preliminary fall in pressure there occurred a slight rise. Injections of ethylmorphine hydrochloride or salt solution (2.5 per cent) gave the same results.

Poos draws the conclusion that the effect on the intraocular blood vessels of all of the drugs is not a pharmacologic but a toxic one and that the vessels react the same way to all toxins regardless of their specific pharmacologic effect. The result is the same type of pressure change in the eye.

The rhythmic changes in pressure are due to a direct vasodilator action on the arterioles and capillaries. This results in an outpouring of a protein-rich aqueous and an increase in the intraocular pressure. The dilatation of the veins causes a marked venous stasis, which in itself effects a further rise in the hydrostatic blood pressure within the eye and thereby increases the already elevated intraocular pressure. By this time, however, the change in the composition of the blood produces a local reactive effect on the capillaries, as a result of which they start contracting. This leads to a fall in pressure, and if the effect of this counterbalancing action were of constant duration and of equal intensity, the intraocular pressure would return gradually to normal. This does not occur, however, according to the author, and the result is a series of more or less rhythmic rises and falls, as one activity overcomes the other.

By artificially lowering the hydrostatic blood pressure before giving the subconjunctival injections, or by removing the influence of the vasoconstrictor fibers to the capillaries by extirpation of the superior cervical ganglion, Poos was able to avoid these rhythmic changes in pressure and thus to prove their dependence on the vascular system within the eye.

F. H. ADLER

Refraction and Accommodation

VISUAL DEFECTS IN 11,021 SCHOOL CHILDREN OF MILAN. M. PAGANI, *Ann di ottal e clin ocul* 65: 812 (Nov.) 1937.

Of a large number of school children examined, 12.5 per cent had visual defects. Hyperopia was found in 3,605, astigmatism in 2,868, myopia in 2,451, anisometropia in 803 and amblyopia in 659. Strabismus was seen in 909, which is approximately 0.10 per cent of the student body during the period of twelve years, or 8 per cent of the number with visual defects.

S. R. GIFFORD

FAMILIAL HYPERMETROPIA OF HIGH DEGREE. CONSIDERATION OF THE PATHOGENESIS OF AMETROPIA. G. CAVALLACCI, *Arch di ottal* 44: 178 (May-June) 1937.

Three siblings of consanguineous parents showed a high degree of hypermetropia. After careful consideration of his own cases and those described in the literature, Cavallacci concludes that an eye showing a

high degree of hypermetropia does not present a true anomaly but manifests, in exaggerated form, the characteristics of the eye with the usual degrees of hypermetropia. This makes the problem of etiology one and the same.

Examination of the cranial measurements in the author's cases and in the cases of usual axial ametropia revealed a frequent association between brachycephaly and hypermetropia and between dolichocephaly and myopia. Cavallacci feels that this is an expression of an embryonal condition common to both the head and the eye, i.e., the cephalic anlage possesses morphologic characteristics that are congenitally fixed and in later development are transmitted either to the head itself or to the optic vesicle.

These congenital morphologic factors are probably hereditary in accordance with the frequent hereditary and familial character of the various ametropias. In conclusion, Cavallacci expresses the opinion that the aspect of consanguinity strengthened the hereditary factor of hypermetropia which was present in the family, resulting in the exaggerated expression of hypermetropia observed in his cases.

Included in the article is a statistical table showing the refractive errors found at the Royal Eye Clinic of Pisa from 1916 to 1936 and demonstrating the rarity of the condition described in addition to the frequency of other ametropias.

F. P. GUIDA

Retina and Optic Nerve

THE CHIASMAL SYNDROME AND RETROBULBAR NEURITIS IN PREGNANCY. A. HAGEDOORN, *Am J Ophth* 20:690 (July) 1937

Hagedoorn reports the case of a pregnant woman in whom retrobulbar neuritis developed, which improved rapidly after delivery. She again became pregnant, and visual disturbances developed, a diagnosis of suprasellar tumor was made. A large suprasellar meningioma was found, but the patient died a few hours after operation. Hagedoorn concludes:

"In pregnant women, perfectly healthy in other respects, may occur a gradually progressive failure of vision and temporal hemianopsia, usually affecting only one side in the beginning, but becoming binocular in the course of the pregnancy. (In my case the first evident symptom was a unilateral central scotoma.) At that time, generally, the seventh month of pregnancy has been reached. Optic pallor or atrophy may follow. The condition generally returns earlier and more seriously in a following pregnancy and may lead to invalidity.

"Nearly always the symptoms disappeared more or less completely after delivery, amelioration beginning even in the first days after delivery.

"If the sella is normal this clinical picture may be caused by the affections listed by Cushing, of which suprasellar meningioma is the most frequent and important, the symptoms of which become manifest only by the physiological hypertrophy of the hypophysis during pregnancy.

"In addition to the possibilities stated by Cushing, the possibility of retrobulbar neuritis in pregnant women must be considered, which

disease is stated to occur in pregnancy In the future, in cases of (retrobulbar) neuritis in a healthy pregnant woman special attention should be given to the visual fields

"In cases with bitemporal hemianopsia, if vision in the better eye diminishes to $1/3$ to $1/2$, pregnancy should be terminated If abortion is refused, one may wait till vision in the better eye is $1/10$ If a living child is not possible at that time I believe treatment by the neurosurgeon is indicated

"A following pregnancy should be avoided" W S REESE

JUVENILE FORM OF FAMILIAL AMAUROTIC IDIOCY V PATERNOSTRO,
Ann di ottal e clin ocul 65: 561 (Aug) 1937

Only 9 cases of familial amaurotic idiocy were found reported in the Italian literature The author reports a tenth case, with a review of the literature His patient was a boy of 7, of Italian parents The family history was unimportant, although the healthy sister was only 4 years of age The child developed normally, both mentally and physically to the age of 5 years, when visual disturbances were noted, especially in bright daylight, vision was better in twilight About a year later convulsions began, accompanied by signs of mental deterioration By this time vision was reduced to perception of light When the child was first seen by the author she was unable to walk or to speak except for the repetition of words Both nerves were white and their arteries threadlike, and some branches were apparently occluded Light punctate deposits were present all over both fundi, including the macular areas No perception of light was present in either eye The history of visual loss affecting first the central and later the peripheral portions of the fields, as indicated by better vision at night in the early stage of the disease, and the fundus picture of "retinitis pigmentosa without pigment" were considered by Stargardt as typical of the juvenile form of amaurotic idiocy as distinct from the picture seen in the infantile form

S R GIFFORD

MULTIPLE CIRCUMSCRIBED RETINAL DETACHMENT J L PAVIA and
R A TARTARI, Arch de oftal de Buenos Aires 12: 436 (July)
1937

A case of multiple circumscribed retinal detachment following traumatism is reported In the temporal portion of the retina there was a parapapillary cyst with a diameter of 3 papillary diameters and a height of 8 diameters, a second cyst, of 7 papillary diameters and 3 papillary diameters from the other cyst, extended to the ora serrata In the nasal half of the retina there were signs of an old rupture and loss of transparency of the lower half of the retina

C E FINLAY

CALCIUM DEPOSITS IN THE NEIGHBORHOOD OF THE CHIASM IN DISTURBANCES OF THE OPTIC NERVE M GLEES, Arch f Augenh
110: 120, 1936

The author found deposits of calcium in the neighborhood of the chiasm in 27 of 40 patients over 50 years of age Roentgenographic

examination showed calcification of the carotid artery in 5 of the 27 patients. Only 1 of these patients presented the typical symptom complex of pseudoglaucoma, whereas one fifth of them with true glaucoma showed similar changes.

It seems doubtful whether the deposits of calcium in the carotid artery have any direct bearing on the causation of the diseases of the optic nerve. So far no direct proof of this has been offered, and Glees' findings do not support such a contention.

F H ADLER

PATHOLOGIC ANATOMY OF DISCIFORM DEGENERATION OF THE MACULA R BRAUN, Arch f Augenh 110: 534, 1937

The eye of a 67 year old man was enucleated because of a mass between the disk and the macula which was suspected of being a melanotic sarcoma. The mass was elevated about 5 D, but there was no detachment of the retina. There were no hemorrhages, and the mass was dark in color. Physical examination of the patient revealed nothing abnormal for a man of his age. The following year the patient returned with a beginning senile macular degeneration in the opposite eye (Braun's description of the ophthalmoscopic appearance of the eye which was enucleated and of the fellow eye with the macular degeneration is certainly not typical of the cases in the literature reported as the Junius and Kuhnt type of disciform macular degeneration).

Braun diagnosed the condition in the enucleated eye as a disciform macular degeneration. Pathologic examination of this eye showed no trace of neoplasm, but a large retroretinal hemorrhage and a diffuse growth of connective tissue. The latter lay partly between the retina and the pigment epithelium and partly between pigment epithelium and the choroid. The elastic lamina showed a circumscribed perforation through which the connective tissue seemed to come from the choriocapillaris.

From this the author concludes that disciform degeneration can arise from the choroid.

F H ADLER

TEMPORARY ARTIFICIAL INDENTATION OF THE SCLERA IN OPERATIONS FOR DETACHMENT OF THE RETINA A JESS, Klin Monatsbl f Augenh 99: 318 (Sept) 1937

Applanation of the retina is induced by puncture prior to electrocoagulation for detachment of the retina, or following it, in cases in which an insufficient quantity of subretinal fluid escapes. Collapse of the eyeball may render attachment of the retina onto the choroid more difficult under these circumstances. Jess obtained favorable results by artificial indentation of the sclera over the area of coagulation by a tampon of adequate size introduced into Tenon's space. The conjunctiva is closed by sutures over the tampon, which is attached to a thread and left in place up to two weeks. It is removed after anesthetization of the conjunctiva and retrobulbar injection of procaine hydrochloride by reopening the conjunctiva as soon as intraocular tension and the shape of the globe have become normal. This method proved serviceable.

in several cases in which the prognosis was unfavorable. Injection of the patient's own blood into Tenon's space after the operation, as done by Ariuga, may probably act in a similar manner as the artificial indentation, however, this method was devised by Arruga for the purpose of immobilizing the entire globe.

K. L. STOLL

Trachoma

ALLERGIC REACTION OF TRACHOMA. I. A. DANILEWSKY and P. G. KAMINSKY, *Ann d'ocul* 175:245 (March) 1938

It is natural to expect that a chronic infectious disease like trachoma might set up different changes in the body. Straub pointed out the differences in sensitiveness to trachoma in infants and in adults. Of the Russian writers, Warchawsky drew attention to the fact that a relative immunity to trachoma develops little by little with age and to the importance of this immunity. Ostrounow doubts that there is a development of any such immunity in adults. He draws attention to the great susceptibility of infants to trachoma and to the fact that the condition develops in children with great intensity and reacts poorly to treatment. Besides clinical observations, research has been made on the particular qualities of blood and serum of trachomatous patients, even after they have developed immunity. Many investigators have tried these tests with the aid of vaccine, and the majority conclude that the reaction has no diagnostic significance and is not specific. The results obtained were quite divergent.

Unfortunately the virus of trachoma and its allergic properties being still unknown, the authors employed the technic approved in the clinic of Glaubeisohn. The preparation of the antigen is given in detail, and the reactions obtained in trachomatous and control patients are shown by three charts. A bibliography accompanies the article.

S. H. MCKEE

TREATMENT OF TRACHOMA BY LOCAL AUTOSEROTHERAPY. H. JOURDAN, *Ann d'ocul* 175:254 (March) 1938

Trachoma is one of the ocular diseases for which a great many therapeutic aids, both physical and medicinal, have been tried, with more or less indifferent results. Also, certain of these treatments are exceedingly painful. On this account Jourdan has thought it of interest to publish the results obtained by the use of local autoserotherapy. The technic for the preparation of the serum is simple. It consists in the removal of a certain quantity of blood by aseptic venous puncture, after centrifugation and fractional sterilization, it is put in sealed ampules. The serum is then ready to be injected into the tarsal conjunctiva. The author usually injects 0.5 cm. (rarely more) after the use of an analgesic either once or twice a week. The injections are well taken, sometimes slight pain is complained of, and sometimes a slight swelling occurs, which never lasts for more than two or three days. In all cases there has been a rapid diminution in symptoms and improvement in the conjunctival and corneal irritation. Pannus has

rapidly disappeared, and photophobia and tearing have improved considerably. Jourdan has also noted a more or less rapid diminution in the granulations themselves, though he has observed few patients long enough to judge of the persistence of the cure.

Eight cases are reported by the author, and the progress of the different patients is described. A bibliography accompanies the article.

S H MCKEE

PYREXIA AND TRACHOMA DELANOE and J SEDAN, *Rev internat du trachome* 15.1 (Jan) 1938

The authors have noted in many instances the favorable effect of an intercurrent febrile disease on the corneal complications of trachoma, the conjunctival status remaining, however, uninfluenced. Three cases are cited in detail. In a recent thesis Carlotti demonstrated that artificially produced fever has a similar favorable action.

J E LEBENSOHN

Tumors

MELANOBLASTOMA OF THE LACRIMAL CARUNCLE. REPORT OF A CASE AND REVIEW OF THE LITERATURE. J O WETZEL, *Am J Ophth* 20: 675 (July) 1937

Wetzel discusses the lacrimal caruncle especially from the standpoint of melanotic tumors and their origin from pigmented nevi. The following summary is given:

"The case of melanoblastoma of the lacrimal caruncle here reported occurred in a 76-year-old man. A brown stain at the inner angle of the eye had been noticed nearly eight years before, a few months after a burn to the eye. It remained stationary for nearly five years, then began to grow as a tumor. On examination, the tumor was brownish black in color, pedunculated, and the distal portion was the size of a grape. The palpebral and ocular conjunctivae were invaded by the tumor, and the sclera was infiltrated by pigment. The globe was removed. Histologic examination showed polymorphous-celled melanoblastoma of a highly malignant type. A small dermoidal inclusion was present beneath the conjunctiva. Twenty-nine cases of pigmented tumor of the lacrimal caruncle were found in the literature. An abstract of each case is presented."

W S REESE

DIFFERENCES IN THE MANNER OF PERFORATION OF THE SCLERA CAUSED BY PRIMARY MELANOSARCOMA AND METASTATIC CARCINOMA OF THE UVEAL TRACT. J BRENDL, *Arch f Augenh* 110. 559, 1937

Histologic sections show that metastatic carcinoma of the uveal tract usually perforates the sclera by following the course of the ciliary nerves, whereas primary sarcoma usually perforates by following the course of the blood vessels.

F H ADLER

SARCOMA OF THE OPTIC PAPILLA REPORT OF A CASE S PREVEC,
Klin Monatsbl f Augenh 99: 513 (Oct) 1937.

Prevec reports a case of sarcoma in the optic disk in a man aged 38 who experienced no ocular difficulties until about two years prior to observation. Vision failed more rapidly during the six weeks preceding examination. A horizontal, oval, mushroom-shaped tumor rose out of the optic disk of the right eye into the vitreous chamber. The tumor resembled an enlargement of the optic disk, presented the somewhat indistinct ramification of the central vessels and was grayish red. The retina was slightly detached below the tumor, so that the transition of the vessels was gradual, while their course could not be followed from the upper circumference of the tumor into the retina. Histologic examination showed that the tumor arose from the lower portion of the optic disk. The tumor consisted of two portions: a small flat portion, representing the primary mass which sprang from the choroidal tissues adjoining the disk, and a larger prominent portion, which involved the lower portion of the optic disk. The upper portion of the disk was free from the tumor masses, which, however, surrounded it beneath the retina. For reasons discussed, Prevec thinks that the larger portion of the tumor in the optic disk was secondary and that the small choroidal portion was primary. A similar case was reported by Cunncone, but the condition was considered by him to be a primary neoplasm of the optic disk.

K. L. STOLL

Uvea

BLOOD TRANSFUSION IN UVEITIS L. V. PRITZKER, Vestnik oftal 11: 695, 1937

Pritzker employed the transfusion of blood in 10 cases of severe serous plastic uveitis in which ordinary therapeutic measures proved ineffectual. In 2 cases of long standing with loss of vision due to organized exudates in the vitreous no positive results were obtained. One of these was a case of traumatic iridocyclitis of one and a half years' standing, the second was a case of serous uveitis with secondary glaucoma. In 8 cases (5 of presumably tuberculous origin) in which some vision was present the effect of the transfusion was noticeable in three or four days, the vitreous was clearing rapidly, the precipitates were melting away, and there was a corresponding increase of the visual acuity, in some cases from ability to count fingers to 0.2 and 0.4.

Pritzker believes that the transfusion of blood stimulates the defensive power of the organism. The phagocytic activity of the leukocytes increases, the opsonin quality of the blood serum is improved, and a large number of antibodies appear.

Detailed histories of the patients and a review of the literature are given.

The conclusions are as follows:

1. The transfusion of blood in cases of uveitis due to various factors is an active nonspecific remedy.

2 It is indicated in cases in which no other treatment is effective or in cases in which the cause is unknown

3 In cases of neglected uveitis in which there is no vision because of changes in the vitreous, the transfusion was ineffective

O SITCHEVSKA

Vision

DOES TIME INFLUENCE PERCEPTION OF DEPTH? N C SHEN and E GEBLEWICZ, *Compt rend Soc de biol* 124: 1173, 1937

Reduction of the illumination period to as little as 4 milliseconds had no appreciable influence on the stereoscopic perception of depth. The apparatus used consisted essentially of a stereoscope, and Pulfrich's figures for the determination of stereoscopic acuity traced on plates of superimposed ground glass, transilluminated by a light controlled by a shutter.

J E LEBENSOHN

A NEW APPARATUS FOR RECORDING DEPTH PERCEPTION G COLAJANNI, *Ann di ottal e clin ocul* 65. 684 (Sept) 1937

The author describes his modification of the Howard apparatus by which both ends of the rods are covered in a relatively small box. The observer, placed at from 6 to 10 meters, moves the two lateral rods to the desired position by an electrically controlled arrangement. Constant illumination is provided by bulbs placed inside the box.

S R GIFFORD

NEW TEST CHART TO DETERMINE THE VISUAL ACUITY M C COLENBRANDER, *Klin Monatsbl f Augenh* 99 213 (Aug) 1937

Colenbrander devised a new test chart for the determination of the visual acuity. The scientific and practical reasons for selecting the arrangement and types of the chart are given. Bierens de Haan and Otto Roelofs found that the visible, separable and legible minimums of distinguishing objects depend equally on the quantity of illumination and that the decrease of vision in pathologic conditions registers analogous in each of these three minimums. Colenbrander, therefore, chose the visible minimum. His object is the picture of a schematic clock, consisting of a small black ring with a black disk in its center and a black triangle outside the ring. The triangle and disk are connected by a line, quasi the hand of the clock. The location of the triangle is the detail which determines the perceptibility. The connecting line, or hand of the clock, is too thin to interfere. Clocks of this description are arranged in a spiral on a round chart, the largest outside and the smallest at the central termination of the spiral. Twelve objects are arranged in each turn of the spiral. The author explains why this new chart fulfils all requirements. The clock is superior to various letters, some of which are easier recognized than others, the axis of the astigmatism has no influence on the visibility of the objects, the objects, i.e., the triangles, are arranged in twelve positions along straight and oblique meridians, the spacing is identical with that used on charts with letters.

or numbers, the chart excels in simplicity, but the naming of the objects may be more difficult than that of letters, yet less difficult than the naming of rings and hooks. The vision can be measured at distances of 4, 5 and 64 meters, and other distances may be calculated by means of tables printed on the chart. Memorizing is rendered impossible, as the chart can be rotated.

K. L. STOLL

Therapeutics

SHORT WAVE THERAPY IN OPHTHALMOLOGY. M. KLEIN and G. BÁNYAI, *Ann di ottal e clin ocul.* 65:705 (Sept.) 1937

After some animal experimentation, the authors concluded that freedom from burns could be assured by depending on the patient's sense of heat never surpassing the current which gave a pleasant sensation of heat. In the treatment of acute inflammation the dose was kept low enough to avoid any sensation of heat, and the duration of treatments was from four to eight minutes. The duration of the treatment for chronic conditions was from five to fifteen minutes. Schliephake's electrodes, from 4 to 8 cm in diameter, were employed, the smaller electrode being placed in the temporal region or from 3 to 5 cm in front of the eye, while the larger one was held from 8 to 10 cm from the skin over the occiput.

One hundred and twenty-one patients were treated, including 18 with inflammation of the lids, orbit and lacrimal apparatus, 95 with inflammation of the anterior segment, and 8 with choroiditis, retinitis and hemorrhages of the retina and vitreous. In all cases the treatment was well tolerated. Subjective symptoms were nearly always improved. Dramatic improvement was observed in pyogenic processes of the lids and orbit. In cases of keratitis, especially of the herpetic type, good effects were observed [only after from eight to fifteen treatments (S. R. G.)]. In cases of retinitis and retinal hemorrhage no harmful results were observed, and the hemorrhages seemed to clear somewhat more rapidly as the result of treatment. No case histories are given.

S. R. GIFFORD

Society Transactions

EDITED BY W L BENEDICT

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

JAMES W WHITE, M D, *Chairman*

April 18, 1938

RUDOLF AEBLI, M D, *Secretary*

UNILATERAL RETINITIS PIGMENTOSA REPORT OF TWO CASES DR SIG- MUND A AGATSTON

The occurrence of bona fide cases of unilateral retinitis pigmentosa again stirs up the controversy as to the etiology

In spite of the fact that numerous theories have been promulgated regarding the pathogenesis of retinitis pigmentosa, no single author has been able to offer an unassailable explanation. The abiotrophic theory is supported by the occurrence of cerebral involvement in atypical cases of tapetoretinal degeneration. The suggestion of a traumatic origin of the disease promoted by Wagermann's experiments does not seem to hold when based on statistics, which show that retinitis pigmentosa following trauma is practically unknown. The angiospastic theory is surely unsatisfactory, because it is difficult to conceive the occurrence of arteriolar spasms at an early age and limited to the retinal vessels. Also, if this were true, retinitis pigmentosa would be a common occurrence in cases of essential and malignant hypertension, which it is not. Neither does it occur in cases of secondary atrophy associated with optic neuritis, retrobulbar neuritis or toxic amblyopia. Certain it is that in all typical cases there is a gradual occlusion. So constant is this vascular change that without it diagnosis is impossible. Moreover, so characteristic is the appearance of the disk and vessels that in the absence of malignant hypertension the disease may be recognized before the periphery or pigment spots are seen. The pigment spots so constantly observed in retinitis pigmentosa is also seen in other diseases, such as retinosis, choroidosis, choroideremia, chorioretinitis and topical senile degeneration of the peripheral portion of the retina. The arterial picture, however, belongs to retinitis pigmentosa. It is not likely that the retinal vessels are narrow because of general atrophy of the retina, of which they are not really a part. In diffuse chorioretinitis in which practically the whole retina is destroyed the retinal arteries are normal.

It seems to me that if one could find an explanation for the gradual narrowing of the vessels a workable theory would be found which would be applicable to the solution of the etiology. It is known now that narrowing of vessels with fibrotic and hyperplastic changes is caused by any form of occlusion. Whether that occlusion is produced by an

embolus, thrombus, pressure or spasm, it makes little difference. And so the vessels found in association with retinitis pigmentosa resemble those observed in cases of malignant hypertension or following intra-bulbar or retrobulbar neuritis. In other words, the changes in the vessels are not secondary to general atrophy but secondary to pressure and occlusion.

One can conceive the occurrence of a topical neurofibromatous formation. This may be limited to the central portion of the optic nerve or may be found elsewhere in the nervous system. This would explain the cases of cerebral involvement and the possible association with otosclerosis. In explanation of this hyperplasia one might draw a parallel with what happens in Recklinghausen's disease. Wagemann's experiments also need not be disregarded. He showed that cutting off the vessels behind the globe in rabbits resulted in pigmentary degeneration. Trauma to the eye will not result in pigmentary degeneration unless the retinal vessels are compressed by scar tissue. My own experiments on rabbits, consisting of the injection of alcohol into the optic nerve trunk, gave results similar to those of Wagemann.

It appears that sudden blocking of the retinal artery does not result in pigmentary degeneration, but gradual occlusion occurring early in life (retinitis pigmentosa has its onset between the ages of 3 to 8 years, according to Horing and others) causes a gradual degeneration of the peripheral portion of the retina with replacement of necrotic spots by proliferating pigment epithelium. This happens because the terminal small branches become fibrotic and early in the disease lose their patency.

Shoemaker in his monograph reported massive hyperplasia within the optic nerve as a postmortem observation. In the pathologic laboratory at the Montefiore Hospital Dr. Smoleroff and I have been able to demonstrate similar changes. The occurrence of unilateral retinitis pigmentosa, of which there should be no doubt, while it militates against other theories does not conflict with the theory of intraneural hyperplasia.

Dr. M. N. Beigelman, in reporting a case in the *ARCHIVES* (6:254 [Aug.] 1931), gave a résumé of 11 cases of unilateral retinitis pigmentosa reported since 1865. In that year the first case was reported by Pedraglia. The oldest patient was 42 and the youngest 10.

The ages of my patients were 45 and 53.

One of them, a man aged 53, had poor vision in the right eye for thirty-eight years. The fundus showed retinitis pigmentosa with atrophy of the optic nerve and narrow blood vessels. The other, a woman aged 45, had failing vision in the left eye which began at the age of 14. There were a posterior cortical cataract, a typical picture of retinitis pigmentosa with sclerosis of the choroidal vessels, thin retinal arteries and pallor of the disks.

COLOBOMA OF THE OPTIC NERVE AND OF THE MACULA. A MICROSCOPIC STRUCTURE STUDY. DR. DAVID WEXLER AND DR. MURRAY LAST.

This article will be published in a later issue of the *ARCHIVES*.

MALIGNANT NEOPLASM OF THE EYELID REPORT OF TWO CASES DR MAYNARD C WHEELER

A man of 68 complained of a lump in his left lower lid of two months' duration. A firm mass occupying practically the entire lid was deforming the tarsus, the skin was not involved. The tumor was removed. A histologic diagnosis of lymphosarcoma was made. One roentgen treatment was given. The patient was free from recurrence six months after excision.

A girl of 19 complained of a lump in her right lower lid which began like a sty three years previously. Later it was curetted. Gradually it increased in size. The temporal two thirds of the lid was occupied by a firm mass involving both the skin and the tarsus. Biopsy showed carcinoma. The lid and tumor were excised. The histologic diagnosis was carcinoma of the meibomian gland. Two years later there was no evidence of recurrence or metastasis.

VON HIPPEL'S DISEASE REPORT OF A CASE DR BENJAMIN C ROSENTHAL

A man 29 years of age with a past history of meningeal involvement came to the New York Eye and Ear Infirmary because of blurring of vision in his left eye. His left fundus showed the typical picture of von Hippel's disease (angiomas of retinae). Examinations of the visual field of the diseased eye showed an irregular positive central scotoma. There was a slight enlargement of the blind spot. There were no hemianopic or quadrant defects of the visual field in either eye. There was a slight concentric contraction of the peripheral field in the left eye. Medical, neurologic and laboratory tests gave negative results.

The father of this patient died at the age of 46 years with symptoms of an intracranial condition. Autopsy was performed, and the pathologic diagnosis was Lindau's disease.

These cases demonstrate the familial nature of the diseases of von Hippel and Lindau. Angiomas of retinae is but a part, or an early manifestation, of a more widespread pathologic syndrome, namely, Lindau's disease.

OPERATIONS FOR ORBITAL FILLINGS DR JOHN WHEELER

This article will be published in a later issue of the ARCHIVES

JAMES W WHITE, M D , *Chairman*

May 16, 1938

RUDOLF AEBLI, M D , *Secretary*

INTRAOCULAR TENSION IN SARCOMA OF THE CHOROID AND CILIARY BODY DR JOHN H DUNNINGTON

This article appeared in the September issue of the ARCHIVES,
page 359

OCULAR HYPERTELORISM REPORT OF A CASE DR WEBB W WEEKS
(read by Dr Benjamin Jaffe)

The following case is reported because of the rarity of the condition. Up to 1935 only 24 such cases had been reported.

Hypertelorism is a developmental defect of the lesser wings of the sphenoid bone, they ossify early in intrauterine life and grow excessively. This causes the typical widely separated eyes. Mental retardation may occur. Hypertelorism also occurs as a familial inherited anomaly.

A man aged 43 complained of sensations of foreign bodies in each eye. A diagnosis of marginal blepharitis and presbyopia was made, and he was given glasses for reading. Vision for distance was 20/15 in each eye without correction. The patient was seen several times later the same year and treated for conjunctivitis and blepharitis.

He did not come to the clinic again until March 1938, when he appeared complaining of foreign bodies in each eye. Nothing unusual was noted except the peculiarly widely separated orbits. The patient volunteered the information that he had rudimentary clavicles. He was sent to the roentgenographic department for a study of congenital bony anomalies, with the provisional diagnosis of hypertelorism in mind. The diagnosis was verified by the roentgen examination.

The roentgen report follows. There was an increase in the transverse diameter of the skull, resulting in a brachycephalic type. The metopic suture was patent. The interorbital and interpupillary distances were increased. The findings indicated hypertelorism. The lower third molars were impacted, and the upper third molars were impacted and unerupted. The clavicles were rudimentary.

DISCUSSION

DR RALPH F LLOYD Did this patient have any malformations of the fingers or toes?

DR BENJAMIN JAFFE No, there were just the rudimentary clavicles, and the molar teeth were unerupted.

AN ANOMALOUS RETINAL VEIN DR A L KORNZWEIG

A girl aged 18 came to the Mount Sinai Hospital to have her eyes retested for glasses. During the routine examination of the fundus of the right eye it was noted that a branch of the inferior temporal vein passed upward toward the macula and split the foveal reflex into two parts, one above and one below the vein. Angioscotometry showed a scotoma above the central point of fixation, proving that the true fovea is above the vein.

PRECANCEROUS MELANOSIS OF THE LIDS AND CONJUNCTIVA DR
ALGERNON B REESE

H G, a man aged 45, first noticed a spot of pigment on the left lower lid about the size of a pinpoint fifteen years before the present examination. Ten years before he estimated that the spot had increased only to about 2 mm in size. Five years before, when he

was seen for the first time at the Memorial Hospital, there was pigmentation of the skin of the lower lid over an area 25 mm long and 6 mm wide, including the margin of the lid, and for a short distance on to the adjacent conjunctiva at the external canthus. There was no elevation or induration of the skin, which, except for the pigmentation, appeared to be entirely normal. In eight months the lesion had spread along the margin of the upper lid for 7 mm from the external canthus. In two months some pigment appeared in the lower palpebral conjunctiva near the fornix. In six months more the pigmentation had extended along the entire extent of the lower lid and for 10 mm along the margin of the upper lid adjacent to the external canthus.

A section of the involved skin was removed at the Beth-El Hospital in 1932. This section has been studied by Dr. Ewing and Dr. Stewart of the Memorial Hospital, who termed the growth a "melanosis."

A diagnosis was made of precancerous melanosis of the skin and conjunctiva, which was beginning, after fifteen years, to show malignant tendencies.

DISAPPEARANCE OF THE IRIS AFTER PROLONGED HYPHEMIA DR GUERNSEY FREY

The case reported here is of considerable clinical interest. A 6 year old boy was seen at the Manhattan Eye, Ear and Throat Hospital on Dec. 18, 1936, shortly after having injured the left eye with scissors. A perforation of the lower lid and a large subconjunctival hemorrhage were noted, with a normal fundus. The boy returned on April 6, 1937, having had scarlet fever three weeks previously, the left eye had been sore, and vision had been impaired for twenty-four hours. Dr. Hulka made a diagnosis of abscess in the vitreous, probably metastatic.

On April 6 the child appeared at the outpatient department of the Queens General Hospital with the anterior chamber of the left eye filled with blood. The tension was low. He was admitted to the hospital. No cause was found for the hyphema, which persisted for three months. During this time various coagulants were administered, including two courses of moccasin venom, but the blood did not absorb until a transfusion was performed. During most of this time the blood occupied the greater part of the anterior chamber, only a small amount of blue iris being exposed. When the blood had finally absorbed, the iris appeared to be entirely absent except for the portion which had previously been observed above the hyphema. There was a yellow pupillary reflex from an exudate in the vitreous, and there was no perception of light. The eye gradually became phthisic and was enucleated in the fall.

The pathologic examination revealed that whereas the iris below appeared to be completely absorbed, it was in fact atrophic and retracted, with ectropion of the pigment layer. There were also evident an old healed penetrating wound of the globe and endophthalmitis.

CONGENITAL FISTULA OF THE LACRIMAL GLAND DR GUERNSEY FREY

A 7 year old boy had a small orifice in the skin about 5 mm temporal to and 1 mm above the external commissure of the eyelids of the right eye, from which tears had discharged since birth. He was admitted to Dr Fletcher's clinic at the Manhattan Eye, Ear and Throat Hospital as a new patient on March 19, 1938. On March 31 photographs were taken, the tract was injected with iodized poppy-seed oil 40 per cent, and roentgenograms were made. A communication of the fistulous tract with the convoluted tubules of the lacrimal gland was demonstrated. After injection of the iodized oil the tearing from the fistula ceased for a week, but then recurred.

Congenital fistula of the lacrimal gland is an extraordinarily rare condition. In the last twenty years but 3 cases have been mentioned in the literature. T. Schornstein reported a case in the *Archives für Augenheilkunde* (109: 86-102, 1935) in which the fistulous opening appeared on the upper lid near the outer commissure and was surrounded by four cilia. A. Cange and H. Duboucher in the March 1931 issue of the *Archives d'ophtalmologie* (page 161-186) reported a case in which the tract was dissected out. They called attention to the fact that congenital cysts of the lacrimal gland may subsequently be converted into fistulas by trauma, operation or spontaneous rupture. The third case, reported by M. E. Alvaro and A. Sampaio Doria in the *Revista oto-neuro-oftalmológica y de cirugía neurológica sud-americana* (12: 283-290, 1937), closely resembled the present one. In "The American Encyclopedia of Ophthalmology" (vol 4, p 2943) reference is made to cases of fistula of the lacrimal gland reported by Mackenzie, Steinheim and Terlinick.

The treatment recommended by these authors consists in transplantation of the secretory duct to a position where it will discharge into the conjunctival cul-de-sac. If this is unsuccessful, the fistulous duct and the lacrimal gland, or that portion of it with which the duct is connected, must be excised. My intention is to pass a probe into the fistulous tract and then cut down on it from the conjunctival surface to effect a new orifice into the superior conjunctival cul-de-sac. At the same time, I shall curet and tie off the present fistulous opening.

LUXATION OF THE LENS IN THE ANTERIOR CHAMBER, OF CONGENITAL ORIGIN, IN TWO MEMBERS OF A FAMILY, FOLLOWED BY SECONDARY GLAUCOMA EXTRACTION OF LENSES DR MARTIN COHEN

CASE 1 —Miss R. S., aged 26, was first examined in February 1934, at which time she had myopia of 10 diopters in each eye. With myopic correction, vision in each eye was 20/40. A downward subluxation of both lenses was recognized at the time. Physical examination gave negative results.

The patient returned on May 1, 1937, with luxation of the right lens in the anterior chamber and secondary glaucoma associated with headache, vomiting and severe ocular pain. She was admitted to the hospital, where a small cataract section above with iridectomy was performed and the lens was extracted with the aid of a wire loop. The resulting visual acuity in this eye was 20/20 with a + 9 diopter lens.

There were some opacities of the vitreous. The fundus appeared normal.

Vision in the left eye, in which the lens was subluxated, amounted to 20/30—2 with a +12 diopter lens, obtained from the aphakic pupillary area. The fundus, which could be seen through the aphakic portion of the pupil, appeared normal.

CASE 2—Miss M. S., a sister of the patient in case 1, was first examined in January 1932 at the age of 7. There was a history of poor vision for three years, which was not improved with glasses. The patient had had various diseases of childhood. Physical examination gave negative results. Both lenses were subluxated downward. The patient was admitted to the hospital, and a needling of the lens of the left eye was performed, without any effect. She was readmitted to the hospital on Sept. 10, 1933, with symptoms of acute pain in the right eye of two days' duration. The conjunctiva was injected, and the intraocular tension was elevated. The lens was luxated in the anterior chamber. A small cataract section above with iridectomy was performed, and the lens was extracted with the aid of a wire loop. The resulting vision in this eye with a +5 diopter sphere was 20/100. The fundus showed a myopic conus and slight pallor on the temporal portion of the disk.

The patient was reexamined periodically, and on April 2, 1938, she was readmitted to the hospital with luxation of the left lens in the anterior chamber and secondary glaucoma associated with headache, nausea and severe ocular pain. A small cataract section was made above. The section was enlarged with scissors, followed by iridectomy, and then the lens was extracted with the aid of a wire loop, resulting in a visual acuity of 20/40 with a +15 diopter sphere. The fundus of this eye appeared normal.

UNILATERAL EXOPHTHALMOS DUE TO A SCLEROSING SARCOMA OF THE ANTERIOR CRANIAL FOSSA. DR. MARTIN COHEN

Mrs. F. B., aged 75, was struck in the left eye by the horn of a cow at the age of 6 years. The injury healed in one year. Following the trauma, the vision was markedly reduced. There was some vision in the involved eye until two and one-half years before the present examination. Since then, only perception of light has been present. Following the injury, there was some protrusion of the left eyeball, which became more apparent about seven years ago. For the past four years there has been no apparent change in the exophthalmos. Physical examination, including the Wassermann test, gave negative results.

Recent examination revealed the following facts. Vision in the right eye, without glasses, equaled 1/200. With glasses, vision was corrected to 20/200. The intraocular tension (Schiotz) was 14 mm of mercury. The pupil reacted to light and in accommodation. There were present opacities of the vitreous and lens. The fundus showed a posterior staphyloma, peripapillary choroidal atrophy, an area of pigmentation in the nasal quadrant of the fundus, attenuated arteries and slightly engorged veins.

Vision in the left, or involved, eye was limited to perception of light. The intraocular tension (Schiotz) was 14 mm of mercury. An exophthalmos of 5 mm was present. Motility of the eyeball was normal in all directions. Transillumination was slightly diminished in the inferior field. External examination showed slight conjunctival injection, also, moderate noninflammatory edema of the inner portion of the upper and lower lids. The bony orbit was slightly thickened above and nasally. There was an old scar of the cornea. The opacities of the lens and vitreous were more marked in this eye. The left pupil appeared smaller than the right and reacted to light and in accommodation. The field of vision was markedly contracted nasally and slightly contracted in the superior field. The details of the fundus could not be recognized.

A roentgenogram of the left orbit was made by Dr. William H. Meyer to demonstrate possible hypertrophy of the soft tissue. The report was as follows: Examination showed a marked increase in density ascribable to bony sclerosis and hypertrophy, suggesting a neoplasm (probably sarcomatous) involving in the main the anterior fossa on the left side.

The invasion extended across the median line into the frontal and ethmoid sinuses and downward on the mesial side of the orbit, into the malar bone and encroaching on the nares. It also extended backward, mainly along the lesser wing of the sphenoid bone toward the middle fossa. Alternating areas of condensation and porosity continued over the anterior portion of the vault, mainly on the left side, into the parietal bones.

The conclusion was that the condition was a sclerosing sarcoma of the anterior fossa, mainly of the left frontal bone.

Biopsy will be performed and reported on at a future date in order to establish a definite pathologic diagnosis. Surgical intervention will not be considered owing to the extent of the growth and the age of the patient. Roentgen treatment will be instituted.

TAY-SACHS DISEASE. REPORT OF A CASE. DR. JOSEPH LAVAL

A child 1 year old developed normally until the age of 5 months. The parents are Jews, and this is the second child of the marriage. The characteristic cherry-red spot was present. It was further noted that even though the ganglion cells of the retina were swollen and resembled xanthoma cells, they were not true xanthoma cells. Accordingly, there is no connection between amaurotic family idiocy and xanthomatosis such as occurs in Gaucher's disease, the Niemann-Pick syndrome or the Schuller-Christian syndrome.

ZONULAR OPACITY OF THE CORNEA. DR. M. DAVIDSON

Zonular opacity or ribbon-shaped, band-shaped or calcareous keratitis is described by Fuchs as the most frequent of the pathologic types of dystrophy, developing in eyes either because of intraocular disease or in senile persons or because of external injurious influences to which the eyes have been subjected in the region of the palpebral fissure. The last type has been observed in hat makers and in persons exposed to mercurial vapors and to the blowing of mild mercurous chloride into the

eyes for long periods. Under a section entitled "Conditions Allied to Dystrophies," Fuchs mentioned lime, lead, silver, naphthalene and iron as giving rise to a somewhat similar picture.

The case presented here is interesting in that it shows what happens to an eye with lime incrustation in the course of years. The patient was examined at the Bureau of Workmen's Compensation because of a lime burn in the left eye, the condition found in the right eye was due to a lime burn received over twenty years before. In addition to a lime incrustation in the upper part of the cornea, there was one in the lower corneal half, with the typical etched subepithelial lines, and chalky white calculi lying at different levels in the corneal stroma as far back as Descemet's membrane, where they were star shaped, lying in round clearings in a homogeneously, somewhat opaque membrane. I saw another patient recently in whom the same transportation of lime had progressed more rapidly, to deposit calculi in the course of two years, without reaching Descemet's membrane. I have not seen any references to this progressive transportation of lime from a lime incrustation toward the aqueous, although the difference between albumin-dissolving alkalis and albumin-precipitating acids in their effects on the eyes was noted long ago, the former being more penetrating.

NEUROPARALYTIC KERATITIS DR MORRIS JAFFE

In neuroparalytic keratitis the abnormal metabolism of the epithelial cells puts them in a position in which they fail to resist the effects of trauma, drying or infection. The condition occurs following a lesion to the fifth nerve in any part of its course. It develops much more rapidly and acutely following ganglionic or infraganglionic lesions than following a lesion above the ganglion. Keratitis develops in about one third of the cases in which the lesion is in the latter situation. The condition has been observed to follow tumors in the brain stem, tabes and basilar meningitis. In cases of disseminated sclerosis, anesthesia of the cornea with ulcerations has been noted. The clinical picture is as follows. Hyperemia of the conjunctiva will appear, which gradually diminishes in intensity. Adequate protection of the eye at this stage will prevent complications. Iritis frequently sets in sometimes before and sometimes after corneal changes. A corneal disturbance starts often within a day or two after the central lesion and is ushered in by the appearance of a stippled haze, due to edema, which may be followed by vesicle formation. This process may remain stationary, resembling a superficial punctate keratitis, but more usually it is followed by massive exfoliation of the epithelium of varying degree. The denuded surface appears dry and hazy. When a secondary infection gains foothold, ulceration develops, which may end in perforation. The cornea is anesthetic.

Neuroparalytic keratitis is exceedingly slow and chronic, with no acute phenomena, principally because of the corneal anesthesia. If treatment is started early and the cornea protected for a sufficiently long time, the prognosis is good, otherwise, it is bad, and the best one can anticipate is widespread leukoma.

The best treatment is prophylactic. The eye should be covered with a pad and bandage over carefully adjusted closed lids. If despite this precaution epithelial degeneration occurs, tarsorrhaphy should be done.

at once. It is usually followed by rapid improvement, and it should be maintained for from six to twelve months. Eventually the corneal tissues appear to become adjusted to the new conditions of their metabolism, and the eye will keep well if ordinary precautions are taken against trauma.

A white man aged 62 suffered from severe pain in the right side of the head, beginning in September 1937. Early in November he noted hyperemia of his eye with partial lagophthalmos. He first came under my observation on Jan. 10, 1938. He had a corneal ulcer below the pupillary margin, with epithelial exfoliation around it, and with plastic iritis. The cornea was completely anesthetic. When an effort was made to close the lids of both eyes gently, the lids of the right eye only partially closed, leaving an exposed area of cornea about 2 mm. wide. The corneal surface was cleansed and touched up with synthetic oil of wintergreen. The eye was bandaged over a period of three and one-half months, resulting in definite improvement.

DACRYOADENITIS. REPORT OF TWO CASES. DR. HENRY MINSKY

The first patient, a 3 year old child, had a history of progressive swelling of the left upper lid of one week's duration, the swelling was so extensive that an abscess was suspected. She had slight fever and a high leukocyte count. The condition improved rapidly under the use of hot compresses. The second patient, a middle-aged man, had involvement of the peripheral portion of the lid only, which resolved, with some breaking down of tissues. These 2 cases are reported to demonstrate a constant sign in the differential diagnosis of orbital dacryoadenitis. In either case the normal sharp edge of the outer two fifths of the upper orbital margin could not be felt because of the fulness which ended abruptly at the junction of the inner three fifths by a bandlike resistance extending toward the margin of the lid for about 1 cm.

ACUTE MEMBRANOUS CONJUNCTIVITIS IN A BRASS WORKER. DR. FRANCES RICHMAN

C. G., white, aged 40, had worked as a tinsmith and brass turner for the past few years. During this time he had had occasional attacks of mild conjunctivitis, acne of the face and gingivitis. His history otherwise was essentially irrelevant.

The present complaint began acutely on Aug. 30, 1937, with pain and redness of the eyes and nasopharynx and a bronchial cough. For three entire days prior to the onset the patient had been steadily grinding brass articles at a wheel that had no blower connections, he reported that the air was filled with the dust thrown off from the grindstone and metal and that this dust filtered in through the perforations in his goggles, entered his eyes, covered all exposed surfaces of skin and was inhaled freely. Perspiration stains on his undershirt and handkerchief were greenish colored, the sputum was tinged with green.

The symptoms progressed rapidly, and he sought treatment at an ophthalmic clinic. The secretion and milky appearance of the conjunctiva suggested a diagnosis of vernal catarrh, and he was given a lotion for his eyes. On the third day the condition of the eyes and throat was

alarming, and he was admitted to the Kings County Hospital to the medical service

At this time the outstanding clinical feature was the presence of a membrane which covered the conjunctiva of both eyes, the nasopharynx and tonsils, the buccal mucous membrane and the gums. Although the membrane was not typically diphtheritic in appearance, it was compulsory to isolate the patient and give him proper antitoxin treatment while waiting for laboratory reports. The health department reported culture of material from the throat to be negative for diphtheria, repeated cultures of material from the eyes and throat showed *Staphylococcus albus* and *Staphylococcus aureus*, and smears showed pus cells and a few fusiform bacilli suggestive of Vincent's infection.

The acute symptoms subsided slowly. Weeks later the eyes still showed marked blepharospasm, photophobia, congestion of the conjunctival vessels, thickening and a milky appearance of the transitional folds, which were still covered with a shreddy secretion, fine reticular scarring of the conjunctiva with vacuolation between the bands of scar tissue, and a generalized, nonstaining, superficial keratitis of the left eye, the cornea remaining sensitive. Four months later, while the conjunctival scarring was being studied with the slit lamp, several foreign particles were seen embedded subconjunctivally at the upper tarsal border of the left eye, visible only in indirect light and entirely flooded out in the direct beam. The finding of the foreign bodies took the condition out of the medical class and placed it definitely among occupational traumas, thus settling the controversial question of compensability. Delicate tests for copper were done on three of the removed particles, with negative results; alternatively, it may be that these were particles of silicate from the grindstone which were sealed in the conjunctiva when it healed over subsequent to the casting off of the original membrane.

This case emphasizes the following interesting points: (1) the non-diphtheritic origin of some membranous formations, (2) the marked sensitiveness of the mucous membranes to the mechanicochemical irritation with fine metallic dust, the reaction being similar to that produced by chemicals and toxins, (3) the type of corneal reaction elicited by the long-continued conjunctival irritation, (4) the importance of using the slit lamp in studying a conjunctival pathologic process, and (5) the essential importance of searching for minute contrast in indirect light only.

DISCUSSION

DR JAMES W. SMITH. I was asked to see this patient by an insurance company three and a half months after the onset of the conjunctival and corneal irritation. They were at a loss to understand why the patient was being treated so long after the simple lodging of dust in the eyes. From Dr. Richman's modest statement one would infer that she missed the diagnosis. She failed to suggest the fact that a rather firm and extensive membrane lined all the palpebral conjunctival surfaces for nearly four months after the original injury and that it was not until the membranous formation had ceased that it was possible to see by slit lamp microscopy eight or ten minute foreign bodies embedded in varying depths of the upper conjunctiva of the left eye.

RETINITIS PIGMENTOSA WITH GLAUCOMA DR CHARLES A PERERA

The combination of pigmentary degeneration of the retina and glaucoma is of sufficient rarity and interest to warrant the following case report. The patient was under the care of Dr Charles H May for many years, and the case is presented with his permission.

Miss F T, a 24 year old Russian Jewess, gave a history of night blindness and poor vision for many years. I first examined her in May 1936. She was underweight. Ocular examination showed vision to be 15/60 and 15/30 in the right and left eyes, respectively. The intraocular pressure was normal. There was moderate weakness of convergence. The anterior chambers were shallow, and the media were clear. The fundi revealed marked narrowing of the vessels, with pallor and blurring of the nerve heads. The peripheral portion of the retina contained numerous small deposits of pigment of irregular shape. Scattered through each fundus were small yellowish areas with indistinct outlines. The visual fields were contracted concentrically to 15 degrees from fixation in each eye. The refraction was measured under cycloplegia with homatropine hydrobromide. Vision of the right eye was 15/30— with a 3.00 sph \ominus +1.25 cyl, ax 105, vision of the left eye was 15/25 with a +3.00 sph \ominus +0.75 cyl, ax 85.

The patient returned one month later, stating that she had noticed temporary blurring of vision of the left eye five days before, associated with headache on the left side and lasting for five hours. Careful examination revealed the same findings as on the previous visit. The slit lamp showed no evidence of active inflammation. Vascular spasm was considered as a possible cause for the symptoms.

Four months later, in October 1936, the patient was seen with inflammation of the left eye of twenty-four hours' duration. Examination showed acute iridocyclitis of the left eye and normal intraocular pressure in each eye. Complete physical examination gave negative results, the laboratory tests, including a Wassermann test of the blood, examination of the sputum, urinalysis and studies of the blood, also gave negative results. Roentgenograms of the teeth showed no oral foci of infection. Under treatment with 1 per cent atropine sulfate and hot compresses locally and salicylates internally, the left eye became quiet in four weeks.

In August 1937 the patient returned with the history of pain and redness of her left eye two days before. The ocular findings corresponded to those noted when I first saw her.

In November 1937 the patient appeared with an attack of acute glaucoma involving the left eye, which was stony hard. She was admitted to the Institute of Ophthalmology of the Presbyterian Hospital, where the intraocular pressure of the left eye was reduced to nearly normal by repeated instillations of a 1 per cent solution of physostigmine salicylate and a 2 per cent solution of pilocarpine hydrochloride. On the third day after the patient's admission, the intraocular pressure of each eye suddenly rose to over 85 mm of mercury (Schiotz), with marked corneal edema. Miotics were of no avail, and a bilateral Lagrange sclerectomy with complete iridectomy was performed. The patient was discharged three weeks later, with

normal tension. She is now instilling a 1 per cent solution of pilocarpine hydrochloride into each eye three times a day. Gonioscopic examination, confirmed by Dr Uribe Troncoso, showed the angles of each anterior chamber to be completely closed, a ciliary process was caught in the scar of the operative wound of one eye.

On the patient's last visit on May 13, 1938, vision of the right eye with correction was 15/40 + and that of the left eye was 15/30. The intraocular pressure was 13 mm of mercury in the right eye and 18 mm in the left eye (Schiotz). The fields and fundi were unchanged.

The literature on glaucoma in cases of retinitis pigmentosa has been well reviewed by Weiss in 1903 and by Kotharevskaya in 1931. At least 32 cases have been reported, many of them by Russian oculists. Some authors have postulated an antagonism between retinitis pigmentosa and glaucoma to explain the rare occurrence of the combination. Many writers have explained the presence of both diseases in the same eye as a result of vascular disease which has caused changes in the vortex veins as well as in the retinal and choroidal vessels. Blessig reported 2 cases of glaucoma and 3 of retinitis pigmentosa in a family of 9 children, thus demonstrating that there may be a congenital and hereditary aspect to these conditions.

The case reported here was that of an atypical form of pigmentary degeneration of the retina, in which an inflammatory element could not be ruled out. In this case a congestive type of glaucoma developed. Vascular disease can explain the simultaneous presence of the two processes.

RETINITIS PIGMENTOSA WITH "HOLE" IN THE MACULA. REPORT OF A CASE. DR CHARLES A. PERERA

This article appeared in the September issue of the ARCHIVES, page 471.

INTRAOCULAR FOREIGN BODY. REPORT OF A CASE. DR DONALD W. BOGART

The patient gave a history of being struck in the right eye with a flying piece of cast iron the day before examination. The vision in each eye was 20/30. There was slight redness of the conjunctiva temporal to the limbus, with a small laceration. A roentgenogram taken routinely showed an intraocular foreign body 1 by 1.5 mm in size localized in the posterior segment in the lower right quadrant. The pupil was then dilated, and the foreign body was easily seen lying 3 disk diameters temporal and inferior to the disk along a branch of the inferior temporal vein. A small blood clot could be seen in the vitreous. It was extracted two days after the accident by the use of magnets. Recovery was uneventful. Ultimate vision was 20/30 without accommodative changes or defects in the visual field.

AN OPERATION FOR CONGENITAL PTOSIS. DR GIROLAMO BONACCOLTO

The surgical technic presented here was first devised by Dr Bardelli of Florence. This operation is intended to correct ptosis of the lid which does not involve paralysis of the superior rectus muscle. An

incision is made along the superior fornix, and after the isolation of the superior rectus muscle two lateral bundles of muscle fibers are cut from the main tendon at 1 cm from their insertion. In this way the insertion of the superior rectus muscle is not disturbed. These two bundles, after they have been separated and cut off from the superior rectus muscle, are, by means of sutures and a special large, flat needle with a sharp edge, passed through the thickness of the upper lid at the height of the superior margin of the tarsus, in front of the tarsus and out of the skin of the lid at about 3 mm from its external edge. By means of this method one can graduate the elevation of the upper lid, which also presents a well balanced horizontal elevation, since the two strips of muscles are implanted at equal distance from the canthi.

PROPTOSIS IN A SIX WEEK OLD INFANT DR. M. GREENBERG

D. M., a 6 week old boy, was admitted to the hospital with a history of proptosis of the left eye of three days' duration. The past history and the family history were unimportant. General physical examination, including an examination of the nose and throat, gave negative results. Roentgenograms were not helpful in making the diagnosis. There was a mild leukocytosis, the white cell count being 11,400, with 44 per cent polymorphonuclears. The temperature was 100 F. An orbital abscess was diagnosed, the orbit was aspirated, pus was found, and incision and drainage were done. The proptosis practically disappeared in four weeks.

Book Reviews

Hypokalzämie und Linse Ein Beitrag zur Behandlung der Tetanie und Cataracta tetanica mit A T 10 Holtz [Dihydratichysterol] (Beiheft der Klinischen Monatsblätter für Augenheilkunde) By Prof A Meesmann Pp 66, with 36 illustrations Stuttgart Ferdinand Enke, 1938

During the past years progress has been reported in the treatment of hypocalcemic tetany with dihydratichysterol, as prepared by Holtz. The close relation between diseases of ectodermal structures like the lens and tetany has led to this review.

A 0.5 per cent solution of dihydratichysterol in oil is given by mouth. The knowledge concerning latent and relative parathyroid insufficiency has been increased. Presenile cataract is frequently observed in cases of the former type. Hence the oculist may be the first one to suggest a possible association.

The hormone of the parathyroid gland regulates the distribution of calcium in the soft tissues. Lack of calcium produces the symptoms of tetany, which consist of tonic convulsions, paresthesia, trophic disturbances in the teeth, nails, hair and skin and tetanic cataract. The latent manifestation of calcium deficiency may occur in a great variety of forms, and relative hypofunction of the epithelial bodies is unusually frequent. The symptoms as they affect the central nervous system, the special sense organs, the circulation, the skin and the digestion are described.

The rudimentary forms of this malady are important. They depend on a constitutional underfunction of the parathyroid gland, and the symptoms become manifest when the gland is overexcited. The classic symptoms of manifest tetany are absent, and the symptoms are not severe and appear only after bodily exertion, psychic exhaustion or during pregnancy. In the diagnosis it is self evident that the mechanical and electrical stimulation of the muscles should be tested and the calcium content of the blood determined. In the rudimentary form, the calcium content varies from 8 to 9.5 mg per hundred cubic centimeters and not infrequently it is found at the lower level of normal (from 9.5 to 11 mg per hundred cubic centimeters).

On examination with the slit lamp tetanic cataract appears in the form of opacities, which are subcapsular and at an early stage involve the cortex at the posterior pole. It is important to carry out slit lamp examination in cases of recent involvement according to the regredient method of Vogt, subcapsular opacities will be discovered in the anterior capsule in the form of small drops. These symptoms are, however, not characteristic of tetanic cataract, as they also occur in cases of cataract associated with myotonic dystrophy and neurodermatitis.

The author reports on 10 patients with cataract but without evidence of tetany, on 15 with tetanic cataract, both groups of whom were observed up to five years, and on 15 patients with tetanic cataract who were observed for not more than one year. The results of treatment

with the solution of dihydrotachysterol were as follows. It was necessary to extract the cataract in only 2 cases. In all of the other cases in which the patients were observed up to five years there was no increase in the subjective or objective symptoms. It is, of course, not possible to say that in later life there may not be a further progress of the opacity of the lens.

Patients with other types of cataracts were examined for the presence of symptoms of tetany. The subject of zonular cataract is gone into carefully, and the difficulties of an exact definition of this condition and the inexact reports found in the literature are mentioned. A study was also made of the calcium content and the symptoms of tetany in cases of congenital, coronary, posterior saucer-shaped and senile cataracts. In the author's opinion it is not possible to say in every case how the zonular cataract develops, as the causes are multiple, just as in the case of so-called rachitic teeth, a large proportion of which are due to hypocalcemia. This relation of hypocalcemia has been shown by the late examination of opacities of the lens in spasmophilic children and by symptoms of latent tetany in persons with zonular cataract. A large proportion of older patients with zonular cataract suffer from hypocalcemia.

The author concludes that the question of epithelial insufficiency must be further investigated, and this applies particularly to the other types of cataract mentioned. Of 27 cases of coronary cataract, there were 8 in which the calcium content was definitely deficient. A similar condition was found in the cases of posterior saucer-shaped cataract, so that exogenic causes cannot be excluded. Of 69 cases of senile cataract, a lowered calcium content was found in 16. It can be stated that in the cases of senile cataract the association with hypocalcemia is not proved. A complete bibliography accompanies this valuable monograph.

ARNOLD KNAPP

Introduction to Ophthalmology By Peter C. Kronfeld. Price, \$3.50. Pp. 331, with 32 figures and 5 plates. Springfield, Ill. Charles C. Thomas, Publisher, 1938.

In his preface the author states that it has been his purpose to "formulate the principles underlying that portion of Ophthalmology which is a necessary part of basic medical education. Details of diagnosis, of methods of examination, and of treatment have been omitted. Individual diseases are discussed to illustrate pathogenetic principles."

The individual chapters deal with anatomy, diseases of the anterior adnexa, diseases of the cornea, uveitis, endophthalmitis, the lens, injuries, the physiology of the retinal circulation, vascular diseases of the eye, the intraocular pressure and its variations, neoplasms, the optic nerve, the visual pathway, the pupil, motor anomalies of the eye and refraction. A brief ophthalmologic dictionary is combined with the index.

Three of the chapters seem to be particularly valuable, and they concern subjects which seem to be of special interest to the author.

The first, on the physiology of the retinal circulation, gives both briefly and concisely much pertinent information on the vascular pressures in the eye which is not easily obtainable. A table and a figure summarize the data.

The second chapter, on the vascular diseases of the eye, treats in a logical and satisfactory manner the various types of retinitis, particularly those associated with the different clinical types of nephritis. The author has summarized thousands of words when he concludes that "there seems to be rather strong evidence for the view that retinitis, irrespective of the character of the primary general disease, is the result of a disturbance of tissue metabolism by insufficient blood supply to the vessel wall and to the retina."

The third chapter, on intraocular pressure, covers the various theories of the production of aqueous and the mechanism of the maintenance of normal tension, it also outlines the several clinical types of glaucoma in a reasonable manner. Kronfeld concludes that there are probably a number of causes of glaucoma just as there are a number of clinical types.

There are a few dogmatic statements in the book to which I take exception and would modify as follows. The close spatial relation of the optic nerve and the posterior nasal sinuses does not explain the neural lesions attributed to disease of the sinuses. There is no evidence that papillitis is an inflammation (using the term as it is used in the field of general surgery), both papillitis and papilledema are probably pressure or edema phenomena. There is a great deal of evidence that retinitis pigmentosa is secondary to lesions of the choriocapillaris rather than a primary degeneration of the retina, nor is it always bilateral and hereditary. Most of the cases of retrobulbar neuritis are not due to an inflammatory lesion of the nerve, and, after reviewing a fairly long series of cases, it is apparent that many of the patients do not recover spontaneously. The cases in which normal vision is recovered are remembered, the others are forgotten. Likewise, tobacco and alcohol are not as a rule more injurious than tobacco alone in the production of toxic amblyopia. In fact, the recovery seems to be more rapid and more complete in the smokers who also use moderate amounts of alcohol than in those who do not. Also, in the type not associated with pellagra or polyneuritis, the primary pathologic process is apparently vascular.

As the author has developed certain phases of the subject in a complete and erudite fashion, it seems to me that to be consistent a consideration of certain other subjects might well have been included in a book of this type. Among these are Adie's syndrome, the irritative phase of Horner's syndrome, chronaxia, the implications of humoral physiology as applied to the eye, a fuller discussion of the fundamental physiology and pathology of allergy, the question of the relation of thromboangitis obliterans to retinal vascular disease, the ocular lesions which accompany certain cutaneous diseases and a discussion of that interesting group of diseases which includes sympathetic ophthalmia, the uveitis associated with alopecia, poliosis, dysacusia and vitiligo and Harada's disease (which may be a clinical variant of the last-mentioned disease).

In the main, the author has carried out his intentions of discussing ophthalmology in its relations to general medicine, and it was a distinct pleasure to read the book. Much of it, however, is an introduction to advanced ophthalmology, and as such, it is not suitable for medical students but can be recommended for ophthalmologists who make some effort to keep abreast of the current literature.

W F DUGGAN

A Child's Graded Reading Book for Eye Specialists. By Henry R Nesburn, M D, and Daniel L Risley, M A Price, \$15 Pp 23 Hollywood, Calif. Harpel-Bean Company, 1938

Oculists frequently are consulted concerning the school difficulties of a backward child or the preparation of a normal child for the task of learning to read and to enter school work. Visual recognition of picture forms, symbol (letter) forms and word forms enter (among other factors) into the situation, and the child's ability to recognize and respond to these forms should be tested.

The authors have prepared a series of graded plates involving visual recognition, interpretation and comprehension of material in each of these form classes and have bound them together into a useful little booklet. While it by no means furnishes a complete analysis of the child's visual functions and comprehension, it is simple and appears to be a careful and well arranged piece of work which has a definite and valuable place in such tests. It does not pretend to be more than it is and leaves the questions of refractive errors, muscle imbalances and fusional abilities where they belong.

It seems to me that the ages set for the recognition of symbol forms are too high—that these tests are usually passed by children at least a year younger. Also, if one were to suggest improvements, the jump from the recognition of picture forms to the recognition of symbol forms is too great. An intermediate step, such as the inclusion of a symbol E chart rating between pictures and letters, would add value.

The child's vocabulary today is interesting (or was it always so?), as among the first twenty words he learns (including six letter words) "no" ranks high, but "yes" is not included.

The booklet was worth preparing and is worth possessing and using.

LEGRAND H. HARDY

Proceedings of the All-India Ophthalmological Society. Volume 5 Session 1936 Pp 270 Madras, India Madras Publishing House, Ltd, 1937

Under the presidency of Dr B K Narayana Rao, the fifth conference of the All-India Ophthalmological Society was held on Dec 18-21, 1936, in the Patiala Block of the King Edward Medical College, Lahore. The society publishes the proceedings of the meeting as soon after the conference as possible.

The program was opened with a symposium on diabetes and ocular diseases. This was followed by papers on corneal grafting, tattooing of the cornea, various types of operations for cataract and on a number of clinical conditions. Of particular interest were a number of papers on glaucoma in connection with epidemic dropsy, particularly regarding the changes in the visual fields and the influence of dark adaptation. Communications on the treatment of detachment of the retina, on contact glasses, on tumors of the orbit, on the incidence of sympathetic ophthalmia in South India, on the surgical treatment of squint and on radium treatment for spring catarrh indicate the wide interest that is taken in India in clinical problems.

ARNOLD KNAPP

News and Notes

SOCIETY NEWS

Canadian Ophthalmological Society—The first meeting of the Canadian Ophthalmological Society was held at the Montreal Neurological Institute on August 24 and 25. Sir Stewart Duke-Elder presented an address on "Progress in Ophthalmology," which will be printed in the *Journal of the Canadian Medical Association*, and there was a symposium on visual standards in Canada, participated in by Dr G Stuart Ramsey, Dr E A McCusker, Dr W G Fraser, Dr F T Tooke, Dr J Vaillancourt and Dr F A Aylesworth.

Ophthalmological Society of Australia—The Ophthalmological Society of Australia (B M A) was organized on March 22, 1938. The officers are president, Sir James W Barrett, vice president, Dr A James Flynn, secretary, Dr Darcy A Williams, treasurer, Dr Leonard J C Mitchell.

The first meeting will be held at Melbourne, April 3 to 6, 1939.

Directory of Ophthalmologic Societies*

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS

President Dr P Baillhart, 66 Boulevard Saint-Michel, Paris, 6^e, France
Secretary-General Prof M Van Duyse, Université de Gand, Gand, Prov
Ostflandern, Belgium
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-
Michel, Paris, 6^e, France

INTERNATIONAL OPHTHALMOLOGIC CONGRESS

Secretary Dr E Marx, Costzeedijk 316, Rotterdam, Netherlands

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

President Dr A F MacCallan, 33 Welbeck St, London, W, England

FOREIGN

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President Dr Stewart Duke-Elder, 59 Harley St, London, W 1
Secretary Dr Thomasina Belt, 13 Mitchell Ave, Jesmond, Newcastle-on-Tyne

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEIPING

President Dr H T Pi, Peiping Union Medical College, Peiping
Secretary Dr C K Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping
Place Peiping Union Medical College, Peiping Time Last Friday of each
month

GERMAN OPHTHALMOLOGICAL SOCIETY

President Prof W Lohlein, Berlin
Secretary Prof E Engelking, Heidelberg

MIDLAND OPHTHALMOLOGICAL SOCIETY

President Dr W Niccol, 4 College Green, Gloucester, England
Secretary T Harrison Butler, 81 Edmund St, Birmingham, England
Place Birmingham and Midland Eye Hospital

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President Prof Dr Mohammed Mahfouz Bey, Government Hospital, Alexandria
Secretary Dr Mohammed Khalil, 4 Baehler St, Cairo
All correspondence should be addressed to the Secretary, Dr Mohammed Khalil

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President Mr Gordon M Holmes, 9 Wimpole St, London, W 1
Secretary Mr H B Stallard, 35 Harley St, London, W 1

OPHTHALMOLOGY SOCIETY OF BOMBAY

President Dr D D Sathaye, 127 Girgaum Road, Bombay 4, India
Secretary Dr H D Dastur, Dadar, Bombay 14, India
Place H B A Free Ophthalmic Hospital, Parel, Bombay 12 Time First
Friday of every month

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date

OXFORD OPHTHALMOLOGICAL CONGRESS

Master Dr Percival J Hay, 350 Glossup Rd, Sheffield 10, England
 Hon Secretary-Treasurer Dr F A Anderson, 12 St John's Hill, Shrewsbury,
 England
 Time July 1939

PALESTINE OPHTHALMOLOGICAL SOCIETY

President Dr Arieh Feigenbaum, Abyssinian Str 15, Jerusalem
 Secretary Dr E Sinai, Tel-Aviv

POLISH OPHTHALMOLOGICAL SOCIETY

President Dr W Kapuscinski, 2 Waly Batorego, Poznan
 Secretary Dr J Sobanski, Lindley'a 4, Warsaw
 Place Lindley'a 4, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President Dr Malcolm Hepburn, 111 Harley St, London, W 1, England
 Secretary Dr C Dee Shapland, 15 Devonshire Pl, London, W 1, England

SOCIÉTÉ FRANÇAISE D'OPHTHALMOLOGIE

Secretary Dr Rene Onfray, 6 Avenue de la Motte Picquet, Paris, 7^e

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President Prof K G Ploman, Stockholm
 Secretary Dr K O Granstrom, Sodermalmstorg 4 III tr, Stockholm, So

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President Dr D Arieh-Friedman, 96 Allenby Str, Tel-Aviv, Palestine
 Secretary Dr Sadger Max, 9 Bialik Str, Tel-Aviv, Palestine

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman Dr Eugene Chan, Cheeloo University School of Medicine, Tsinan,
 Shantung, China
 Place Cheeloo University School of Medicine Time Last Thursday of alter-
 nate months

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON
OPHTHALMOLOGY

Chairman Dr S Judd Beach, 704 Congress St, Portland, Maine
 Secretary Dr Derrick T Vail Jr, 441 Vine St, Cincinnati
 Place St Louis Time May 15-19, 1939

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY,
SECTION ON OPHTHALMOLOGY

President Dr Harry S Gradle, 58 E Washington St, Chicago
 Executive Secretary-Treasurer Dr William P Wherry, 1500 Medical Arts
 Bldg, Omaha
 Place Washington, D C Time Oct 9-14, 1938

AMERICAN OPHTHALMOLOGICAL SOCIETY

President Dr Frederick Tooke, 1482 Mountain St, Montreal, Canada
 Secretary-Treasurer Dr Eugene M Blake, 303 Whitney Ave, New Haven, Conn
 Place Hot Springs, Va

CANADIAN OPHTHALMOLOGICAL SOCIETY

President Dr W Gordon M Byers, 1458 Mountain St, Montreal
 Secretary-Treasurer Dr Alexander E MacDonald, 421 Medical Arts Bldg,
 Toronto

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President Mr William Fellowes Morgan, 50 W 50th St., New York
 Secretary Miss Regina E Schneider, 50 W 50th St, New York

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr L C Gardner, 11 N Main St, Fond du Lac
 Secretary Dr G L McCormick, 626 S Central Ave, Marshfield
 Place Rochester, Minn Time Nov 11, 1938

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President Dr Edwin B Goodall, 101 Bay State Rd, Boston
 Secretary-Treasurer Dr Trygve Gundersen, 243 Charles St, Boston
 Place Massachusetts Eye and Ear Infirmary, 243 Charles St, Boston Time
 8 p m, third Tuesday of each month from November to April, inclusive

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President Dr F C Cordes, 384 Post St, San Francisco
 Secretary-Treasurer Dr C Allen Dickey, 450 Sutter St, San Francisco
 Place San Francisco Time June 19-22, 1939

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr H L Goss, Cobb Bldg, Seattle, Wash
 Secretary-Treasurer Dr Purman Dorman, 1115 Terry Ave, Seattle, Wash
 Place Seattle or Tacoma, Wash Time Third Tuesday of each month, except
 June, July and August

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President Dr L J Friend, 425 E Grand Ave, Beloit, Wis
 Secretary-Treasurer Dr Thorsten E Blomberg, 501-7th St, Rockford, Ill
 Place Rockford, Ill, or Janesville or Beloit, Wis Time Third Tuesday of
 each month from October to April, inclusive

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Don M Howell, Alma, Mich
 Secretary-Treasurer Dr Louis D Gomon, 308 Eddy Bldg, Saginaw, Mich
 Place Saginaw or Bay City, Mich Time Second Tuesday of each month,
 except July and August

SIoux VALLEY EYE AND EAR ACADEMY

President Dr R A Kelly, 304 N Main St, Mitchell, S D
 Secretary-Treasurer Dr J C Decker, 515 Frances Bldg, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman Dr Grady E Clay, Medical Arts Bldg, Atlanta, Ga
 Secretary Dr John R Hume, 921 Canal St, New Orleans

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President Dr John Hunter McRae, 26 Sheldon Ave, S E, Grand Rapids
 Secretary-Treasurer Dr Dewey R Heetderks, 405 Medical Arts Bldg, Grand
 Rapids
 Time Third Thursday of alternate months

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr C M Harris, Johnstown
 Secretary-Treasurer Dr C Wearne Beals, 41 N Brady St, DuBois

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President A presiding officer is selected for each meeting alternately until all members have served

Secretary Dr John C Long, 324 Metropolitan Bldg, Denver
Place Capitol Life Bldg, Denver Time 7 30 p m, third Saturday of each month, October to May, inclusive

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR,
NOSE AND THROAT

President Dr William M Good, 63 Center St, Waterbury, Conn
Secretary-Treasurer Dr S J Silverberg, 201 Park St, New Haven

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President Dr Grady E Clay, 384 Peachtree St, N E, Atlanta
Secretary-Treasurer Dr J Mason Baird, 511 Medical Arts Bldg, Atlanta

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr C W Rutherford, 23 E Ohio St, Indianapolis
Secretary Dr Marlow W Manion, 23 E Ohio St, Indianapolis
Place Indianapolis Time First Wednesday in April

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr H H Lamb, American Bank Bldg, Davenport
Secretary-Treasurer Dr B M Merkel, 604 Locust St, Des Moines
Place Davenport

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr Francis E Le Jeune, 632 Maison Blanche Bldg, New Orleans
Secretary-Treasurer Dr Edley H Jones, 1301 Washington St, Vicksburg, Miss
Place Gulfport, Miss Time May 8, 1939

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman Dr F N Smith, Grand Rapids Clinic, Grand Rapids
Secretary Dr Dewey R Heetderks, 26 Sheldon Ave, S E, Grand Rapids

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Frank N Knapp, 318 W Superior St, Duluth
Secretary-Treasurer Dr George E McGeary, 920 Medical Arts Bldg, Minneapolis
Time Second Friday of each month from October to May

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President Dr Roy Grigg, Bozeman
Secretary Dr A W Morse, 507 Phoenix Bldg, Butte

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY,
OTOLOGY AND RHINOLARYNGOLOGY

Chairman Dr Norman W Burritt, 30 Beechwood Rd, Summit
Secretary Dr A Russell Sherman, 671 Broad St, Newark
Place Atlantic City Time June 1939

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman Dr Algernon B Reese, 73 E 71st St, New York
Secretary Dr Chester C Cott, 333 Linwood Ave, Buffalo

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr J M Lilly, 302 Old St, Fayetteville
 Secretary-Treasurer Dr Frank C Smith, 106 W 7th St, Charlotte
 Place Charlotte Time October

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr N A Youngs, 322 De Mers Ave, Grand Forks
 Secretary-Treasurer Dr F L Wicks, 516-6th St, Valley City
 Place Fargo Time May 1939

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr L O Clement, 406 State St, Salem
 Secretary-Treasurer Dr Paul Bailey, 833 S W 11th Ave, Portland
 Place Good Samaritan Hospital, Portland Time Thrd Tuesday of each month

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President Dr N Darrell Harvey, 112 Waterman St, Providence
 Secretary-Treasurer Dr Linley C Happ, 124 Waterman St, Providence
 Place Rhode Island Medical Society Library, Providence Time 8 30 p m,
 second Thursday in October, December, February and April

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr S B Fishburne, 1430 Marion St, Columbia
 Secretary Dr J W Jervey Jr, 101 Church St, Greenville
 Place Columbia Time Nov 1, 1938

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Kate Savage Zerfoss, 165-8th Ave, N, Nashville
 Secretary-Treasurer Dr W D Stinson, 805 Medical Arts Bldg, Memphis

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President Dr T E Fuller, 100 W Board St, Texarkana, Texas
 Secretary Dr O M Marchman, 1719 Pacific Ave, Dallas

UTAH OPHTHALMOLOGICAL SOCIETY

President Dr V P White, 143½ S Main St, Salt Lake City
 Secretary-Treasurer Dr E B Fairbanks, Boston Bldg, Salt Lake City
 Time Thrd Monday of each month

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President Dr Charles T St Clair, 418 Bland St, Bluefield, W Va
 Secretary-Treasurer Dr M H Williams, 30½ Franklin Rd, S W, Roanoke

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
 AND THROAT SECTION

President Dr George Traugh, 309 Cleveland Ave, Fairmont
 Secretary Dr Welch England, 621½ Market St, Parkersburg

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON
 EYE, EAR, NOSE AND THROAT

President Dr Andrew Rados, 31 Lincoln Park, Newark
 Secretary Dr William F McKim, 317 Roseville Ave, Newark
 Place 91 Lincoln Park South, Newark Time 8 45 p m, second Monday of
 each month, October to May

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr L E Brown, Second National Bldg, Akron, Ohio
 Secretary-Treasurer Dr C R Anderson, 106 S Main St, Akron, Ohio
 Time First Monday in January, March, May and November

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr J Mason Baird, Medical Arts Bldg, Atlanta, Ga
 Secretary Dr Alton V Hallum, 478 Peachtree St, Atlanta, Ga
 Place Academy of Medicine, 38 Prescott St Time Second Friday of each month from October to May

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman Dr Frank B Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore
 Secretary Dr Fred M Reese, 6 E Eager St, Baltimore
 Place Medical and Chirurgical Faculty, 1211 Cathedral St Time 8 30 p m, fourth Thursday of each month from October to May

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President Dr E Clifford Place, 59 Livingston St, Brooklyn
 Secretary-Treasurer Dr Frank Mallon, 1135 Park Pl, Brooklyn
 Place Kings County Medical Society Bldg, 1313 Bedford Ave Time Third Thursday in February, April, May, October and December

BUFFALO OPHTHALMOLOGIC CLUB

President Dr Ivan J Koenig, 40 North St, Buffalo
 Secretary-Treasurer Dr Meyer H Riwchun, 367 Linwood Ave, Buffalo
 Time Second Thursday of each month

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Each member, in alphabetical order
 Secretary Dr A H Benz, 706 Medical Arts Bldg, Chattanooga, Tenn
 Place Mountain City Club Time Second Thursday of each month from September to May

CHICAGO OPHTHALMOLOGICAL SOCIETY

President Dr Georgiana Dvorak-Theobald, 715 Lake St, Oak Park, Ill
 Secretary-Treasurer Dr Earle B Fowler, 55 E Washington St, Chicago
 Place Medinah Michigan Avenue Club, 505 N Michigan Ave Time Third Monday of each month from October to May

CINCINNATI OPHTHALMIC CLUB

Chairman Each member, in rotation
 Secretary-Treasurer Dr E R Thomas, 819 Carew Tower, Cincinnati
 Place Holmes Memorial Library, Cincinnati General Hospital Time 8 15 p m, third Monday of each month except June, July and August

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman Dr Paul Moore, Republic Bldg, Cleveland
 Secretary Dr G Leslie Miller, 14805 Detroit Ave, Cleveland
 Time Second Tuesday in October, December, February and April

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman Dr Alexander G Fewell, 1924 Pine St, Philadelphia
 Clerk Dr W S Reese, 1901 Walnut St, Philadelphia
 Time Third Thursday of every month from October to April, inclusive

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman Dr Hugh G Beatty, 150 E Broad St, Columbus, Ohio
 Secretary-Treasurer Dr W A Stoutenborough, 21 E State St, Columbus, Ohio
 Place Deshler Wallick Hotel Time 6 p m, first Monday of each month

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman Dr Edgar G Mathis, 416 Chaparral St, Corpus Christi, Texas
 Secretary Dr E King Gill, 416 Chaparral St, Corpus Christi, Texas
 Time Second Thursday of each month from October to May

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr Lester H Quinn, 4105 Live Oak, Dallas, Texas
 Secretary Dr J Dudley Singleton, 1719 Pacific Ave, Dallas, Texas
 Place Dallas Athletic Club Time 6 30 p m, first Tuesday of each month
 from October to June The November, January and March meetings are
 devoted to clinical work

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr E G Linn, 604 Locust St, Des Moines, Iowa
 Secretary-Treasurer Dr Grace Doane, 614 Bankers Trust Bldg, Des Moines,
 Iowa
 Time 7 45 p m, third Monday of every month from September to May

DETROIT OPHTHALMOLOGICAL CLUB

Chairman Members rotate alphabetically
 Secretary Dr William Fowler, 1066 Maccabee Bldg, Detroit
 Time 6 30 p m, first Wednesday of each month

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President Dr L A Hulsebosch, 191 Glen St, Glen Falls
 Secretary-Treasurer Dr Joseph L Holohan, 330 State St, Albany
 Time Third Wednesday in October, November, March, April, May and June

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President Dr R A Gough, 602 W 10th St, Fort Worth, Texas
 Secretary-Treasurer Dr Charles R Lees, 806 Medical Arts Bldg, Fort Worth,
 Texas
 Place Medical Hall, Medical Arts Bldg Time 7 30 p m, first Friday of each
 month except July and August

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President Dr Dewey R Heetderks, 405 Medical Arts Bldg, Grand Rapids, Mich
 Secretary-Treasurer Dr Robert G Laird, 116 E Fulton St, Grand Rapids, Mich
 Place Various local hospitals Time Third Thursday of alternating months,
 September to May

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND
 THROAT SECTION

President Dr Louis Daily, 1215 Walker Ave, Houston, Texas
 Secretary Dr Herbert H Harris, 1004 Medical Arts Bldg, Houston, Texas
 Place Medical Arts Bldg, Harris County Medical Society Rooms Time
 8 p m, second Thursday of each month from September to June

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 Secretary Dr John S Knight, 1103 Grand Ave, Kansas City, Mo
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 January and March meetings are devoted to clinical work

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 Place Van Cleve Hotel Time 6 30 p m, monthly, first Tuesday from October
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 Time Second Thursday of October, December, February and April

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 Place Omaha Club, 20th and Douglas Sts, Omaha Time 6 p m, dinner,
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 Secretary Dr George H Shuman, 351-5th Ave, Pittsburgh
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 month, except June, July, August and September

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 Secretary Dr O M Rott, 421 Riverside Ave, Spokane, Wash
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 D C
 Secretary-Treasurer Dr Frank D Costenbader, 1726 I St, Washington, D C
 Place Episcopal Eye, Ear and Throat Hospital Time 8 p m, first Monday
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ARCHIVES OF OPHTHALMOLOGY

VOLUME 20 (old series Volume 77)

NOVEMBER 1938

NUMBER 5

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IMPLANTATION OF HOLLOW GROOVED BODY INTO ORBIT FOR FILLING

LATE AFTER ENUCLEATION OF EYEBALL

JOHN M. WHEELER, M.D. †

NEW YORK

Many authors have described or advocated implantation of various substances into Tenon's capsule immediately after removal of the eyeball. This paper is not concerned with these procedures but rather with the introduction of a body behind Tenon's capsule late after healing from enucleation has taken place. A sharp distinction should be made between putting filling into Tenon's capsule while it is open and putting filling behind the organized mat of Tenon's capsule that results from collapse of the walls of the cavity and healing of the flaps of the capsule that come in apposition behind the conjunctival covering of the fundus of the socket.

Patients object to an appearance of sinking of an artificial eye and particularly to sinking below the upper orbital margin above the artificial eye. Along with the sinking there is, in some degree, smoothing of the fold of the upper lid. For such a deformity implantation should not be made into the upper part of the orbit but into the muscle cone behind the fundus of the socket. This tends to correct the backward position of the prosthesis and to carry to their proper place the structures that belong in the upper part of the orbit. Such an operation is particularly indicated in cases of traumatic injury with defects in the floor of the orbit, with letting down and loss of orbital contents. In some cases filling along the floor as well as in the muscle cone may be needed. For implantation along the floor, fascia lata is used. For filling inside the muscle cone, different substances have been used, among them vital tissues, such as fascia, cartilage, bone and fat. There seems to be no advantage in the use of vital tissue, as a properly placed nonvital body can stay in place indefinitely and hold its own against contraction. If the contents of the orbit are not badly injured, fat may

† Dr. Wheeler died Aug. 22, 1938.

Read before the Section of Ophthalmology of the New York Academy of Medicine, April 18, 1938.

be satisfactory, but if there is considerable scar tissue in the orbit, a fat graft is liable to contract. Moreover, in several cases proliferation has taken place in a fat graft, with the formation of lipoma.

Smooth spheres of gold or glass have been used, and they could be recommended except that after a period of weeks, months or years they are liable to migrate from the muscle cone, taking with them their capsules of newly formed tissue with which they have become surrounded. Some years ago, conscious of the frequent misbehavior of smooth spheres in the muscle cone, I implanted cork. The implant was cut in the form of a sphere, and then four grooves were cut in the sphere, each groove to receive one of the four rectus muscles. These grooved spheres stayed in place well, but a peculiar phenomenon made their use inadvisable. Long after their implantation the tissues in front of them sloughed, and they appeared in the cul-de-sac. Why this hap-



Fig 1—Hollow glass body for implantation behind Tenon's capsule late after enucleation of the eyeball. Notice the four grooves for the reception of the four rectus muscles.

pened I do not know, but I suppose that some chemical change took place with a degree of disintegration of the cork and necrosis of the tissue about the cork. The success that I met with, as far as the staying in place of the grooved spheres of cork was concerned, led me to the use of a smooth body made of glass. A glass blower made the four grooves to receive the four rectus muscles, and implantation of this body has been rewarded by permanent good results. Roentgenograms have demonstrated that the axis of the grooved sphere corresponds with the axis of the orbit, so probably the rectus muscles stay permanently in the grooves. So far, none of these bodies has given trouble late after implantation. Figure 1 shows the smooth glass body with the four grooves made to receive the rectus muscles.

OPERATIVE TECHNIC

Block anesthesia of the orbit is not fully effective. Infiltration of the orbit with procaine hydrochloride is permissible, although it entails the disadvantage of partially filling the orbit which is to receive the sphere and so putting Tenon's capsule on the stretch at the conclusion of the operation. Avertin or aveitin and ether inhalation are satisfactory.

An ordinary speculum is introduced to expose the fundus of the socket. With a knife or scissors, a horizontal incision is made through the conjunctiva completely across the fundus behind the palpebral fissure (fig 2 *A*). The conjunctiva is dissected from Tenon's capsule freely with scissors and with the least possible

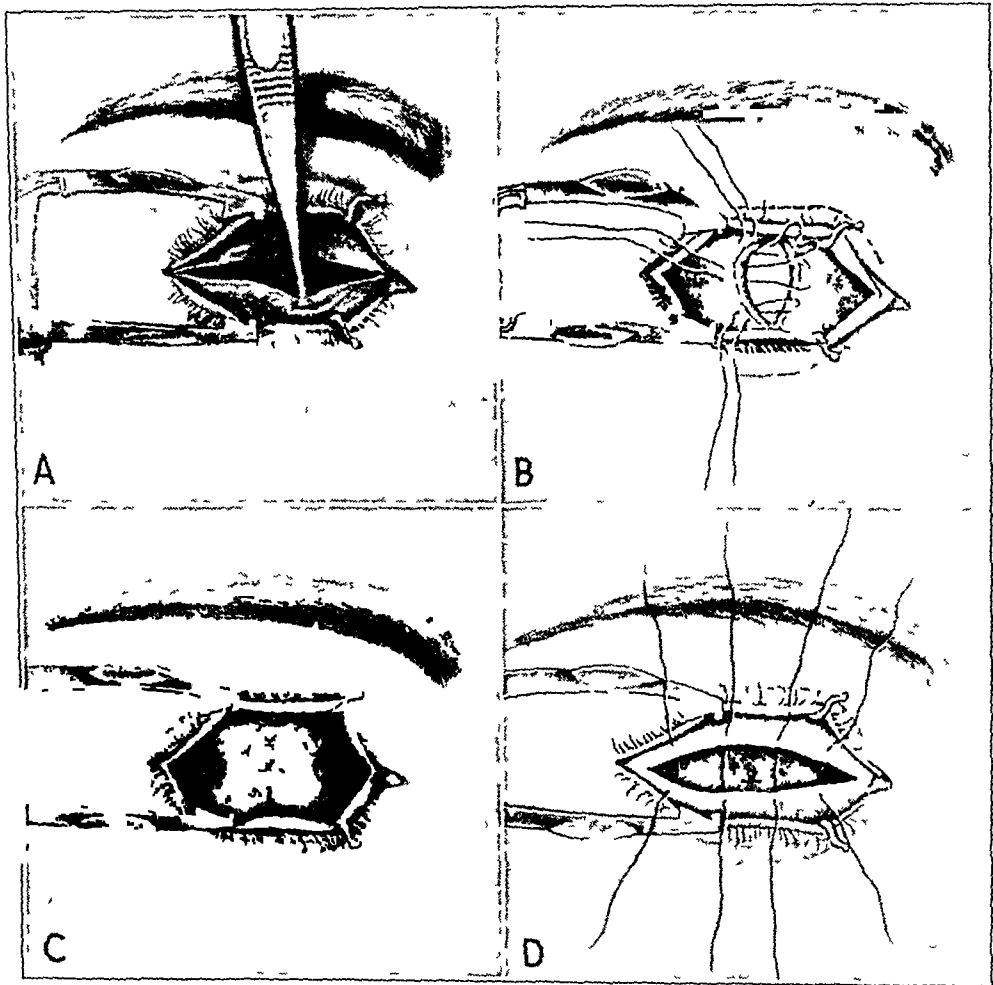


Fig 2—Steps in the operative procedure. In *A*, with the speculum in place, a horizontal incision is made in the conjunctiva completely across the fundus of the socket. In *B* the conjunctiva has been dissected from the underlying Tenon's capsule, and the conjunctival flaps have been placed in the blades of the speculum with the eyelids. A vertical incision has been made through Tenon's capsule deep into the orbital tissue. A hollow grooved body has been placed in the orbit, and sutures have been placed to cause overlapping of the flaps of Tenon's capsule. In *C* the overlapping flaps of the capsule are secured by 000 chromic catgut sutures. In *D* the conjunctiva is released from the blades of the speculum and the conjunctiva is closed by fine silk or catgut sutures.

traumatism Then the conjunctiva is taken in the blades of the speculum along with the lids A vertical incision is made in Tenon's capsule, and the dissection is carried into the orbital tissue within the muscle cone As the orbital dissection is carried backward, it should take the direction of the axis of the orbit and pass nasalward toward the apex, otherwise the external rectus muscle is liable to be cut The orbital incision should go to within about 1 cm of the orbital apex Usually the ophthalmic artery need not be cut, but occasionally it is injured, and it may have to be clamped The mat of Tenon's capsule is dissected up a few millimeters on each side of the vertical incision If the orbital dissection is adequate, the grooved sphere will slip easily into the newly formed cavity within the muscle cone The direction of the grooves in the implant and their position should be such that the four rectus muscles will fit into them The axis of the implant then corresponds approximately with that of the orbit It should be pressed well back into the orbit Mattress sutures of fine chromic catgut are placed in Tenon's capsule, so that there will be an overlapping of the flaps of the capsule of 4 or 5 mm Figures 2B and C illustrate the manner in which the flaps are brought squarely into an overlapping position For this suturing, atraumatic needles are of advantage, as the needles and suture material slip through almost without resistance Next, the conjunctival flaps are raised from the blades of the speculum, and the horizontal conjunctival wound is closed by fine silk or catgut sutures, as shown in figure 2D A firm pressure dressing should be applied at the conclusion of the operation and left in place for about one week The pressure will hold the flaps accurately in place and will prevent hemorrhage into the orbit The conjunctival sutures may be removed when the pressure dressing is taken off, and a light dressing may be worn for a few days The socket is ready for a prosthesis three weeks after operation

DISCUSSION

DR CLYDE E McDANNALD What is the approximate size of these implants, and what are the possibilities of erosion of a glass implant? I bring up this question of erosion because when I was an intern at the New York Eye and Ear Infirmary a man appeared one day with severe pain in his eye and stated that he had undergone a Mule's operation thirteen years previously in London He had taken out his handkerchief, and wiped his eye, and since then he had had severe pain I referred him to one of the surgeons, who found that the ball had completely eroded and broken into innumerable fine particles I wonder if there is a possibility of this taking place with the implant described here

DR JOHN M WHEELER I do not know I have been doing the operation that I am advocating for a little over three years, and that is too short a time to judge on the particular point of possible erosion I remember the case Dr McDannald spoke about, it is the only one of its kind of which I have heard The implantation was made into the sclera, but implantations of glass by the hundreds were done on American soldiers during the World War As far as I know none of the implants has disintegrated I think that glass or gold placed in the orbit behind Tenon's capsule will stay indefinitely At least, I hope so

The grooved glass implants have been made by Mager and Gougelman in different sizes, but I think that the implant of preference is one with an anteroposterior diameter of 18 mm

DETERMINATION AND SIGNIFICANCE OF THE SCOTOPIC RETINAL VISIBILITY CURVE

ELEK LUDVIGH, PH D

BOSTON

For the past half century the relation between visual purple and vision has been realized to be of fundamental importance. Many investigators¹ have compared the absorption spectrum of visual purple with the scotopic visibility curve. Some of the more recent comparisons have been made by Hecht and Williams,² Dartnall and Goodeve³ and Blum.⁴ The comparisons made by Hecht and Williams and by Blum are inadequate because the absorption spectrum of visual purple was compared with the scotopic ocular visibility curve, which means that no corrections were made for the selective absorption of light by the refractive media of the human eye. These comparisons are also inadequate because the absorption spectrum of visual purple used was that determined by Koettgen and Abelsdorff,^{1c} whereas the more recent work of Lythgoe,⁵ of Krause and Sidwell⁶ and of Bayliss, Lythgoe and Tansley⁷ has shown that the absorption of light of short wavelengths by visual purple is much greater than was formerly supposed.

From the Howe Laboratory of Ophthalmology, Harvard University, and the Massachusetts Eye and Ear Infirmary

1 (a) Trendelenburg, W. Quantitative Untersuchungen über die Bleichung des Sehpurpurs in monochromatischem Licht, *Ztschr f Psychol u Physiol d Sinnesorg* **37** 1, 1905. (b) Koenig, A. Ueber den menschlichen Sehpurpur und seine Bedeutung für das Sehen, *Sitzungsber d k Akad d Wissensch*, 1894, p 577. (c) Koettgen, E., and Abelsdorff, G. Absorption und Zersetzung des Sehpurpurs bei den Wirbeltieren, *Ztschr f Psychol u Physiol d Sinnesorg* **12** 161, 1896. (d) Hecht, S. The Visibility of the Spectrum, *J Optic Soc America* **9** 211, 1924. (e) Venable, W. M. The Absorption Spectrum of Visual Purple, *ibid* **10** 133, 1925.

2 Hecht, S., and Williams, R. E. The Visibility of Monochromatic Radiation and the Absorption Spectrum of Visual Purple, *J Gen Physiol* **5** 1, 1922.

3 Dartnall, H. J. A., and Goodeve, C. F. Scotopic Luminosity Curve and the Absorption Spectrum of Visual Purple, *Nature*, London **139** 409, 1937.

4 Blum, H. F. Visual Purple and Rod Vision, *Science* **87** 193, 1938.

5 Lythgoe, R. J. The Absorption Spectra of Visual Purple and of Indicator Yellow, *J Physiol* **89** 331, 1937.

6 Krause, A. C., and Sidwell, A. E. The Absorption Spectra of Visual Purple and Its Photodecomposition Products, *Am J Physiol* **121** 215, 1938.

7 Bayliss, L. E., Lythgoe, R. J., and Tansley, K. Some New Forms of Visual Purple Found in Sea Fishes, with a Note on the Visual Cells of Origin, *Proc Roy Soc, London, s B* **120** 95, 1936.

The comparison made by Dartnall and Goodeve,³ although they used modern data on the absorption spectrum of visual purple, is inadequate because again the comparison was made with the scotopic ocular visibility curve. It is essential that the modern data on the absorption spectrum of visual purple be compared with those on the scotopic retinal visibility curve. In order to determine the scotopic retinal visibility curve, it is necessary to transform the scotopic ocular visibility curve by allowing for the selective absorption of light by the refractive media of the human eye. This transformation has hitherto been impossible because of lack of the necessary data. The only available quantitative data relating to this subject are those of Roggenbau and Wetthauer⁸ on the bovine eye. Unfortunately, one cannot assume that the absorption spectrum of the bovine eye is the same as that of the human eye. Among many other differences, the bovine eye is larger and younger than the adult human eye. In fact, that the limits of transmission of the ocular media in the violet and ultraviolet portions of the spectrum vary considerably with the genus of animal has been shown by Shoji.⁹ Recently, in collaboration with E. F. McCarthy I determined the absorption spectrums of the refractive media of each of several optically normal human eyes. A description of the apparatus and of the method used in this investigation and also of the results has appeared in an article in the ARCHIVES.¹⁰ The purpose of the present paper is to present a comparison between the absorption spectrum of rhodopsin and the scotopic retinal visibility curve and to discuss the significance of the agreement found.

The comparison made by Hecht and Williams² is shown in chart 1. The data of Koettgen and Abelsdorff^{1c} at 420, 440, 640 and 660 millimicrons were discarded by Hecht and Williams on the grounds that they had been inadequately determined. The discrepancy between the positions of the two curves on the wavelength axis was explained by Hecht and Williams in terms of Kundt's rule, according to which the absorption spectrum of a substance in solution moves toward the long wave end of the spectrum as the refractive index of the solvent increases. They pointed out that the absorption spectrum of visual purple is obtained while the visual purple is in dilute bile salts solutions. They assumed that the visual purple in the living retina is in

8 Roggenbau, C., and Wetthauer, A. (a) Zur Frage der Erwarmbarkeit der einzelnen lichtbrechenden Teile des Auges nach Bestrahlung durch einen leuchtenden Korper, *Ztschr. f. Augenh.* **64** 143, 1928, (b) Ueber die Durchlassigkeit der brechenden Augenmedien fur langwelliges Licht nach Untersuchungen am Rindsaue, *Klin. Monatsbl. f. Augenh.* **79** 458, 1927.

9 Shoji, Y. Untersuchung uber die Absorption der ultravioletten Strahlen durch die Augenmedien, *Mitt. a. d. med. Fakult. d. Univ. zu Tokyo* **29** 61, 1922.

10 Ludvigh, E., and McCarthy, E. F. Absorption of Visible Light by the Refractive Media of the Human Eye, *Arch. Ophth.* **20** 37 (July) 1938.

some sense in solution in the rods, saying that "The terminal segments of the rods are, from all observations and descriptions, fairly dense and highly refractive bodies

It is in these that the visual purple is dissolved in the living retina" They suggested "The difference in the positions of the two curves is explainable in terms of Kundt's rule This would assume that the visual purple we can extract from the eye and whose maximum of absorption in bile salts solution is at $503\text{ }\mu\mu$, is present in solution in the rods where its absorption maximum is at $511\text{ }\mu\mu$, and in very dilute solution in the cones where its maximum is at $554\text{ }\mu\mu$ (or more probably, as corrected for macular transmission, at $540\text{ }\mu\mu$)" This explanation of the difference between the two curves in chart 1 seems inadequate It has been pointed out

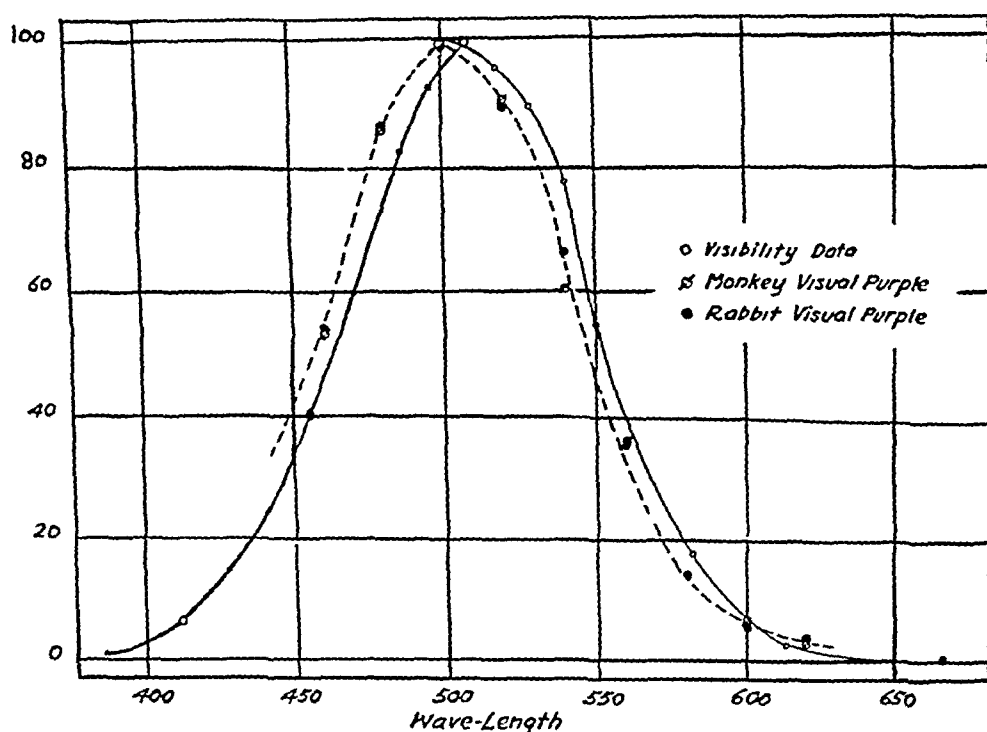


Chart 1—The solid line is the scotopic ocular visibility curve in terms of energies (Hecht and Williams), the dash line, the absorption spectrum of visual purple (Koettgen and Abelsdorff)

by Dartnall and Goodeve³ and also by Blum⁴ that Kundt's rule is unreliable Krause¹¹ has noted that according to Kuhne dried visual purple is fairly light-stable This lends weight to his suggestion that the reaction of visual purple to light may involve water and be bimolecular, a bimolecular reaction occurring in a large excess of water, giving the appearance of a unimolecular reaction Tansley¹² has

11 Krause, A C The Biochemistry of the Eye, Baltimore, Johns Hopkins Press, 1934, p 76

12 Tansley, K The Regeneration of Visual Purple Its Relation to Dark Adaptation and Night Blindness, J Physiol 71 442, 1931

pointed out that the results of her experiments on the amount of visual purple present in the retinas of dark-adapted rats may be expressed by the equation for a unimolecular reaction. This evidence and also the high water content of the retina suggest strongly that even though rhodopsin may be in the terminal segments of the rods, it may still be in weak aqueous solution. In this case Kundt's rule could not validly be used to explain the discrepancy between the two curves even if it were known to apply to solutions of visual purple.

As a matter of fact, however, Kundt's rule would have to accomplish a great deal more than correct the difference between the two curves in chart 1. In chart 2 the data of Hecht and Williams on the scotopic ocular visibility curve and the modern data of Lythgoe⁵ on

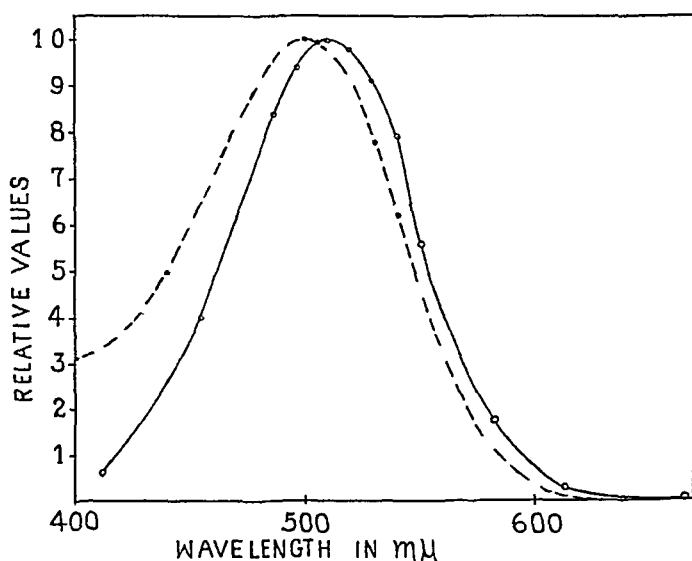


Chart 2—The solid line is the scotopic ocular visibility curve in terms of energies (Hecht and Williams), the dash line, the absorption spectrum of visual purple (Lythgoe)

the absorption spectrum of visual purple are shown for comparison. Lythgoe's values for p_H 7.0 were used. According to Nakashima,¹³ the p_H of the dark-adapted retina is about 7.3. Lythgoe's values for p_H 7.0 and p_H 7.6 are so nearly the same, however, that interpolation did not seem warranted. It may be noted in passing that the data of Krause and Sidwell⁶ on bovine visual purple are in essential agreement with those of Lythgoe⁵ on the visual purple of the frog. It is seen in chart 2 that not only is there a considerable discrepancy between the maxima of the two curves but the curve representing the absorption spectrum of rhodopsin is much higher in the blue end of the spectrum.

¹³ Nakashima, M. Beitrage zur Kenntnis des Sehpurpurs, abstracted, Zentralbl. f. d. ges. Ophth. **22** 772, 1930.

than that representing the scotopic ocular visibility curve. Even if Kundt's rule were operative and shifted the dash line curve about 9 millimicrons to the right so that the maxima of the two curves coincided, a great discrepancy in the blue end of the spectrum would still remain.

The reasoning which causes one to expect agreement between the absorption spectrum of rhodopsin and the scotopic retinal visibility curve is as follows. It is believed that visual purple is the light-sensitive substance involved in rod or scotopic vision. According to the law of photochemistry enunciated by von Grotthus¹⁴ and Draper,¹⁵ only that light which is absorbed can initiate a chemical change. Hence, if visual purple is the photosensitive substance for scotopic vision, the sensitivity of the rods to light of different wavelengths must be dependent on the extent to which light of various wavelengths is absorbed by rhodopsin. Furthermore, Einstein's¹⁶ law of photochemical equivalence, or the law of Stark¹⁷ and Einstein, states essentially that the number of molecules of the absorbing substance which decompose is equal to the number of quanta absorbed. The question as to whether or not this law may be applied to other than the first stage of the photochemical reaction need not be of concern here, particularly in view of the fact that Dartnall, Goodeve and Lythgoe¹⁸ appear to have demonstrated that the quantum efficiency of the reaction is probably 1. The scotopic ocular visibility curve is determined by finding for different wavelengths the amount of energy to which the eye must be subjected in order to produce a given sensation of brightness. The sensation of brightness in question may be that of threshold brightness or of some brightness above the threshold. It may now be assumed that equal brightness means that an equal number of quanta has been absorbed by the visual purple no matter what the wavelength of the stimulating light.

14 von Grotthus, T. Abhandlungen über Elektrizität und Licht, in Ostwald, F. W. *Klassiker der exakten Wissenschaften*, Leipzig, Wilhelm Engelmann, 1906, no. 152, p. 101.

15 Draper, J. W. On Some Analogies Between the Phenomena of the Clinical Rays and Those of Radiant Heat, *Phil. Mag.* **19** 195, 1841, On the Allotropism of Chlorine as Connected with the Theory of Substitutions, *ibid.* **27** 327, 1845.

16 Einstein, A. Ueber einen die Erzeugung und Verwandlung des Lichtes betreffenden heuristischen Gesichtspunkt, *Ann. d. Phys.* **17** 132, 1905, Thermodynamische Begründung des photochemischen Äquivalentgesetzes, *ibid.* **37** 832, 1912, Nachtrag zu meiner Arbeit "Thermodynamische Begründung des photochemischen Äquivalentgesetzes," *ibid.* **38** 881, 1912.

17 Stark, J. Weitere Bemerkungen über die thermische und chemische Absorption im Bandenspektrum, *Physikal. Ztschr.* **9** 889, 1908, Ueber die zerstaubende Wirkung des Lichtes und die optische Sensibilisation, *ibid.* **9** 894, 1908.

18 Dartnall, H. J. K., Goodeve, C. F., and Lythgoe, R. J. The Effect of Temperature on the Photochemical Bleaching of Visual Purple Solutions, *Proc. Roy. Soc., London*, s. B. **136** 35, 1937.

If I_o is the incident energy, I_a the energy absorbed by the visual purple, a the absorption coefficient of visual purple and c the concentration of visual purple, then Beer's law may be expressed as follows

$$(1) I_a = I_o (1 - e^{-ac})$$

One can express e^{-ac} by expanding it into the series

$$(2) e^{-ac} = 1 - ac + \frac{a^2 c^2}{2!} - \dots$$

With small values of c , the terms with powers higher than the first may be neglected. Substituting equation 2 in equation 1, one gets

$$(3) I_a = I_o ac \text{ or } a = \frac{I_a}{I_o c}$$

Now the number of incident quanta, n_o , is proportional to the incident energy for any given wavelength. That is,

$$(4) n_o = k I_o$$

Also, the number of absorbed quanta, n_a , is proportional to the absorbed energy, and since the absorbed energy is of the same wavelength as the incident energy, the constant of proportionality remains k , so that

$$(5) n_a = k I_a$$

Dividing equation 5 by equation 4 gives

$$(6) \frac{I_a}{I_o} = \frac{n_a}{n_o},$$

and substituting this in equation 3 gives

$$(7) a = \frac{n_a}{n_o c}$$

But, as has been seen, in the experimental conditions under which the visibility curve is determined, the value of n_a at every wavelength is the same, since sensations of equal brightness are produced at every wavelength. Also, the concentration, c , is the same at every wavelength, since the state of adaptation is constant. Expressing $\frac{n_a}{c}$ as a new constant, k' , gives

$$(8) a = \frac{k'}{n_o},$$

which states that the absorption coefficient is inversely proportional to the number of incident quanta necessary at each wavelength to produce a given sensation of brightness. Now the usual visibility curve plots the reciprocal of the amount of energy necessary to produce a given sensation of brightness at each wavelength, i.e., visibility $= V = 1/I_o$, but since the value of I_o and the wavelength are known, visibility may equally well be expressed as the reciprocal of the number of quanta necessary to produce a given sensation of brightness at each wavelength, i.e.,

$$(9) V = \frac{1}{n_o}$$

and substituting equation 9 in equation 8 gives

$$(10) a = k' V,$$

which states that the absorption coefficient of rhodopsin at any wavelength is proportional to the visibility expressed in quantum terms

at that wavelength. The values of a for various values of λ (wavelength) are known for visual purple. The values of $1/I_0$ for various values of λ are also known, and, indeed, these values determined at low intensities are the data which are plotted in the scotopic ocular visibility curve. The first task is to transform the scotopic ocular visibility curve, by means of the data which E. F. McCarthy and I obtained on the absorption spectrum of the refractive media of the human eye, into a scotopic retinal visibility curve. The observers used by Hecht and Williams in their determination of the visibility curve had an average age of "about 25 years." Therefore, E. F. McCarthy and I computed the average absorption spectrum for the refractive media of four eyes with an average age of 21.5 years, as described in a previous paper¹⁰. The scotopic ocular visibility curve was now transformed by utilizing this average selective absorption data. The result of this transformation is that the visibility curve is expressed in terms of energies incident on the retina rather than in terms of energies incident on the cornea. Taking the reciprocals of the energies incident on the retina necessary to produce a given sensation of brightness gives the scotopic retinal visibility curve.

Now a further transformation is needed. The scotopic retinal visibility curve as found by the foregoing method is expressed in terms of energies, but, according to the Stark-Einstein law the number of molecules decomposed in a photochemical reaction is proportional not to the amount of energy absorbed but to the number of quanta absorbed. Since the quantum energy varies with the wavelength, a further transformation must be made in order to express the scotopic retinal visibility curve in terms of quanta, i. e., it is necessary to know the reciprocal of the number of quanta required at each wavelength to produce a given sensation of brightness. This transformation is readily accomplished by dividing the necessary incident energy at each wavelength by the quantum energy at that wavelength. This gives the number of quanta necessary at each wavelength to produce a given sensation of brightness. Taking the reciprocal of each such number gives the scotopic retinal visibility curve expressed in terms of quanta. The total effect of both transformations is shown in chart 3. The scotopic visibility curve after both transformations is much higher than the transformed curve in the blue end of the spectrum, and its peak, as determined by several methods of interpolation, is shifted more than 10 millimicrons toward the violet end of the spectrum. Only 3 millimicrons of this shift can be attributed to the Stark-Einstein effect. Chart 4 shows the scotopic retinal visibility curve expressed in terms of quanta as compared with the absorption coefficients of rhodopsin. Since all that is necessary is that the absorption coefficients of rhodopsin should be proportional to the scotopic

retinal visibility curve in terms of quanta, it is legitimate to give both curves the same maximum ordinate, and this has been done. It is to be seen that chart 4 shows a much greater degree of agreement between the two curves than does chart 2. The comparison made by Hecht and Williams, shown in chart 1 is, of course, not adequate because (1) they did not have available to them the modern data on the absorption spectrum of rhodopsin, (2) they did not have available to them the data on the absorption spectrum of the ocular media, (3) they failed to utilize the data of Koettgen and Abelsdorff in the blue end of the spectrum, where disagreement would have been evident, and, finally, (4) they failed to make the necessary Stark-Einstein transformation. Chart 4 shows that the maxima of the two

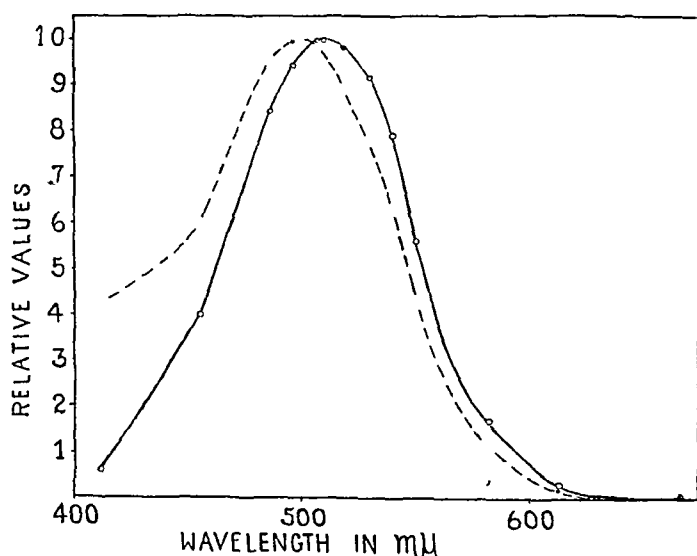


Chart 3—The solid line is the scotopic ocular visibility curve in terms of energies (Hecht and Williams), the dash line, the scotopic retinal visibility curve in terms of quanta (Ludvigh)

curves are almost coincident. The difference between the maxima of the two curves has been reduced from over 9 millimicrons to less than 1 millimicron. The determination of the maximum for each of the curves is subject not only to experimental error but to sampling error. Furthermore, the maximum in each case must be found either by interpolation or by means of an equation giving an empiric fit for the values in the vicinity of the maximum. In view of these sources of error, it may be concluded that the difference of less than 1 millimicron between the maxima of the two curves of chart 4 is not significant.

The foregoing considerations lead to the conclusion that it is unnecessary to invoke Kundt's rule to explain the discrepancy between

the maximum of the absorption spectrum of rhodopsin and the maximum of the scotopic visibility curve, because any significant discrepancy disappears when the comparison between the two sets of data is properly made. Since it is a priori improbable that Kundt's rule is operating, and since there is no discrepancy between the maxima left for it to explain, it may be concluded that Kundt's rule is not applicable to visual purple under the conditions in question. Hecht and Williams have suggested that the Kundt shift may account for the difference between the scotopic and the photopic visibility curve. But if the Kundt shift does not account for the difference of 9 or 10 millimicrons between the maxima of the two curves shown in chart 2, then it seems unlikely that it accounts for the difference of

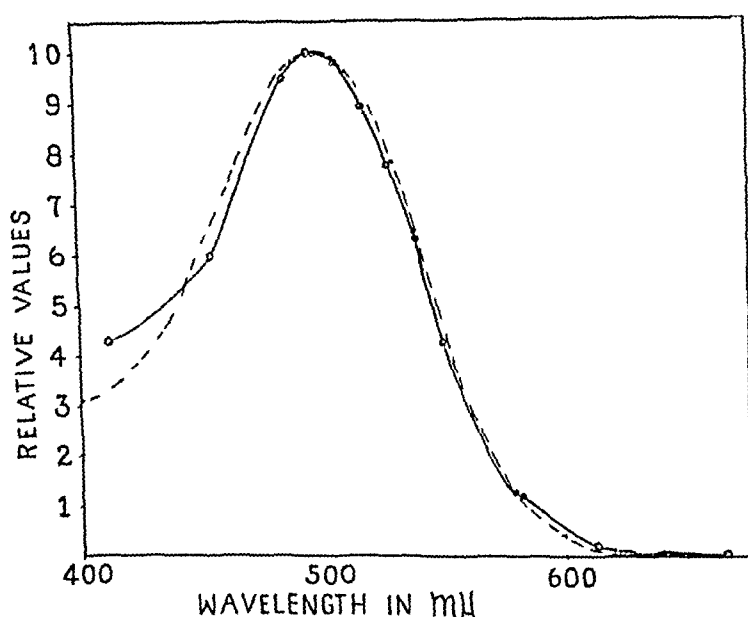


Chart 4—The solid line is the scotopic retinal visibility curve in terms of quanta (Ludvigh), the dash line, the absorption spectrum of visual purple (Lythgoe)

almost 50 millimicrons between the scotopic and the photopic visibility curve

The height of the scotopic retinal visibility curve at wavelengths less than 430 millimicrons indicates that the eye is more sensitive to light of these short wavelengths than would be expected from the absorption spectrum of visual purple. Three possible explanations for this discrepancy are suggested. 1 The selectivity of the absorption by the refractive media of the young human eye may not be so great as the calculations made by E. F. McCarthy and me indicate for the reasons discussed in a previous paper¹⁰. 2 Strong fluorescence by the retina might make the apparent sensitivity of the eye to light of short wavelengths greater than it actually is. In our determination of the absorption spectrum of the refractive media of the human eye

while the media were in situ in the eye, the retina was not included.
 3 The presence of stray light may cause the measured sensitivity of the eye to light of short wavelengths to be considerably greater than its true sensitivity. Hecht and Williams in determining the scotopic ocular visibility curve do not appear to have taken any precautions to exclude this factor. Stray light may in part account for the different results obtained by different observers¹⁹ when working at short wavelengths.

There are several interesting conclusions to be drawn from chart 4. One conclusion depends on the fact that the scotopic retinal visibility curve expressed in terms of quanta is clearly unsymmetric and not well fitted by a one term exponential formula. The high degree of symmetry of the scotopic ocular visibility curve expressed in terms of energies and also the accuracy with which it may be fitted by a simple probability equation have led several authors²⁰ to suggest that these two facts are of importance for a theory of vision. Thus Hecht and Williams² and Hecht^{1d} noted that the scotopic ocular visibility curve is symmetric and that Nutting's²¹ formula for the similar photopic ocular visibility curve is based on a probability function. Hecht assumed that "the absorption coefficient of the sensitive substance in the eye will be proportional to the reciprocal of the amount of energy needed to produce a given brightness in the eye." Then the absorption spectrum of visual purple will also be symmetric. This assumed symmetry Hecht and Williams explained by a further hypothesis. They stated "The symmetrical or nearly symmetrical curves that represent simple absorption spectra resemble strikingly the distribution curves of errors, of population, etc., which are familiar in the theory of statistics. It may be that the shape of an absorption curve represents the fact that the absorbing substance is composed not of a uniform series of resonators, but of a group of resonators whose free periods may be expressed by the common distribution curves

19 Hartman, L. W. The Visibility of Radiation in the Blue End of the Visible Spectrum, *Physiol. Rev.* **11** 327, 1918. Coblentz, W. W., and Emerson, W. B. Relative Sensibility of the Eye to Light of Different Colors and Some Practical Applications to Radiation Problems, Bulletin 14, United States Department of Commerce, Bureau of Standards, 1918, p. 167. Nutting, P. G. The Visibility of Radiation, *Phil. Mag.* **29** 301, 1915.

20 (a) Priest, I. G. The Law of Symmetry of the Visibility Function, *Physiol. Rev.* **11** 498, 1918. (b) Troland, L. T. The Enigma of Color Vision, *Am. J. Physiol. Optics* **2** 23, 1920. (c) The Nature of the Visual Receptor Process, *J. Optic. Soc. America* **1** 3, 1917. (d) Brilliance and Chroma in Relation to Zone Theories of Color, *ibid.* **6** 3, 1922. (e) Hecht^{1d}. (f) Hecht and Williams².

21 Nutting, P. G. The Luminous Equivalent of Radiation, Bulletin 103, United States Department of Commerce, Bureau of Standards, 1908, p. 261.

of the statisticians" But, as has been shown, the absorption spectrum of visual purple should resemble not the scotopic ocular visibility curve expressed in terms of energies but the scotopic retinal visibility curve expressed in terms of quanta Furthermore, since the latter curve is asymmetric, any explanation of its symmetry, whether by means of a population distribution of the free periods of a group of resonators or otherwise, is superfluous

Prior to the work of Hecht and Williams, Troland^{20b} drew from the symmetry of the visibility curve a conclusion opposite to that of Hecht and Williams He stated "This extreme symmetry of the visibility curve seems to me to militate somewhat against chemical theories of the visual receptor process Resonance curves, in general, are asymmetrical, and the same is true of absorption curves, which are particular examples of resonance curves None of these curves fit the probability function" The knowledge that the scotopic retinal visibility curve expressed in terms of quanta is markedly asymmetric both eliminates the objection of Troland and makes unnecessary the assumption of Hecht and Williams

Another important conclusion to be drawn from the agreement between the corrected visibility curve and the absorption spectrum of visual purple concerns the question of the "purity" of solutions of visual purple Thus the results of Lythgoe,⁵ of Kiause and Sidwell⁶ and of Bayliss, Lythgoe and Tansley⁷ all show that the absorption spectrum of the visual purple solutions which they used was asymmetric and higher than the scotopic ocular visibility curve in the blue end of the spectrum, as appears in chart 2 This asymmetry was thought to be inconsistent with the scotopic visibility curve and was attributed to the presence of yellow impurities Thus Bayliss, Lythgoe and Tansley stated in connection with the yellow impurities "If it were possible to make a full correction for these substances, it is probable that the absorption curve of visual purple would be roughly symmetrical when plotted to a scale of wavelengths" Lythgoe also noted that the absorption of visual purple is relatively high in the blue end of the spectrum and attributed this to yellow impurities, although the better solutions "contain only a small amount of yellow impurities" He stated that "with successive purifications the density of the unbleached extract becomes progressively less at any one of the short wavelengths" and "with a quite pure extract it would probably be very small in the region of 400 $\mu\mu$ " The present solutions almost certainly contain yellow impurities, and before one can derive values for the density of visual purple at the shorter wavelengths, it is necessary to assume values for the absorption of these impurities" Lythgoe after making allowance for the assumed values

of the absorption spectrum of the yellow impurities, found the absorption spectrum of the "parent" visual purple, which is seen to be roughly symmetric. Granit and Munsterhjelm²² compared the height of the b wave of the frog's electroretinogram with Lythgoe's data on the absorption spectrum of the "parent" visual purple and observed that "the curve for the b wave extends too far towards the violet end to fit the curve for pure visual purple." They pointed out "the necessity of finding some substance other than visual purple or a modification of visual purple in order to explain the presence of asymmetrical curves."

But, as has been shown, the scotopic retinal visibility curve expressed in terms of quanta agrees not with the absorption spectrum of Lythgoe's assumed "parent" visual purple but with his experimental data uncorrected for the presence of yellow impurities. In view of the fact that Lythgoe's uncorrected data are in agreement with those of Krause on bovine visual purple, it appears that the photosensitive substance involved in scotopic vision is not the calculated "parent" visual purple described by Lythgoe but visual purple in the form in which he actually measured its absorption spectrum. If the absorption spectrum of visual purple is due in part to yellow impurities, then these impurities must also be photosensitive. Another point to be noted is that the data of Granit and Munsterhjelm on height of the b wave agree much more closely with the absorption spectrum of visual purple than they do with that of the hypothetical "parent" visual purple. Therefore, it would seem that the solutions of visual purple obtained by Lythgoe and also those obtained by Krause and Sidwell are indeed practically pure and do not contain nonphotosensitive impurities to any marked extent.

Examination of chart 3 shows that at low intensities the retina is much more sensitive to light of short wavelengths than has commonly been supposed. This fact should be considered in relation to the blue arc phenomenon²³ and also to any theory of color vision which attributes the sensation of blue to the operation of the rods²⁴. The significance of the scotopic retinal visibility curve for theories of color vision will be considered more fully in a subsequent communication dealing with the photopic retinal visibility curve.

22 Granit, R., and Munsterhjelm, A. The Electrical Responses of Dark-Adapted Frog's Eyes to Monochromatic Stimuli, *J. Physiol.* **88** 436, 1937.

23 Newhall, S. M. The Constancy of the Blue Arc Phenomenon, *J. Optic Soc. America* **27** 165, 1937.

24 Roaf, H. E. Influence of Coloured Lights on Sensitivity of Eye to Various Regions of the Spectrum. Study in Relation to Theories of Colour Vision, *Quart. J. Exper. Physiol.* **18** 243, 1927, *Colour Vision*, *Physiol. Rev.* **13** 43, 1933.

SUMMARY

A scotopic ocular visibility curve expressed in terms of energies is transformed into a scotopic retinal visibility curve expressed in terms of energies by utilizing experimental determinations of the selective absorption of light by the refractive media of the human eye.

This scotopic retinal visibility curve expressed in terms of energies is then transformed into a scotopic retinal visibility curve expressed in terms of quanta by application of the Stark-Einstein law of photochemical equivalence.

The curve thus obtained is asymmetric and agrees with that representing the modern determination of the absorption spectrum of visual purple.

The significance of the scotopic retinal visibility curve is briefly discussed with respect to Kundt's rule, the Purkinje phenomenon, the purity of visual purple solutions, the b wave of the electroretinogram and certain visual theories.

PHYSIOLOGIC AND CLINICAL OPHTHALMOLOGIC PROBLEMS IN RELATION TO INDIVIDUAL VARIABILITY

A BRUCKNER
BASEL, SWITZERLAND

(Continued from page 541)

CONGENITAL COLOR DEFICIENCIES

In a minority of persons the form of color vision hitherto discussed, with the exception of a few individual variations which do not detract from its general character, is not applicable. These are the so-called color defectives—of whom, as modern investigations show, several types are distinguishable.

The majority of recent authors agree that besides the true partly color blind, who do not perceive certain definite colors, e. g., red and green, there are persons who are able to perceive all color qualities but whose color sense is, nevertheless, different from that of the normal person. These are called anomalous.

Concerning the vision of the congenitally color blind. It is generally accepted that it is impossible to conclude from the fact that they correctly name the colors of certain objects that they experience the same color sensations as do normal persons. The color blind have from childhood heard definite things in their surroundings called by definite names, and these names have become impressed on their minds, so that it is natural that they should use them. However, it is by no means certain that their sensations are identical with those of normal persons looking at the same things. The color-blind person probably perceives with greater subtlety than the normal person slight differences in the brightness and saturation of colors. Therefore, a person's color sense cannot be determined only by the results obtained when he names colors presented to him.

To classify persons according to their differentiation of the colors in the spectrum, for instance, is also wrong in my opinion. I emphasize this point, since Edridge-Green,³⁴ for instance, has classified persons as to their color sense into seven classes, according to the different hues which they discern in the spectrum. The one who sees but a single color is monochromatic, the one who distinguishes two colors is dichromatic, the one who sees seven colors in the spectrum, according to those of

³⁴ Edridge-Green, F. W. Classification of the Colour-Blind, *Ophth. Rev.* 35
10, 1916

Newton, is heptachromatic, that is has normal color sense. Among these the anomalous are included, despite the fact that the intensity of light, according to the Bezold-Brücke phenomenon, must influence the number of colors discriminated.

Partial Color Blindness—The color-blind subject, in contrast to the normal person, who needs three lights for the establishment of all the hues of the spectrum, can perceive all spectral hues by mixing only two—one of each half of the spectrum. Therefore, the person who is color blind is called a dichromat, the normal person, a trichromat. This name is based not on the quantity of sensations but on the number of lights necessary for mixing all the hues.

Probably the person who is color blind sees only blue and yellow in addition to the white-black sensations. The long wave half of the spectrum appears yellow, the other half, blue. Between them is a so-called neutral point, probably uncolored, where the normal person sees the pure green.

As I have already observed, the gaging of the color sense of color-blind persons is possible by means of two lights. Such gaging curves have been obtained by a great number of research workers. I shall cite only König and Dieterici, von Kries,³⁵ Tscherning and Larsen,³⁶ Tschermak³⁷ and von Brücke and Inouye.³⁸ The curves obtained by König and Dieterici are seen in figure 14. There are two sharply distinct types. Persons with these types were formerly called red blind and green blind. Now they are ordinarily designated protanopes and deuteranopes, according to the terminology of von Kries. The names formerly used may suggest that the one group is blind only for red and the other for green, though both probably are not able to perceive either red or green and see all colors only as yellow or blue of different brightness and saturation. As figure 14 shows, the two curves coincide in the part of the spectrum made up of short waves, but not in that made up of long waves. The same holds true for the curves of von Kries (fig. 15).

The maximum of the warm curve (fig. 14), that is, the part of the spectrum made up of long waves, is situated between 550 and 560 milli-

35 von Kries, J. Die Gesichtsempfindungen und ihre Analyse, Leipzig, Veit u. Comp., 1882.

36 Tscherning, M., and Larsen, H. Les anomalies des couleurs, J. de physiol. et de path. gén. **24** 475, 1926, La vision des couleurs, *ibid.* **24** 492, 1926.

37 Tschermak, A. Ueber spektrale Verteilung der Sättigung und über Dreilichter-Eichung des Spektrums, Arch. f. Augenh. **109** 1, 1935.

38 von Brücke, E. T., and Inouye, W. Ueber die Anordnung der homogenen Lichter auf der Mischlinie des Rotgrünblinden mit unverkürzten Spektrum, Arch. f. d. ges. Physiol. **141** 573, 1911.

microns for the protanopes and between 570 and 580 for the deuteranopes, while the maximum of the cold curve, the part of the spectrum made up of short waves, for both groups is to be found at 450 millimicrons

From these curves the following appearance of the spectrum for the dichromats has been suggested. There is in about the middle of the spectrum a noncolored, or neutral, zone, which lies at a different point for each type, namely, for the protanopes at about 495 millimicrons and for the deuteranopes at about 500 millimicrons. But the chief difference is to be found at the red end of the spectrum, which for the protanopes is dark and therefore shortened, while for the deuteranopes it generally has the same brightness as for persons with normal color sense. The saturation increases on both sides of the neutral zone and decreases again toward the ends of the spectrum, as shown in the curves.

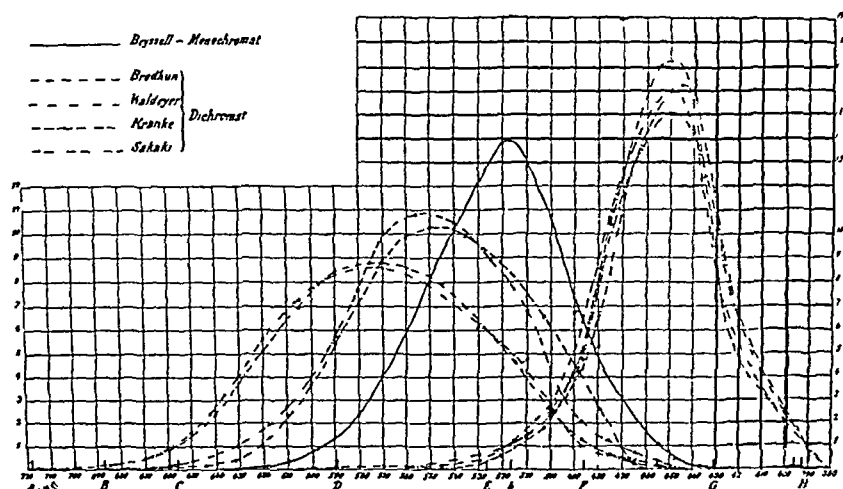


Fig 14 Gaging curves for dichromats. The curves of Brodhun and Waldeyer are for deuteranopes, and those of Kranke and Sakaki are for protanopes. Beysell's curve is that of a totally color-blind person. (After König and Dieterici²⁷)

Recently Tschermak published a report of experiments which show that the value of the color, or, better, the saturation, does not diminish toward the ends of the spectrum. This finding seems to me to be doubtful. If the curves of König and Dieterici for dichromatic vision are transformed in the same manner that I described for normal color vision, their course does not agree with Tschermak's suggestion. The mean curve for the dichromats is shown in figure 16, in which the continuous line represents the protanopes and the dotted line the deuteranopes.

If Hering's antagonistic theory is right, the areas of yellow and blue for the dichromats should be equal. Apparently this is not so, perhaps for the same reason that I have already discussed, in con-

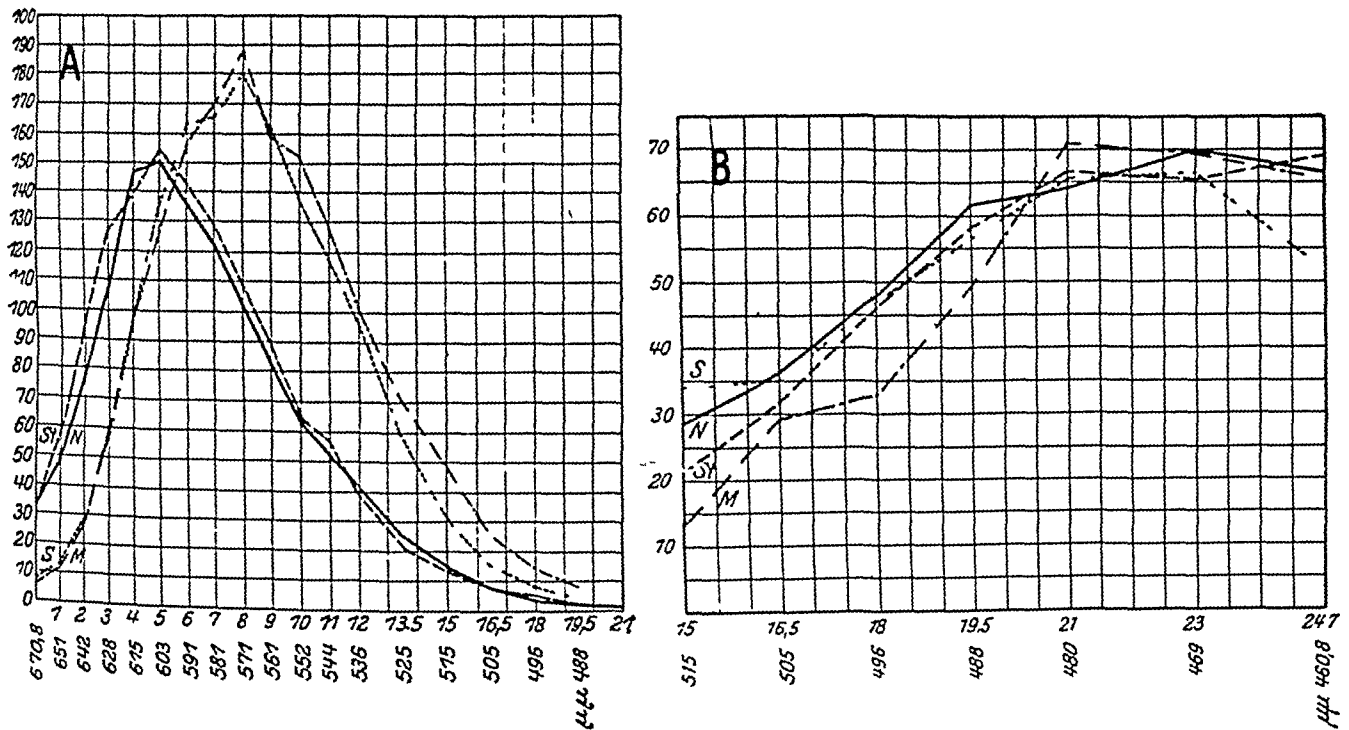


Fig 15—A, distribution of the red values in the dispersal spectrum of gaslight for 2 protanopes (*S*, indicated by the dotted line, *M*, by the dash and dot line) and for 2 deuteranopes (*N*, indicated by the continuous line, *St*, by the dash line) B, distribution of the blue values in the dispersal spectrum of gaslight for 2 protanopes (*S*, indicated by dotted line, *M*, by dot and dash line) and for 2 deuteranopes (*N*, indicated by a continuous line, *St*, by a dash line) (After von Kries, J Ueber Farbensysteme, Ztschr f Psychol **13** 241, 1897)

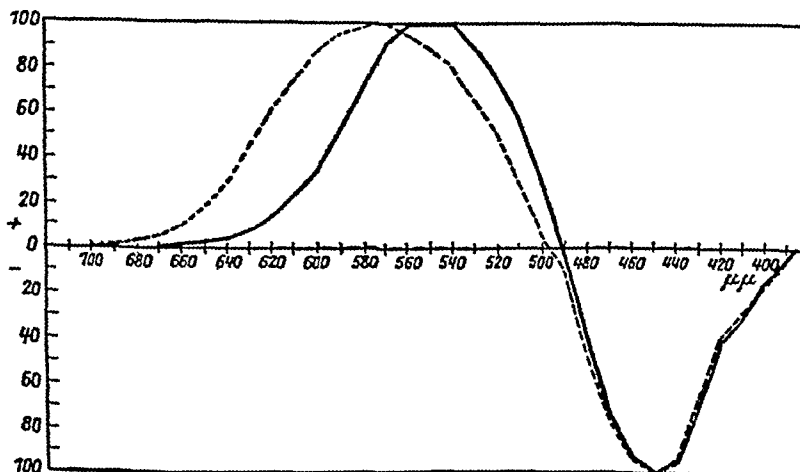


Fig 16—König's gaging curves for dichromats transformed into Hering's curves The continuous line is for a person who is red blind, the dash line is for a person who is green blind (After Bruckner³⁰)

nection with the curves of normal color vision, namely, the yellow-reddish character of the daylight or the influence of diascleral light

Anomalous Trichomats—The group of anomalous trichomats was first discovered by Lord Rayleigh³⁸ in 1881. They need three gaging lights in order to produce all hues, hence their name, anomalous trichomats. Lord Rayleigh investigated color equations by a mixture of a spectral red of 670 millimicrons (a line of lithium) and a spectral yellowish green of 530 millimicrons (a line of thallium), which appears equal to the yellow of 589 millimicrons, the line of sodium. The proportions of the two components required to reach an equation was nearly the same. A few of his observers required more green, and a few, more red. Mixed lights prepared to meet the requirements of these groups were naturally too green or too red for normal persons. Further experiments by Donders³⁹ and von Kries confirmed this result.

The terminology is at best a little complicated. At first the group which needs more green was called green anomalous, the other, red anomalous. Then von Kries introduced the term protanomal for those who need more red and deuteranomal for those who require more green in the test equation named the Rayleigh-Donders equation.

When tests are made some anomalous are found who not only accept a strictly definite proportion of the two mixed lights of 670 and 530 millimicrons but who accept with the homogeneous yellow light of 589 millimicrons a larger amount of different proportions of the two other lights when the luminosity of the yellow light alone is correspondingly altered. Some of them accept the equation of pure yellow and pure green, or of yellow and pure red. They were called the extreme deuteranomals or protanomals. Probably they represent transition types between the normal and the color blind. But it seems necessary to gage the whole system of these types and not to limit the test to the single Rayleigh-Donders equation, as has hitherto usually been done for persons with these extreme forms of anomaly.

Originally, the anomalous were looked on merely as a theoretically interesting variety. The detailed investigations by Nagel³⁹ and Guttman⁴⁰ at the beginning of the century proved that these so-called anomalous trichomats are in many respects defective. They have always higher specific thresholds than normal persons. They require a larger stimulus, in the form of increased size of field, intensity of light, saturation or duration. For instance, a small colored field can be perceived by the anomalous at a shorter distance than by normal persons.

This fact of higher thresholds leads to consideration of the anomalous as color defective and therefore to their exclusion from the navy and

39 Nagel, W. A. Die Diagnose der anomalen trichromatischen Systeme, *Klin Monatsbl f Augenh* **42** 366, 1904.

40 Guttman, A. Untersuchungen über Farbenschwache, *Ztschr f Psychol u Physiol d Sinnesorg (Abt 2)* **42** 24 and 250, 1907, **43** 146, 199 and 225, 1908.

the railway service, because there differentiation of colored signals is necessary. In England, as far as I know, these regulations have not been strictly adopted, but in Germany, Switzerland and recently also in France the anomalous are excluded from the railway service.

I shall not go into detail about the results of investigations of the color thresholds of the anomalous. They are only in part satisfactory. I shall confine myself to presenting curves showing the thresholds for the duration of the colored light—*die Farbenzeitschwelle*.

These results were obtained by using a so-called tachistoscope, which allows the exposure of a colored field for a very short fixed time, usually only a few thousandths of a second. The investigation showed that generally the time of the exposure required for the perception of the specific color depends on the luminosity of the screen on which for a moment the colored light appears. The duration is, as will be seen in figure 17, a linear function of the brightness of the screen. Red has the lowest threshold, green, the highest.⁴¹

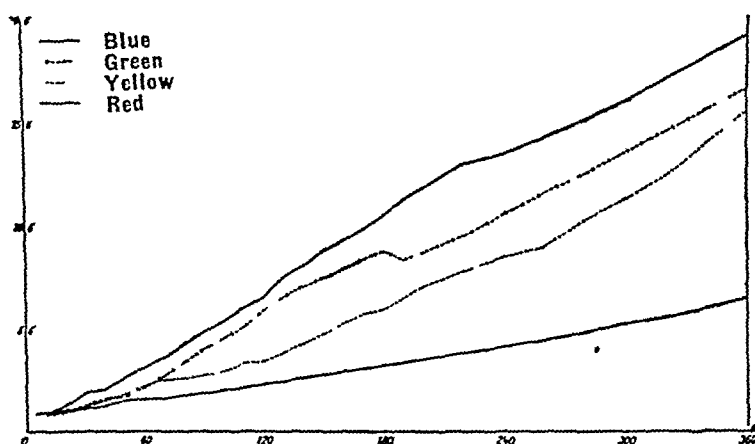


Fig 17—Curves of the thresholds for the duration of the colored light in relation to the luminosity of the screen on which the color appears (abscissas). The numbers for the degrees indicate the turbidoid values of gray, 0 equals black, 360, white. The ordinates indicate the time in thousandths of a second (σ) during which the colored field was exposed. (After Bruckner and Kirsch⁴¹)

This fact has already been established by Guttman, who when using spectral colors found the thresholds much lower than those obtained with pigment colors. He therefore suggested that the former have a higher specific value than the latter. But this explanation is not correct. The differences found by Guttman are related only to the brightness of the surface on which the colored light appears, in the spectroscopy the field is ordinarily very dark, say black. Therefore, in contrast to the other experiments, the thresholds are very low.

The anomalous trichromats have, compared to normal persons, high thresholds for the length of exposure to colored light. This holds especially for green for deuteranomals (fig 18). Protanomals were not

⁴¹ Bruckner, A., and Kirsch, R. Untersuchungen über die Farbenzeitschwelle, *Ztschr f Psychol u Physiol d Sinnesorg* (Abt 2) 46:229, 1912.

examined by this method. The increase of this one threshold seems to be sufficient ground for excluding the anomalous from the railway service, because mistakes may be followed by disastrous consequences.

Gaging curves for the ordinary forms of anomalous color vision are given by many investigators, e. g., König and Dieterici, Tscherning, Pitt and others. I shall show only the curves of König and Dieterici transformed into curves corresponding to Hering's for pure colors, the coordinates having been altered in the manner already described (figs

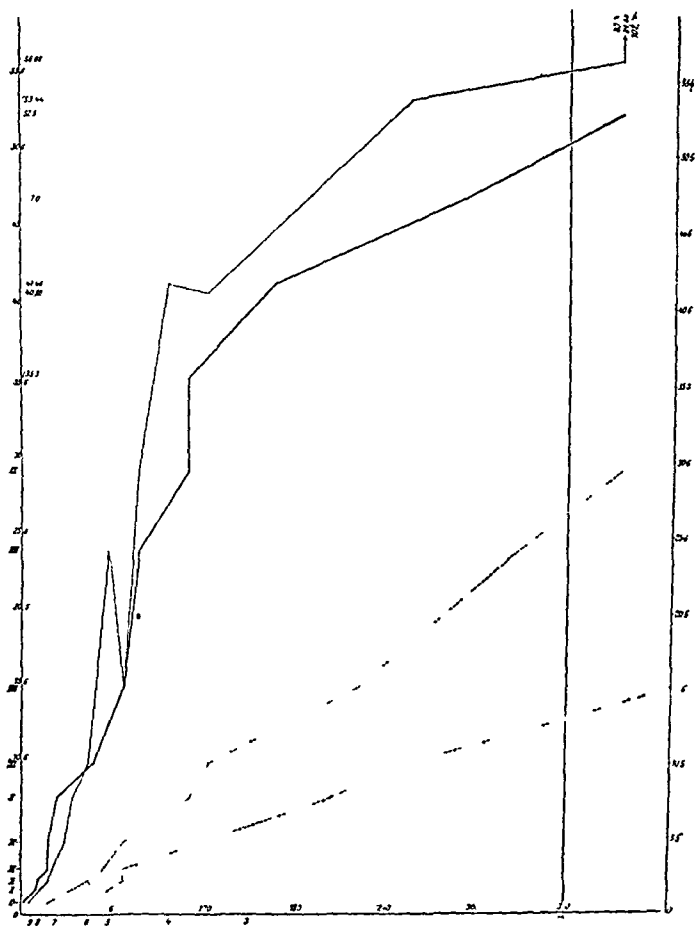


Fig. 18—Duration thresholds for green of two deuteranomals (continuous lines) and two normal persons (dotted lines) (After Bruckner and Kirsch⁴¹)

11 and 12) It is to be observed that for green the curve of the anomalous is a good deal lower than that of normal persons. This agrees with the fact that in practical life this group is especially deficient in the green color sense. For protanomals the same finding is not yet established by this method, especially with regard to red.⁴²

⁴² I should mention the opinion of Hess, who found, especially in deuteranomals, an increased yellow-blue sensitivity. His results do not appear to be fully convincing.

Of the rare deficiency of color vision for yellow-blue, I shall say only a few words. Some persons with what is described as yellow-blue blindness have also an insufficient red-green sense. This fact suggests that the deficiency in color vision is not congenital but acquired. Yet in some cases it may be congenital, though not necessarily hereditary. In a few cases described recently by Engelking⁴³ as instances of tritanomalous deficiency the condition was hereditary, as shown by the pedigree. But it seems to me it is not absolutely justifiable to conclude that this anomaly is determined by a genetic cause in the so-called X chromosome, as other forms are supposed to be.

Diagnosis—For the clinical diagnosis of the disturbances of color sense it is naturally not possible to obtain gaging curves. Simple tests have been used for many decades past. The oldest test is that of Seebeck,⁴⁴ who in 1837 suggested the use of color pigments. About fifty years later Holmgren⁴⁵ suggested the use of colored wools. The person whose color sense was to be tested was required to match a given color with the other colored wools. From the mistakes in matching the physician drew his conclusions as to the patient's color sense. It is now known that many anomalous trichromats are able to pass this test, apparently because the individual fields are not small enough. The size of the colored field is of the greatest importance for the recognition of colors by the anomalous.

Far better for this purpose are the so-called pseudoisochromatic tables, first used by Stilling and improved by other authors. First place is to be given to the Ishihara test⁴⁶ and to the latest editions of the tables of Stilling⁴⁷. In these tables small colored fields of different form and size are printed. In these the confusion colors of the color blind are printed on a card in the form of small spots which are so arranged that normal persons and those who are color blind recognize different figures. For the illiterate there are other cards, with winding lines which the patient is required to trace with a pointer.

Other charts, based on the same principle of pseudoisochromatism, namely colors which falsely seem to the color blind to be the same, have been published by Nagel, Podesta, Schaaff, Pollack and Bostroem. All these tests are much better than the color-matching tests of Holmgren and are more suitable for investigations of school children or for determining the incidence of color deficiency in the population.

43 Engelking, E. Die Tritanomalie, ein bisher unbekannter Typus anomaler Trichromasie, Arch f Ophth **116** 196, 1925.

44 Seebeck. Ueber den bei manchen Personen vorkommenden Mangel des Farbensinnes, Ann d Phys u Chem **42**:177, 1837.

45 Holmgren, F. Die Farbenblindheit in ihren Beziehungen zu den Eisenbahnen und der Marine, Leipzig, F C W Vogel, 1878.

46 Ishihara, J. Test for Colour-Blindness, ed 7, Tokyo, Japan, Kanehara & Co., 1936.

47 Stilling, J. Stillings pseudo-isochromatische Tafeln zur Prufung des Farbensinnes, ed 18, Leipzig, Georg Thieme, 1929.

These tests are suitable for rapid orientation, and when properly applied hardly a single color-deficient person escapes detection. Nevertheless, I have observed rare cases in which color defect could be detected only by tests with the spectroscope. As to the constitution of the color sense itself, these tests do not reveal much. They determine merely whether or not the person examined has a normal color sense. It is not possible by their use to establish with certainty the type of color defect present.

This may be more exactly diagnosed by the anomaloscope constructed by Nagel⁴⁸ for this special purpose. This apparatus,⁴⁹ a small spectroscope, is used in Germany, and for the past ten years has been used in Switzerland also, for the examination of all candidates for the railway service. It has been adopted in France during recent years. From experience in Switzerland, where all candidates are examined by Stilling's tables and the anomaloscope, I regard this combined method as completely sufficient.

The anomaloscope (figs 21 and 22) reproduces the original Rayleigh-Donders equation. The patient is shown a field one half of which is illuminated by a light of 589 millimicrons and the other half by a mixture of lights of 670 and 530 millimicrons. It is therefore easy to determine the two types of anomalous as well as the true color blind. If one uses homogeneous red and homogeneous yellow or green and yellow in the case of color blindness, an equation is obtained by regulating only the luminosity. Therefore, according to the axiom that if two quantities are equal to a third they are equal to one another, it is permissible to suppose that in this case green and red seem equal, as is supposed to be the case in color blindness.

As I have already said, there are two types of persons with color blindness and with color anomaly. The relations between them are not clear. Hering⁵⁰ suggested that the protanopes and deuteranopes may be differentiated by different pigmentation of the macula or of the lens. But von Kries rightly denied this, on the ground of the gaging curves. It is now known that they have substantially different systems of color sense. The same holds for protanomals and deuteranomals.

Frequency of the Different Types of Color Deficiency—As to the frequency of the different types of color deficiency, recent statistical investigations provide reliable information.

48 Nagel, W. A. Zwei Apparate für die augenärztliche Funktionsprüfung, Adaptometer und kleines Spektralphotometer (Anomaloskop), *Ztschr f Augenh* 17 201, 1907.

49 The anomaloscope is manufactured not only by Schmidt & Haensch, Berlin, but by Optik und Mechanik A. G. in Basel.

50 Hering, E. Ueber individuelle Verschiedenheiten des Farbensinnes, in *Wissenschaftliche Abhandlungen*, Leipzig, Georg Thieme, 1931, vol. 2.

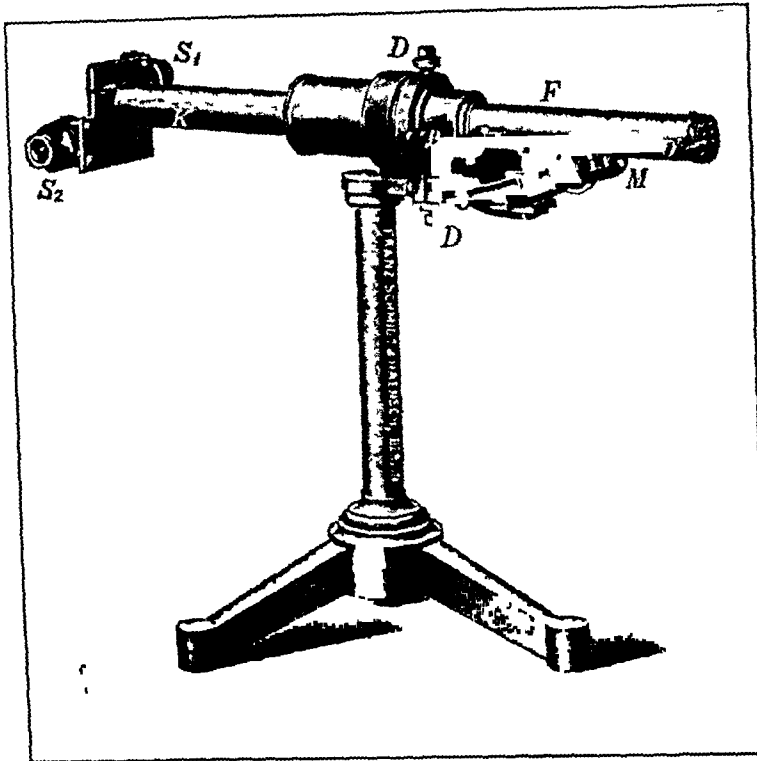


Fig 21 ^{54a}—Nagel's anomaloscope *K* indicates the collimator tube, *D*, the direct vision prism, *B*, the diaphragm, *F*, the ocular tube, *O*, the aperture of the ocular tube through which observation is made, *M*, the screw for changing the situation of the ocular tube, and *S*₁ and *S*₂, the slit mechanism (After Nagel ⁴⁸)

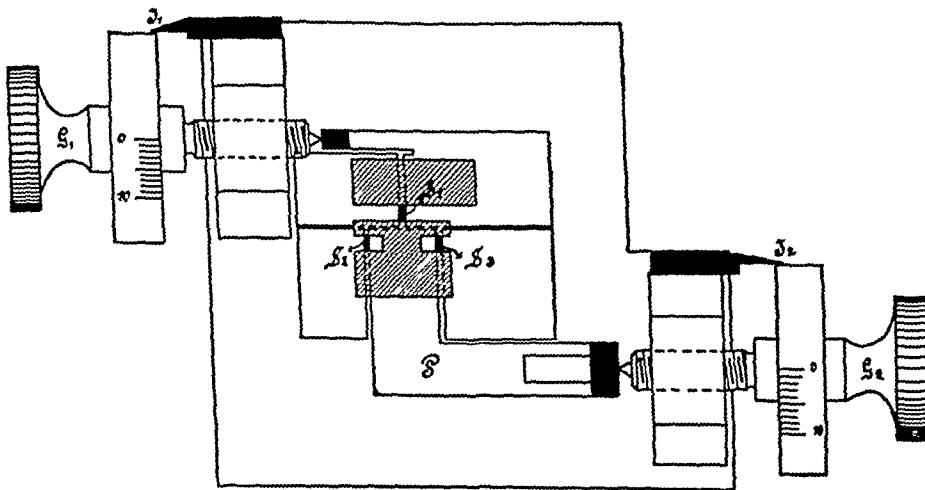


Fig 22 —Nagel's anomaloscope View of the slit mechanism *S*₁ indicates the slit for homogeneous light of 589 millimicrons, *S*₂ and *S*₃, the slits for the red and green lights of the mixed field, *G*₁ and *G*₂, screws for changing the width of the slits (After Nagel ⁴⁸)

The older statistics not based on the use of the anomaloscope are not satisfactory. I may mention the researches of Waaler⁵¹ in Norway, and those of von Planta,⁵² Brunner⁵³ and Wieland⁵⁴ in Basel, carried out in schools. The investigations concern a total of about 11,000 boys and 12,000 girls. As seen in table 2, boys predominate over girls in color deficiency, as was known long ago. The most frequent form is the deuteranomalous and the least frequent the protanomalous, while deuteranopia and protanopia take an intermediate position (table 3).

Heredity of Color Deficiency—In the population of Europe about 8 per cent of the men are color deficient, but only about 0.4 per cent of the women. This proportion is due to the fact that the heredity of

TABLE 2—*Frequency of Congenital Color Deficiencies*

Author	Frequency, Percentage	Number Examined	Mean Error	
	In Boys			
Waaler	8.01	9,049	± 0.29	
von Planta	7.95	2,000	± 0.37	
Author	Theoretic Frequency, Percentage	Actual Frequency, Percentage	Number Examined	Mean Error
	In Girls			
Waaler	0.642	0.44	9,072	± 0.07
von Planta	0.631	0.43	3,000	± 0.12

TABLE 3—*Frequency of the Different Types of Congenital Color Deficiency in Boys*

Color Deficiency		Results of Waaler, Percentage	Results of von Planta, Percentage
a	Protanope	0.88	1.60
b	Protanomalous	1.03	0.60
c	Deuteranope	1.04	1.50
d	Deuteranomalous	5.06	4.25
Total		8.01	7.95

color deficiency is sex-linked recessive, that is to say, it is bound to the so-called X chromosome.

The regular form of sex-linked heredity is transmitted by the phenotypically normal woman from her affected father to some of her sons (fig. 23). The daughters also are partially genotypically normal,

51 Waaler, G. H. M. Ueber die Erbliehkeitsverhältnisse der verschiedenen Arten von angeborener Rot-Grünblindheit, *Acta ophth* 5:309, 1927, *Ztschr f indukt Abstammungs- u Vererbungs* 45:279, 1927.

52 von Planta, P. Die Häufigkeit der angeborenen Farbensinnstörungen bei Knaben und Mädchen und ihre Feststellung durch die üblichen klinischen Proben, *Arch f Ophth* 120:253, 1928.

53 Brunner, W. Ueber den Vererbungsmodus der verschiedenen Typen der angeborenen Rotgrünblindheit, *Arch f Ophth* 124:1, 1930.

54 Wieland, M. Untersuchungen über Farbenschwache bei Konduktorinnen, *Arch f Ophth* 130:441, 1933.

partly affected but phenotypically also normal. There can be a color-deficient daughter only when both X chromosomes are affected.

This fact naturally suggests the necessity to investigate the relation between the different types of deficiency for red-green sensations,

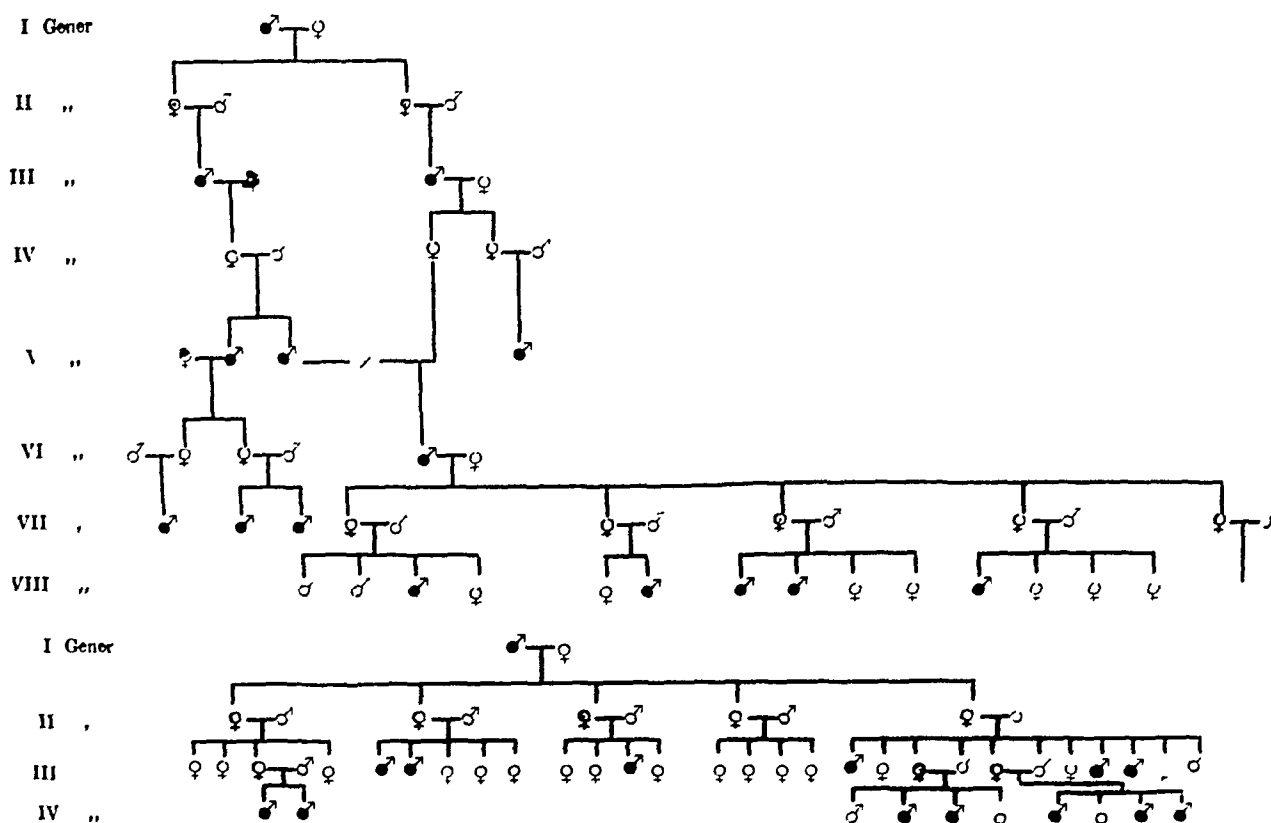


Fig 23—Pedigrees of Horner (1876), heredity of red-green blindness. The black circle indicates a color-blind person, the circle with a dot in the center, a woman transmitting the defect, a white circle, a normal person, and X, marriage of a color-blind man and a woman transmitting color blindness, ♂ indicates male and ♀, female. (After Franceschetti, in Schieck, F, and Bruckner, A. *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930.)

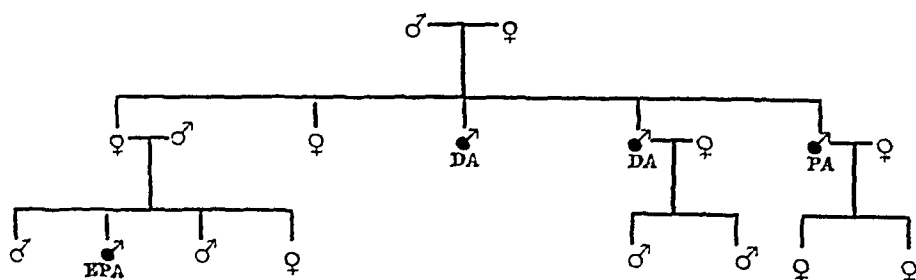


Fig 24—Appearance of deuteranomaly and protanomaly in the same family (After Brunner⁵³). DA indicates deuteranomaly, PA, protanomaly, EPA, extreme protanomaly.

especially to determine if there is a closer connection between deuteranopes and deuteranomals, on the one hand and between protanopes

and protanomals, on the other hand. Here the works of Waaler and Brunner have enriched knowledge remarkably.

Waaler expressed the opinion that the protanopes and the deuteranopes are genetically different. He published some pedigrees which supported this suggestion, and Brunner pursued these investigations still further. I shall discuss only a few important points of these results.

As already noted, a female will be color blind if both X chromosomes are affected by the gene of color deficiency. If one of them is affected by the factor for protanopia and the other by that for deuteranopia and these two are not so-called allelomorphs, the child cannot be color blind. Waaler called them compounds. The pedigree in figure 24 shows that a phenotypically normal woman has color-deficient sons of both types, which can be explained only if one supposes that the two genes for different forms of color deficiency split off into the different forms in the sons.

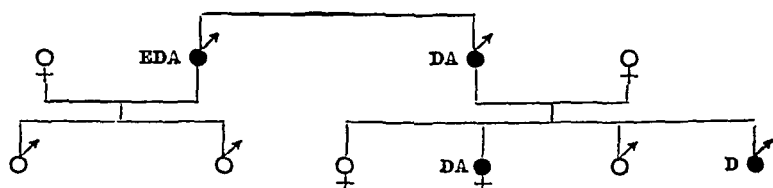


Fig. 25—Appearance of different degrees of color deficiency in the same family. *D* indicates deuteranopia, *DA*, deuteranomaly, *EDA*, extreme deuteranomaly (After Brunner⁵³).

A pedigree like this abolishes the former suggestion of Hering that the two types of protanopes and deuteranopes are produced only by subordinate or physical differences. One must suppose that these two types are fundamentally genetically different or, according to Morgan, that the locus of their genes in the chromosome is different.

A second fact of some importance is obvious from the pedigree (figs 25 and 26). There are in the same family persons with different degrees of color deficiency, for instance, deuteranopes and deuteranomals or deuteranopes and extreme deuteranomals. As far as heredity is concerned, it is of interest that the deficiency of a lower degree always dominates over that of a higher degree. But it must not be forgotten that the diagnosis in all these cases was made by testing by the Rayleigh-Donders equation alone. Therefore, it is not known what gaging or measurement of the color thresholds would show.

Nevertheless, it is conceivable that in respect to heredity there may be quantitative differences in the gene for color sense. How this is to be explained is still uncertain, but perhaps the same circumstances are valid as hold good in regard to the heredity of sex, according to Goldschmidt.

In this connection I may refer to some investigations carried out in the ophthalmologic clinic of Basel by Wicand⁵⁵ and Meister⁵⁶ concerning the color thresholds. Fleischer⁵⁶ observed that the color sense of women who transmit color deficiency was sometimes slightly deficient, although it had hitherto been considered normal. But Fleischer did not determine this by precise methods. Wicand and Meister used an apparatus constructed by Pulfrich of the house of Zeiss at Jena, the so-called *Stufenphotometer*. This apparatus allows manifold applications, especially in the investigation of color thresholds.

Two of Ostwald's small colored cards were used as a pure red and pure green. In addition, the light reflected by a gray card of the same brightness as the colored one was exactly measured in order to determine the amount of green or red necessary to permit appreciation of the color. These experiments showed that for normal persons 5 to 6 per cent of red and 10 to 17 per cent of green was necessary. For the anomalous the thresholds were considerably raised, for green, 30 per cent or more.

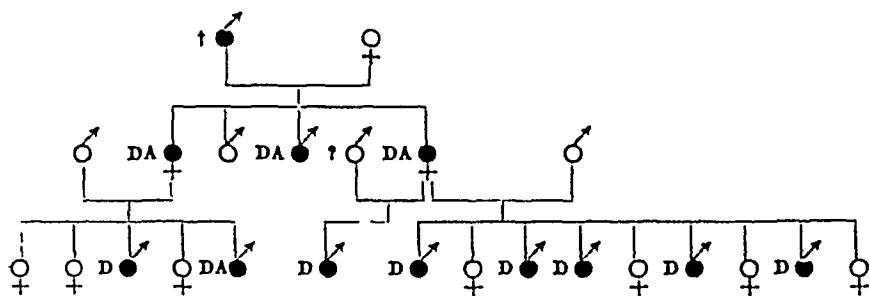


Fig 26—Occurrence of deuteranopia (*D*) and deuteranomaly (*DA*) in the same family (After Brunner⁵³)

But women who transmitted color deficiency, irrespective of the form of deficiency they transmitted, had a remarkable increase in the threshold for red and still more in that for green. This is true also of the mathematical evaluation of errors.

The conclusion seems justified that even in the apparently normal woman who transmits deficiency, slight deficiency in color vision is present. Study of heredity has shown similar phenomena, for instance concerning the apparently dominant color of some flowers. These examples suggest that there is some deficiency in the gene for color vision of the one who transmits deficiency.

I believe that exact investigation of color thresholds will elucidate many points that are still doubtful. But for the present it may be

55 Meister, H. Untersuchung spezifischer Schwellen mittels des Pulfrichschen Stufenphotometers, *Ztschr f Sinnesphysiol* 65 217, 1934

56 Fleischer, B. Ueber die Vererbung geschlechtsgebundener Krankheiten, *Ber u d Versamml d deutsch ophth Gesellsch* 42 4, 1920

stated that the majority of persons having normal color sense are supplied with such an amount of hereditary mass responsible for this visual function that small differences in color vision do not produce a remarkable difference of reaction in practical life. Apparently a much larger deficiency in the X chromosome is necessary to produce the ordinary characteristic types of alteration of color sense. Perhaps in such cases some remains of genetic substance may exist, and therefore the deficient persons may have a slight faculty, for instance, for perceiving red. Some statements of Nagel⁵⁷ and other authors who were themselves color blind seem to suggest this. However, the two great groups of protanopes and deuteranopes certainly persist, and transition types between them do not occur.

Research in genealogy has also demonstrated that the opinion of Georges Elias Muller⁵⁸ is not quite justified. He supposed the anomalous to represent altered systems of color vision, *Alterationssysteme*, their character being only a slightly different reaction to colored lights. In some sense he may be right. But with regard to heredity research has shown that the scientific standpoint must be changed and that genetically determined differences do exist.

Many questions are as yet insufficiently investigated, especially the exaggerated contrast of the anomalous, which I shall discuss later.

Total Color Blindness—Only a few comments need be made about congenital total color blindness. Persons with such a defect are not able to perceive colors, they see only black, gray and white. It is possible to produce excitation for all these colors by changing the intensity of only a single homogeneous light. Therefore, the persons are called monochromats. To them the brightness of the different parts of the spectrum or of other colors does not appear at all as it does to normal persons or to deuteranopes, to the totally color blind they appear exactly as the different colors or parts of the spectrum appear to the normal dark-adapted eye.

Total congenital color blindness is, as shown by the study of pedigrees, a recessive hereditary condition (fig. 27). The eye which has a much diminished visual acuity—not more than a tenth or a seventh of normal vision—is dazzled by light and has nystagmus. Dark adaptation is not diminished, but apparently light adaptation is. Figure 28 shows the typical behavior of a totally color-blind child in daylight.

If it is supposed that in such an eye only the dark adaptation apparatus functions, and since only cones are to be found in the human macula, the reduction of visual acuity is explained, because the normal

57 Nagel, W. *Farbenumstimmung beim Dichromaten*, *Ztschr. f. Psychol. u. Physiol. d. Sinnesorg.* (Abt. 2) **44**, 5, 1909.

58 Muller, G. E. *Darstellung und Erklärung der verschiedenen Typen der Farbenblindheit*, Göttingen: Vanderhoeft & Ruprecht, 1924.

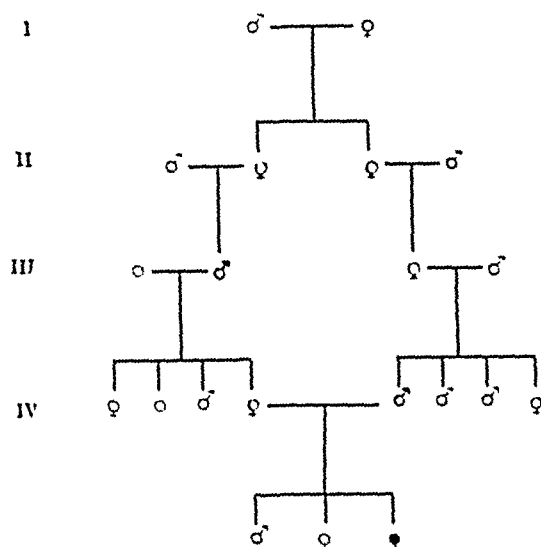


Fig 27—Pedigree in total color deficiency. Manifestation of the deficiency owing to consanguinity of the parents. The black circle indicates a person with color deficiency, the white circle with the black dot, a person who transmits the deficiency and the white circle, a normal person. (After Vogt and Franceschetti, in Schiek, F, and Bruckner, A. *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol 1.)



Fig 28—A boy who is totally color blind, with characteristic photophobia.

dark-adapted eye has also a reduced visual acuity and a relative central scotoma. Nystagmus is due to the loss of central fixation. The fact that the person is dazzled is easy to understand, because the apparatus of light adaptation either is absent or does not function.

Development of Color Sense—A few words may be said about the hypothesis concerning the phylogenetic development of color sense.

Schrodinger⁵³ suggested that monochromatic vision was, phylogenetically, the oldest form, in such vision only different degrees of brightness, and no color, can be distinguished. Schrodinger expressed the opinion that in earlier periods of the earth's history the misty condition of the atmosphere due to its saturation with moisture allowed the light to reach the surface of the earth only in small quantities. Therefore, the color sense was undeveloped.

The faculty of distinguishing between yellow and blue is mostly considered to be a phylogenetically older faculty than that of the recognition of green and red. With regard to the heredity of color sense deficiencies, this opinion seems probably to be correct. Perhaps there may be a form of mutation. But to establish definite hypotheses, such as that of Ladd-Franklin and Lemmon⁵⁹ (who suggested that the red-green sense was developed by splitting off the elements corresponding to the yellow sensation), seems to me hardly permissible with present day knowledge.

PHENOMENA OF LOCAL ADAPTATION

I have hitherto dealt only with the color sense of the central part of the retina, of the macula or fovea. In the peripheral portions of the retina the color vision diminishes in proportion to the increasing eccentricity of the stimulated spot in the retina. The handbooks always repeat the statement that all colors can be perceived up to about 20 degrees and that there follow a zone which is red blind and then a red-green blind zone, total color blindness being assumed at the extreme periphery.

This opinion is generally accepted, but it is not correct. That color blindness of the peripheral parts of the retina is only relative was demonstrated by Tschermak,⁶⁰ a pupil of Hering, about forty years ago. If the stimulus is intense enough, namely, of sufficient objective saturation and extent, one is able to perceive all colors even at the extreme periphery of the field of vision.

If a piece of well saturated green or red paper about 10 cm in diameter is placed in the extreme periphery of the visual field, one sees

59 Lemmon, V. A. A Modification of the Ladd-Franklin Theory of Colour Vision, *Am J Physiol Optics* 6 449, 1925.

60 Tschermak, A. Beobachtungen über die relative Farbenblindheit im indirekten Sehen, *Arch f d ges Physiol* 82 559, 1900.

the color without difficulty. But if the eye does not move, the color becomes invisible after a short time and only an uncolored sensation remains. Therefore, one may conclude that the stimulation of the same area for a short time causes color blindness from exhaustion. In comparison with the macula the periphery of the retina becomes fatigued more quickly.

This experiment is a remarkable illustration of the fact that the psychic correlative, the saturation, is determined not only by the specific type of the physical stimulus acting on the peripheral organ but by the condition of the body, and that changes in this are of the greatest importance.

PHENOMENA OF REVULSIVE LOCAL ADAPTATION

I shall now discuss the phenomena of revulsion—“*die Umstimmungserscheinungen*,” as Hering called them. These phenomena of the change in the response to stimulus are still often looked on as accidental occurrences or as curiosities. Hering studied the phenomena of adaptation by intensive investigations about sixty years ago, and his results are still valid. The chief suggestion is that the visual organ changes more or less rapidly under the influence of each successive illumination, whether the area is small or large. The change in response to stimulus is not confined to the zone directly affected by the light but extends to the whole retina, though in decreasing degree as the distance from the directly stimulated spot increases.

These phenomena are also called local adaptation, because they are caused by stimulation of a limited zone of the retina only, in contrast to general adaptation, for instance that of dark and light adaptation, which concerns the whole visual apparatus.

If one stares at a black spot for twenty seconds and then looks at a white field, one sees a very bright white spot on the latter. The white light does not produce the same effect in that part of the retina previously less affected when one was looking at the black spot. The same experiment can be carried out with a red spot, when a green after-image, as this phenomenon is called, will be perceived, or with a yellow spot, which produces a blue after-image.

In these experiments the change in response is studied in the zone of the retina which was previously affected by light. In other experiments is seen the alteration of excitability, depending on the environment of the part stimulated or on the differences of two neighboring fields. For instance, on looking at figure 29 one has the impression that the two gray V's are not of the same luminosity. When the surrounding black and white fields are covered, one sees that the gray lines are of the same brightness. They change only by reason of the quality of the two adjacent fields.

If after an exact fixation for about a minute the after-image is studied, it is perceived that its brightness completely accords with the inverse brightness of the original image. This fact demonstrates the validity of the phenomena called simultaneous and successive contrast.

If one looks at figure 30, fixing the eye exactly on the center of the white square for one or two minutes, another phenomenon will be observed. The immediately surrounding black becomes brighter, while

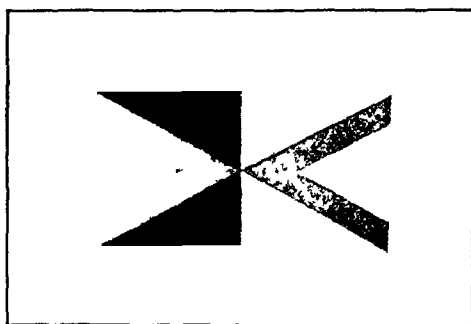


Fig. 29—The two "V"s are of the same brightness on both sides. That on the dark background, however, appears by contrast to be lighter than that on the light background. (After Hering.)



Fig. 30—Demonstration of spatial induction of same color.

the white spot itself becomes more or less dark. That is a spatial induction in the same color (*gleichsinnige Induktion*), which is the inverse of the antagonistic contrast already demonstrated. One may say that the antagonistic induction overcomes the simultaneous contrast, later the induction of the same sense predominates, which passes over into the successive contrast and the negative after-images.

These experiments show the extremely manifold reactions and the alternating effects in the visual substance. These are continually changing under the influence of stimulation by light and of regeneration taking place within the visual substance.

One may object that the alterations shown by these experiments are effected by an extreme degree of fatigue, such as does not normally influence the eye. But careful observation proves that the same effects arise, though in less degree, by short exposure. The contrast phenomenon is noticed even with momentary exposures.

Black Sensation—The facts just reported are of importance in clearing up the question of the black sensation. The physicist maintains that black is seen when objectively there is no stimulus. A thing seems black when all light falling on it is absorbed and not reflected. That is the definition of a black body. But it accords only with the physical circumstances, not with the physiologic or the psychologic.

When one is in an absolutely dark room, the visual sensation is not a deep black but a more or less dark gray. When one awakes before dawn, all objects in the room look grayish. As the illumination increases, the difference in the brightness of the objects becomes more and more noticeable, some becoming brighter and others darker, until one perceives a real black. This sensation is called forth by contrast or simultaneous induction. This can be demonstrated by taking a gray paper with a hole 1 cm in diameter in the center and observing through it an evenly illuminated gray paper placed below it. When the paper with the hole in it is moved toward and then away from the window, the color of the paper observed through the hole will appear to change from gray to black.

This experiment demonstrates, as was first shown by Heering,⁶¹ that the black sensation arises only in an indirect manner in the visual substance. The opinion that black is not a sensation because there is no stimulus corresponding to it was adopted by von Kries⁶¹ and later by Kirschmann.⁶² But an ordinary man is certainly convinced that black is a sensation just as much as white or a color.

The same applies to brown as to black. Only a blackish orange can be perceived if the surroundings are illuminated and the intensity of the orange is not too strong. To prove this one can use the same gray paper with the hole 1 cm in diameter that was just mentioned, with an orange paper placed below it.

Importance of Contrast—The importance of contrast for distinct vision is great. Only by contrast can individual objects be distinguished sharply from one another. Reading also is possible only through contrast.

In consequence of the diffusion of light in the eye caused by irregularities in the media, the image thrown on the retina is not sharp. Therefore, there can be no exact vision. Figure 31 shows the suggested

61 von Kries,⁸ p. 8

62 Kirschmann, A. *Farbenterminologie*, *Neue psychol. Studien* 2:127, 1926

course of the distribution of the light on the retina effected by two bright objects with dark surroundings. The height of the ordinates representing the luminosity on the different parts of the retina does not change suddenly, corresponding to the limit of the white field. The psychic effect would be a very faded image.

Simultaneous contrast by its alternating effect (*Wechselwirkung*) depresses the lower part of the curve and raises the other. In this manner, psychically, a sharp boundary is again produced. Figure 32, published by Tschermak,⁶³ suggests a possible explanation of this complicated matter.

The biologic significance of local adaptation or induction is founded on the fact that in the immediate neighborhood of a stimulated zone in the retina the excitability for this form of stimulus is increased.

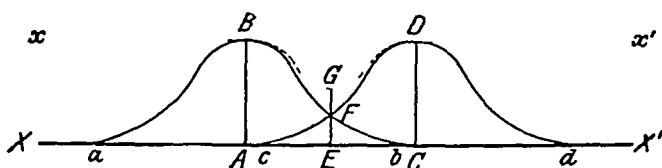


Fig 31—*aBb*, and *cDd* represent the diffuse effect of light on the retina by two adjacent luminous objects (points). The distribution of the intensity of light is indicated by the dotted line *rGr'*. (After F. B. Hofmann³)

Because small movements of the eyes are constantly occurring, even when the gaze is fixed on a point, and because there is still more movement when the eyes are moved, the increased excitability causes a high degree of sensation. It brings about a constancy of sensation which would rapidly change or vanish if an object was looked at fixedly. That this regulative function does not overcome extreme degrees of fatigue may be observed by fixing the gaze on slightly grayish spots on a white paper for two or three minutes, the spots will disappear completely.

A series of observations seems to prove that the function of contrast depends in some degree on general light or dark adaptation, which I shall speak about later.

The observation that in the morning with increasing illumination the contrast between the color of different objects becomes more distinct leads to the conclusion that the dark-adapted eye is less able to produce the phenomenon of contrast than the light-adapted eye. Certainly, that is to a great extent the reason why the dark-adapted eye has a low visual acuity—not more than about one sixtieth of that of the light-adapted eye. It is usually considered that visual acuity is dependent on the anatomic structure alone, but that is too simple an

⁶³ Tschermak, A. Ueber Kontrast und Irradiation, *Ergebn d. Physiol.* 2: 726, 1903.

explanation. It does not take into consideration, for instance, the constant small movements of the eye which are present even when an attempt at exact fixation is made. Nevertheless, it cannot be denied that the anatomic structure is the foundation on which visual acuity is based.

But if this is imputed solely to the anatomic structure of the retina, the explanation seems to be insufficient. Although the fovea centralis has only cones and no rods, and although the rods are probably the sole

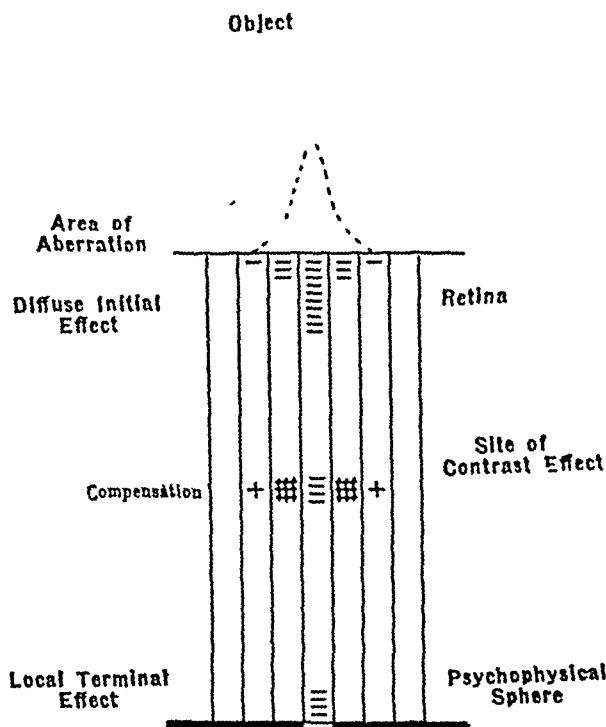


Fig. 32.—Scheme to illustrate formation of contrast in central parts of the retina. A diffuse initial effect is produced in the retina, corresponding to the aberration of light, in conformity with the punctiform object. By contrast effect, in more central parts of the visual pathway the parts of the object which correspond to the less illuminated points of the retina are obscured (indicated by cross hatching). At the same time the place of maximal exposure to light (corresponding to the height of the dotted line) is also reduced in its exciting effect by its surroundings, which are less illuminated. As final effect there arises in the psychophysical sphere a sharp though less bright image, corresponding to the object. (After Tschermak.⁶⁸)

apparatus which functions in the fully dark-adapted eye, the light-adapted eye has near the fovea a visual acuity of about one fifth. The apparatus for dark vision or scotopia has therefore, for some reason, a lower value for visual acuity. Foremost of all, I think that here the phenomena of contrast must be taken into consideration, because the other phenomena of local adaptation are diminished in comparison with the light-adapted eye, e. g., the ability to produce negative after-images

If with the dark-adapted eye one gazes at a white field with low illumination for two or three minutes, one perceives hardly any after-image⁶⁴

On the other hand, a high degree of light adaptation also is not suitable for the manifestation of contrast. As a result, the visual acuity decreases when the general illumination increases, and the resulting light adaptation of the eye exceeds a definite degree. Figure 33, published by Hecht, shows that central visual acuity is dependent on the degree of general illumination. In greater degrees of illumination the function increases to a certain point but finally decreases when the illu-

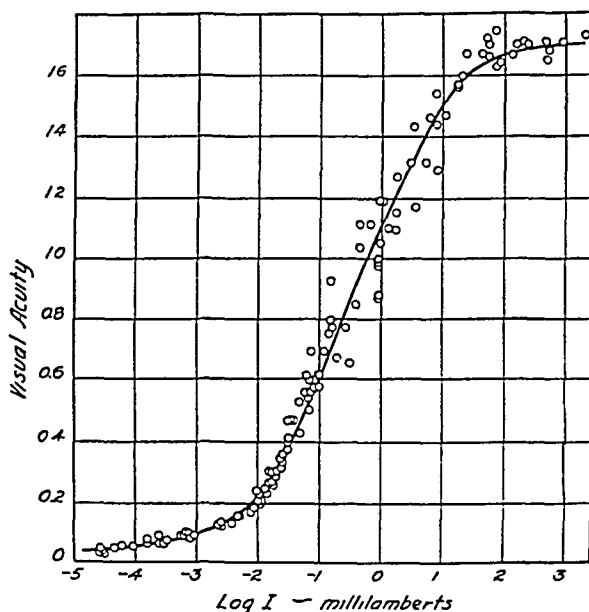


Fig 33—Relation between visual acuity and illumination. The original intensities have been multiplied by 0.072 to convert them into millilamberts (After Hecht, S. Visual Acuity and Illumination, Arch Ophth **57** 564, 1928)

mination begins to dazzle the eyes. An evidence of damage by light is the red vision, or erythropsia, which is produced in an extreme degree by dazzle from sunlight reflected by snow. However, to deal fully with this interesting question would take me too far from the subject.

LOCAL DIFFERENCES IN ADAPTATION

I have already mentioned the phenomena of change in excitability which are less pronounced in the fovea than in the periphery of the retina. This may be due to the fact that the macula lutea is doubly

⁶⁴ Bruckner, A. Untersuchungen zur Dunkeladaptation des menschlichen Auges, reprinted from the report of a joint discussion on vision, Physical and Optical Societies, London, June 1932.

represented in the visual cortex of the brain. The anatomic proof of this was elucidated by Arved Pfeifer⁶⁵. He demonstrated that in the posterior part of the corpus callosum the nerve fibers corresponding to the macula divide and cross from one side to the other. The number of neural elements excited by a stimulus is therefore larger and the sum of the chemical processes greater in degree. Moreover, the sum total of the fibers corresponding to the macula of each eye comprises much more than half of all the fibers for one eye. This proves that the "weight" of the sensation in the sense of the quantity of the underlying process (Hering) is much greater when the macula is stimulated than when a peripheral area is involved. Certainly this favors central vision by producing greater constancy of sensation.

On the other hand, the pronounced local adaptation of the retinal periphery is of no less importance. Some authors attribute to the retinal periphery a special sense for the perception of movement. It is indeed remarkable how acute is perception of small movements, e. g., that of a fly as seen by indirect vision. Biologically this function is of importance because by it one easily recognizes any danger coming from either side or from above. This so-called sense of movement is not a specific one, but is due to quick local adaptation, which enables one to perceive the small differences of brightness dependent on changes in the position of objects.

It would also be of interest to explain the phenomena of contrast from another point of view. According to Hecht's application of the all or nothing law to the eye, each element in the retina has its own threshold, with the increase of light still further elements are stimulated. In the peripheral portion of the retina of the light-adapted and in the slightly dark-adapted eye (therefore in moderate illumination) the number of stimulated elements is relatively small, and therefore, in accordance with this supposition, the intensity of the chemical (or physical) processes induced must generally be smaller than in the completely light-adapted eye in higher illumination. Apparently in the first instance the visual substance must be sooner exhausted or changed in regard to its response to stimulus, which is in accordance with the phenomena of local adaptation.

Measurement of Phenomena of Local Adaptation—All the facts mentioned prove the necessity for an exact measurement of the phenomena of local adaptation, especially that of contrast. This task is very difficult, because through the influence of an illuminated field an increasing change is constantly effected, even though contrast develops

65 Pfeifer, R. A. Myelogenetisch-anatomische Untersuchungen über den zentralen Abschnitt der Sehleitung, in Foerster, O., and Wilmanns, K. Monographien aus dem Gesamtgebiet der Neurologie und Psychiatrie, Berlin, Julius Springer, 1924, no. 43.

momentarily, it increases with the duration of the stimulus. Therefore, not only the difference between the two contrasting fields and their size is decisive, but also the duration of the influence. Some experiments concerning the quantitative measurement of contrast have been carried out by Pretori and Sachs⁶⁶ and some psychologists, but the results seem to be insufficient to allow the deduction of laws therefrom.

Difficulty of exact measurement of the phenomena of local adaptation is the reason that there is insufficient information concerning individual differences of adaptation. The anomalous trichromats are affected in a higher degree by contrast. Therefore, gray spots beside red appear green to them—a phenomenon not seen by any one having normal color sense. Nevertheless, statements made by those with normal color vision show that even among them differences exist.

Individual differences in the development of contrast are perhaps founded on differences in the amount of the hereditary mass, and as a consequence in the amount of the chemical processes in the sensitive substance. The same suggestion holds good as for the differences of local adaptation in the central and peripheral zones of the retina.

PHENOMENON OF CONSTANCY

A further form of change of perception is the phenomenon of constancy, which Hering mentioned long ago. During the last twenty-five years it has been intensively studied by psychologists. Katz and Jaensch⁶⁷ and their pupils especially devoted a great deal of research to this subject.

A white paper of definite brightness in daylight will appear slightly but definitely darker when observed in shadow, but it will still appear to be white. When the brightness of the shaded white is measured, it is seen to be much less than it was in full illumination. With allowance for the illumination or, better, the shade, the sensation is heightened. Therefore, the white paper seems brighter and, allowing for the luminosity, nearer to the sensation perceived when the paper was illuminated by ordinary daylight. The phenomenon is called transformation, because the color is transformed, nearly to the sensation called forth by ordinary conditions.

The change in color produced in this manner is also called albedo color, because albedo signifies the relation of the quantities of light

⁶⁶ Pretori and Sachs. *Messende Untersuchungen des farbigen Simultan-contrastes*, Arch f d ges Physiol **60** 71, 1895.

⁶⁷ See the short abstract by A. Gelb. *Die Farbenkonstanz der Sehdinge*, in Bethe, A., and others. *Handbuch der normalen und pathologischen Physiologie*, Berlin, Julius Springer, 1929, vol. 12, pt. 2, p. 524.

falling on and reflected from a surface. One is said to perceive this physical relation by the phenomenon of constancy.

This phenomenon is always convincing when demonstrated in non-colored illumination or shadow. But it is also seen when the illumination consists of colored light, when one can often perceive the specific colors of the objects, namely the colors perceived in white illumination such as daylight.

These illustrations show how these phenomena of constancy, etc., help in the better orientation of man in his environment in that they stabilize it by producing a constancy of sensation. In this way one recognizes objects more easily than if they called forth different sensations. Naturally, it is necessary to realize the differences of illumination and take them into consideration, although this allowance is not fully conscious. If this were not done, there would be many errors of judgment.

A similar phenomenon of transformation is observed in spatial judgment. In judging the size of an object at a distance, e. g., a man, one takes into account both the distance and the known size of a man and does not regard him as being as small as if his known size were not taken into account.

The position of an object may also be considered. For instance, a piece of wire when held toward one in an inclined position seems to be nearly as long as in a vertical position when one is able to recognize its position. Some observations concerning these facts have been communicated by Gellhorn⁶⁸

All these examples demonstrate how one is able to recognize the same object under different physical or physiologic conditions which should produce different sensations and to acknowledge it as the same object by physiologic or psychologic conversion.

SEAT OF LOCAL ADAPTATION

The question now arises: Where are these processes localized in the visual tract?

The phenomena of constancy or transformation must be bound to higher psychic functions, which are ordinarily called judgments in a larger sense. Here doubt seems to be excluded.

Helmholtz⁶⁹ considered that the genuine contrast phenomena were based on a kind of judgment, but he did not exactly define what he meant by this. But it is difficult to understand how such strictly regulated phenomena can be subject to judgment, since they are also perceived by persons who have no idea of the laws governing them. Therefore,

68 Gellhorn, S. *Beiträge zur Physiologie des optischen Raumsinnes*. II. Ueber die Beziehungen zwischen physiologischer und mathematischer Perspektive, *Arch f d ges Physiol* 208:361, 1925.

69 Helmholtz,¹⁸ p. 250.

one must consider local adaptation as a process physiologically determined

Hering, although he never expressly said so, probably suggested that the local adaptation was localized in the retina. Other authors localized the processes of contrast in the layer of amacrine cells of the retina, because their dendrites provide a connection between neighboring parts of the retina. But there are observations which seem to prove clearly that the phenomena in question are not carried out in the retina alone.

Some writers have noticed that it is possible under certain conditions to see in the monocular field the blindspot, which is itself devoid of sensation. Even in binocular vision some observers are able to see both blindspots. This visibility of the blindspot has been the subject of repeated research. At first the blindspot appears as a dark, well defined disk, often surrounded by a small bright corona, corresponding to sensitive parts of the retina. When colored illumination is used, the blindspot appears in the contrast color. It can also be perceived as a negative white or colored after-image, depending on the nature of the preillumination. In order to facilitate the task it is helpful to make use of a vertical line between a white and a black field falling exactly across the area of the visual field corresponding to the blindspot, as seen in figures 34 and 35.^{70a}

Since in the peripheral organ there is no light-sensitive element corresponding to the blindspot, the fundamental processes producing visibility of the blindspot must lie in a more central part of the optic pathway. Such an induction between adjacent parts seems possible only between nerve cells, not between fibers, and it must be supposed to be situated either in the corpus geniculatum externum or in the visual cortex in the posterior lobe of the brain.

The opportunity to examine 2 men injured in the war enabled me to make some instructive observations concerning this question.⁷¹

A German officer was wounded by a fragment of a shell which severed the optic pathway immediately above the corpus geniculatum externum and produced homonymous hemianopia and other nervous deficiencies. It was possible by illuminating the seeing half of the visual field to produce a sensation in the contrast color in the blind half. If the illumination was white, the blind half appeared black, with colored illumination, for instance blue, the blind half appeared yellow. Negative after-images were produced in this manner according to the well

70 (a) Bruckner, A. Ueber die Sichtbarkeit des blinden Fleckes Arch f d ges Physiol **136** 610, 1910. (b) Tschermak, A. Ueber Merkllichkeit und Unmerklichkeit des blinden Fleckes, Ergebn d Physiol **24** 330, 1925.

71 Bruckner, A. Zur Frage der Lokalisation des Kontrastes und verwandter Erscheinungen in der Sehsinns substance, Ztschr f Augenh **38** 1, 1917.

known laws This observation proves that in this case the phenomenon of local adaptation was localized not in the corpus geniculatum externum but probably in the visual cortex of the posterior lobe of the brain

The second case was that of a young soldier wounded in the back of the head in the visual region He had a defect in the visual field

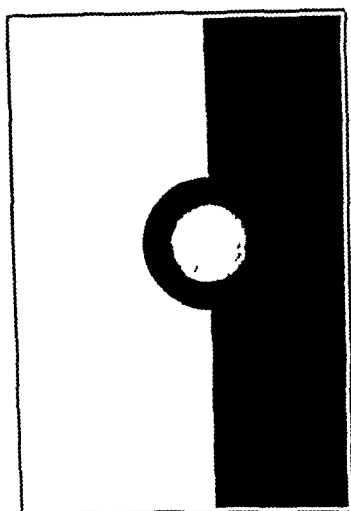


Fig 34—Appearance of the blindspot of the left eye when it falls on the boundary between white and black The center of the blindspot is brightened by contrast (After Bruckner ^{70a})

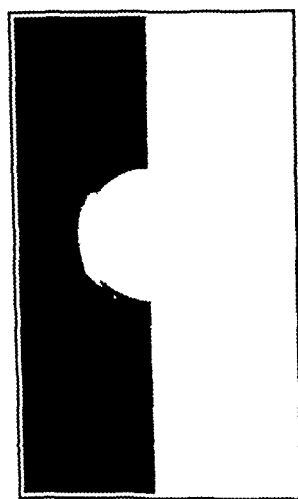


Fig 35—Appearance of the blindspot of the left eye on the boundary between black and white The blindspot appears bright, the center appears darker by contrast In the periphery of the blindspot there are slight appearances of simultaneous contrast within the seeing retina (After Bruckner ^{70a})

of both eyes, the nature and form of which can be seen in the figure 36 It was possible to produce contrast colors in the blind area as in the first case About the particular point of these observations there is no need to speak This observation shows that the phenomena of contrast

seem to be localized in centers lying still higher than the visual cortex, that is to say, in transcortical centers

Of some interest is the fact that both cases demonstrate a reciprocal influence of both hemispheres, because in the hemianopia in the first case and according to the form of the scotoma in the second case both hemispheres must be affected directly or indirectly by contrast. These cases suggest that perhaps there is a transcortical center which fuses the impressions of both visual spheres.

This suggestion falls into line with phenomena of binocular contrast. When one eye is stimulated by a colored field, the other eye sees the contrast color in the corresponding surrounding zone, although not in so distinct a manner as in monocular contrast.

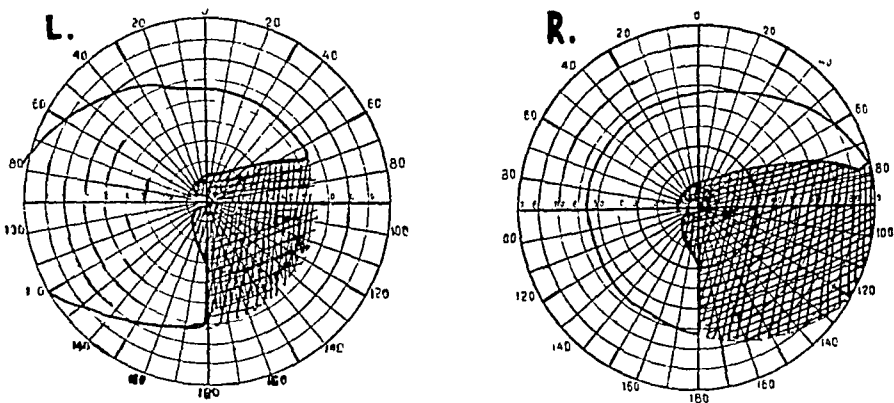


Fig. 36—Defect in visual field of a wounded soldier (After Bruckner⁷¹)

Nevertheless, it seems to me that the centers of the physiologic processes on which the phenomena of local adaptation and contrast are localized are probably situated in all stations in the visual pathway. As Jaensch has suggested, the different stations probably influence each other.

All this shows that nature strives to unite the whole visual system and to guarantee thereby the constancy of visual reception.

With respect to the question of the localization of the phenomena of local adaptation some recent investigations are of interest, especially those dealing with the change of the electric behavior under the influence of light. They show a constant current which in the eye of the vertebrate is directed in the outer part of the circuit from the cornea to the posterior pole of the eye. This so-called duration current is subject to characteristic changes when the eye is illuminated.

Recently so-called electroretinograms of the human eye have been made by Sachs⁷². Figure 37 demonstrates some of his observations⁷³.

⁷² Sachs, E. Die Aktionsströme des menschlichen Auges, ihre Beziehung zu Reiz und Empfindung, *Klin. Wchnschr.* 8 136, 1929.

⁷³ Kohlrausch,²³ p. 149.

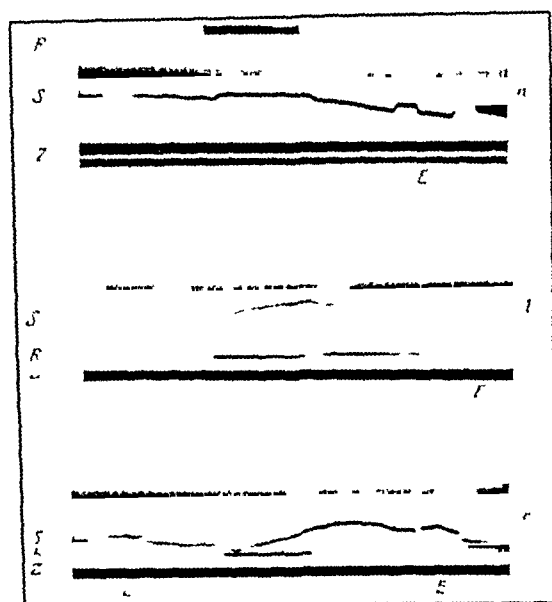


Fig 37—Electroretinogram of a man adaptation state and secondary elevation on red stimulation and blue stimulation, specific color effect? *a*, dark adaptation, red, 2 lux, 10° visual field, fixed centrally, *b*, light adaptation, red, 5 lux, 10° visual field, fixed 14° peripherally, *c*, dark adaptation, blue, 1 lux, 10° visual field, fixed centrally *R* indicates photographic or electromagnetic marking of stimulus, *S*, string, *Z*, time in one-fifth second intervals, *E*, gaging with 0.1 millivolt, by string, electrodes and eye (Original curves after E Sachs,⁷² reduced to one third of the original size)

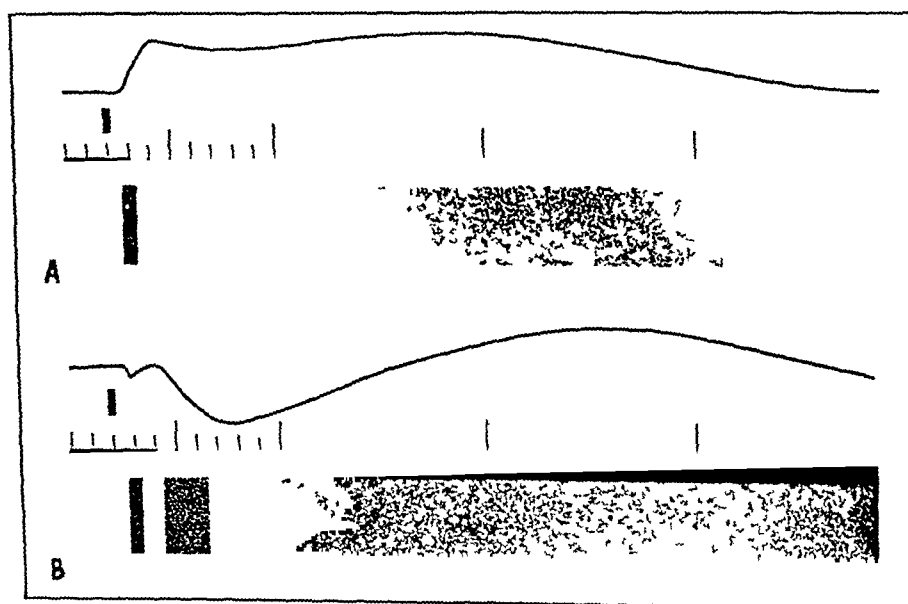


Fig 38—Parallelism between the retinal currents of the hematothermal species and the periodic visual sensations of man *A*, momentary illumination with red of very long waves, *B*, with blue Corresponding retinal current and visual sensation are shown one above the other, since the actual time interval is not accurately known Time in seconds, the first second divided in tenths (After Kohlrausch²³)

Red light produces in the dark-adapted eye a slight oscillation, which is in accordance with the feeble stimulating value of red light on the dark apparatus of the eye. In the light-adapted eye the curve runs in another direction. Illumination with blue light on the dark-adapted eye caused a remarkable oscillation, in accordance with the brightness of the sensation from blue light in the eye. These results agree with those obtained from experiments on the eyes of animals.

As another type of the action current corresponding to the periodic course of the after-image I show figure 38, published by Kohliusch⁷³

Probably the quantitative differentiation of the stimulating processes is already effected in the peripheral organ. In the continuation of the optic pathway the different processes in the retina are represented by different frequencies of the electric current, as shown by Adrian and Kohliusch. The different color sensations run parallel to those in the central organ. This opinion was expressed by Frohlich⁷⁴

When it is remembered that electric phenomena are perhaps only accompaniments of exciting chemical processes of an antagonistic character, the explanation of local adaptation, given before, becomes still more evident. All parts or stations of the optic pathway interfere with this phenomenon.

74 Frohlich, F. W. Die Empfindungszeit, Jena, Gustav Fischer, 1929

(To Be Concluded)

CONGENITAL BUPHTHALMOS

COMPLICATED BY DISLOCATION OF LENS AND HEMORRHAGE INTO
VITREOUS WITH COMPLETE RECOVERY OF CENTRAL VISION

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The following case is reported because of the favorable result that followed a severe injury and a stormy course in the only buphthalmic eye of a patient

History—D L, a white man aged 31, of Italian extraction, entered the ophthalmologic service of the Cook County Hospital on April 4, 1936. He gave a history of having been struck in the right eye by a finger fifteen hours previously. After the injury he became totally blind in his right eye.

When the patient entered grammar school it was found that both of his eyes were larger than normal and that he had no vision in his left eye. The diagnosis was said to be bilateral congenital glaucoma. In 1911 a Lagrange type of operation was performed at the usual location on the right eye by Dr. Ben Witt Key, of New York. After this, the patient regained sufficient vision to finish the grades. He wore glasses for seven years after the operation, but discarded them in 1918. In 1933, owing to repeated attacks of severe pain, the left eye was enucleated. Until the recent injury, he was able to read the newspapers and see motion pictures. His general health had always been good, and there was no history of ocular disease in his family.

Ophthalmologic Examination—Vision in the right eye was limited to perception of light with faulty projection. Vision in the left eye was nil (a prosthesis was worn).

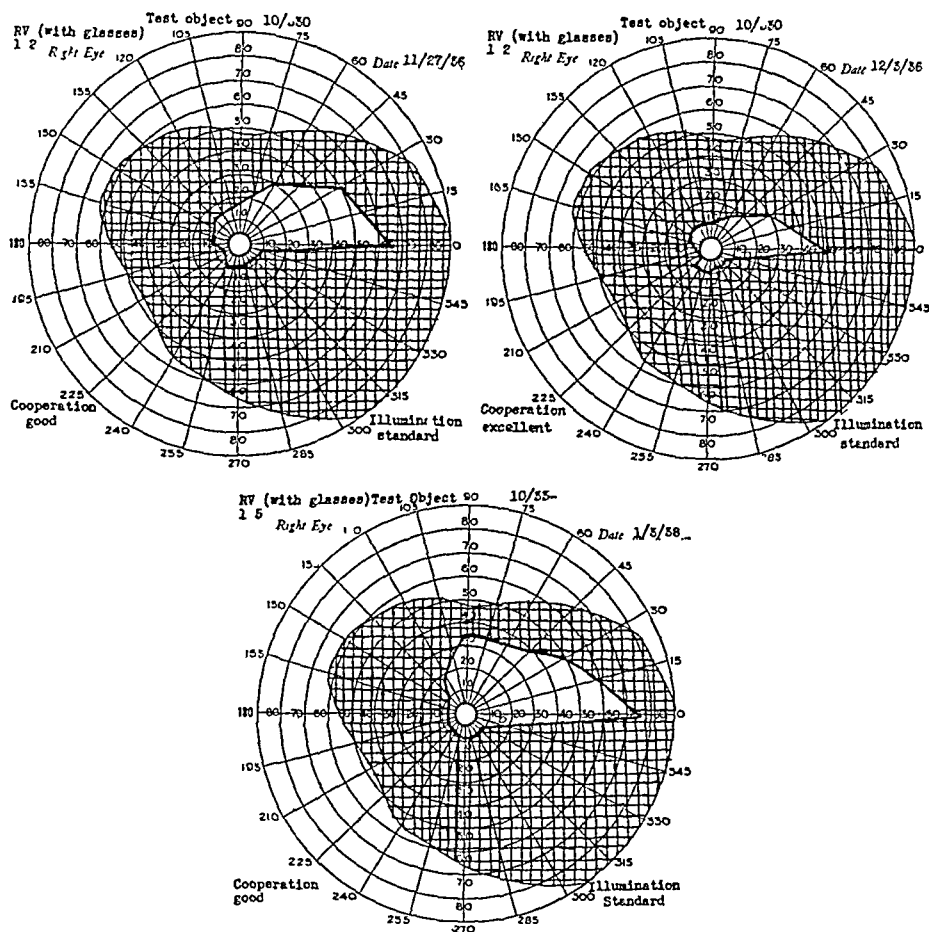
Both lids of the right eye were markedly ecchymotic. The external ocular movements were normal. Recent subconjunctival hemorrhages were present on the nasal and inferior quadrants of the bulb. The sclera was uniformly distended and appeared bluish. The cornea appeared globular and measured 17 mm in the transverse diameter. No ulcerations or opacities were seen on its surface. The anterior chamber was filled with blood. A red fundus reflex could not be elicited. The tactile tension was normal.

The stump of the left eye was smooth and even and appeared somewhat retracted. The conjunctival cul-de-sac was narrowed, only limited movements of the prosthesis were present.

Physical Examination—The patient was well developed and well nourished and did not appear acutely ill. The skin presented diffusely scattered patches of vitiligo. The nose and ears were normal. The teeth showed evidence of decay in the region of the upper right and lower left molars. The tonsils were enlarged, and cheesy material exuded from their crypts on pressure on the pillars. The thyroid gland was not palpable, and no glands or masses were felt in the neck. The blood pressure was 126 systolic and 84 diastolic. The heart, lungs, abdomen and extremities were normal.

Laboratory Examination—The urine, the blood count, the blood chemistry, the bleeding time and the coagulation time were normal. Intradermal tuberculin tests gave negative results. The basal metabolic rate was -6 . Roentgenograms of the chest and skull revealed no abnormal changes.

Diagnosis—The condition was diagnosed as (1) traumatic intraocular hemorrhage involving the right anterior chamber and possibly the deeper structures of the right eye and (2) congenital buphthalmos.



Visual field of patient taken on three different dates

Treatment and Course—The patient was put at absolute rest in bed. Hot applications and an ointment containing 1 per cent atropine sulfate were applied three times daily to the right eye. Four weeks later, with the foregoing regimen, the external swelling and redness had largely abated. The subconjunctival swelling and hemorrhages had disappeared. The anterior chamber appeared deep and was free from blood. The iris was tremulous, dull and lusterless. A coloboma of the pupil was present between 11 and 1 o'clock. A dark black mass was seen occupying the pupillary area. The tactile tension remained normal.

On June 2 a red fundus reflex was first obtained, and the vision had improved to ability to count fingers at 2 feet (60 cm). The tension was 14 mm of mercury (Schiotz).

On June 17 the vitreous had cleared sufficiently to permit a hazy view of the disk and the retinal vessels. A mass of grayish white tissue about 2 by 5 by 8 mm was noted in the vitreous. This mass was firmly adherent to the ciliary body at a point about 2 mm in front of the ora serrata at 3 o'clock. From this point of attachment the mass was observed moving backward and forward in harmony with movements of the eye, alternately obscuring and clearing the pupillary space.

On October 26, with a +10.00 sphere combined with a +2.00 cylinder, axis 5, the vision in the right eye had improved to 12-3. Studies of the peripheral visual field revealed an almost complete loss of the nasal field associated with a marked defect in the inferior temporal quadrant. A faint circumciliary flush was present. The white mass in the vitreous remained unchanged. The optic disk and retinal vessels were clearly visible. The nerve head appeared pale. The vessels of the disk were pushed nasalward, and a slight glaucomatous excavation was present. The tension was 18 mm of mercury (Schiotz). Three carious teeth were extracted and the daily administration of atropine sulfate was discontinued.

On November 17 the patient complained of pain in his right eye. The eyeball felt hard to touch and still appeared irritated. The tension was 28 mm of mercury (Schiotz). The visual fields exhibited a further concentric contraction in all meridians. A mixture of 2 per cent pilocarpine hydrochloride and 0.5 per cent physostigmine salicylate administered three times daily brought about only a temporary reduction in tension. On withdrawal of the miotic, an immediate rise to 38 and 45 mm of mercury followed. Epinephrine packs (1:1,000) under the upper lid had only a temporary effect in lowering the tension.

On Jan. 27, 1937, tonsillectomy was performed. After this operation the patient's general physical condition improved greatly and he gained 12 pounds (5.4 Kg.) of weight within the next four weeks.

On February 17 a +10.50 sphere combined with a +4.00 cylinder, axis 180 (for near vision, addition of a +3.50 sphere), improved the vision of the right eye to 15 with ability to read Jaeger test type 1 at 13 inches (33 cm). The tonometric readings had dropped to 13 mm of mercury and remained within normal limits since then without the use of miotics. The patient was discharged.

He was not seen again until Jan. 2, 1938. At that time the vision in his right eye with glasses was 15 with ability to read Jaeger test type 1 at 13 inches. The tension was 13 mm of mercury (Schiotz). The visual field had increased 10 degrees in the temporal and 15 degrees in the superior meridian. The globe was pale. The white mass in the vitreous appeared as described previously, but seemed thinner, resembling a veil. The retinal veins were dilated. The disk was pale, but showed only a slight glaucomatous excavation.

COMMENT

The buphthalmic eye, owing to its prominence and greatly thinned scleral coat, is more prone to injury than the normal eye. The suspensory ligament is overstretched. Rupture of the zonule leading to dislocation of the lens frequently follows even the slightest traumatism.

In the case reported, at the time of the injury a backward dislocation of the lens took place. This was obscured by the associated massive hemorrhage into the anterior chamber and the vitreous. The freely swinging white mass just back of the ciliary region, observed at a later date, was attached by part of the torn suspensory ligament and

undoubtedly contained shrunken remnants of lens material covered by connective tissue and exudate

The transient rise of tension and the persistence of pericorneal irritation disappeared on improvement of the patient's general physical status and the elimination of all frank foci of infection. The drop in tension, however, might have occurred without tonsillectomy, as there were no signs of uveitis as a cause for the increase in intraocular pressure. The visual fields were characteristic of glaucoma. The entire nasal field and a sector-shaped defect in the inferior temporal field were lost. The optic nerve showed evidence of atrophy, with a slight excavation of the lamina cribrosa. These changes most likely anteceded the recent disturbance and were presumably present at the time the patient first presented himself for treatment.

The remarkable final central visual acuity, the maintenance of normal intraocular tension and the absence of any further constriction of the visual fields over a period of a year offer strong supportive evidence of the efficacy of an early and properly performed drainage operation in a case of congenital glaucoma.

106 Market Street

CIRCULATION OF THE AQUEOUS

VII A MECHANISM OF SECRETION OF THE INTRAOCULAR FLUID

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AND

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In the course of an investigation on the mechanism of reabsorption of the intraocular fluid¹ it was found that the ciliary body shows an irreciprocal permeability to water, allowing fluid to pass with ease from the ciliary capillaries to the posterior chamber but obstructing the passage of fluid in the reverse direction. Phenomena of this type have been demonstrated in many other tissues². Hitherto, however, no adequate physical explanation of such phenomena has been suggested. The present investigation was undertaken with the aim of discovering, if possible, the character and mechanism of the irreciprocal permeability of the ciliary body. In brief outline, it was first discovered that the ciliary body shows an irreciprocal permeability to certain dyestuffs as well as to water and that the behavior of the dyes indicates that an electrical phenomenon is involved. This led us to an investigation of the source of the energy required to perform the physical work involved in the irreciprocal permeability and finally to an analysis of the components of the mechanism by which this energy was transformed into physical work. In the end it was found that an adequate understanding of the mechanism of irreciprocal permeability to dyestuffs could account also for the irreciprocal permeability to water.

It will be seen that the investigation which we are reporting is a complex one involving several different types of experiments. In order to facilitate the continuity of the argument and to avoid confusion from

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital

This investigation was supported in part by a grant from the John and Mary Markle Foundation

1 Friedenwald, J S, and Pierce, H F. Circulation of the Aqueous. II Mechanism of Reabsorption of Fluid, *Arch Ophth* 8 9 (July) 1932, *Tr Am Ophth Soc* 31 143, 1933

2 Gellhorn, E. Das Permeabilitätsproblem. Seine physiologische und allgemein-pathologische Bedeutung, in Gildemeister and others. *Monographien aus dem Gesamtgebiet der Physiologie der Pflanzen und der Tiere*, Berlin, Julius Springer, 1929, vol 16

details of experimental technic, the experimental protocols appear in small type and the text in large type, the two portions being so arranged that they can be read continuously. A summary is inserted at the end of each section.

I PERMEABILITY OF THE CILIARY BODY TO VARIOUS ACID AND BASIC DYES³

Most previous investigators⁴ on the permeability of the blood-aqueous barrier have used as their criterion the appearance of the dye in the aqueous humor after intravascular injection. They have shown that most acid crystalloid dyes appear in the fluid while basic dyes do not. These investigations give no clue as to whether the dye has entered the ocular cavity by way of the ciliary body or through some other tissue, e. g., the iris. As long ago as 1882 Ehrlich⁵ showed that fluorescein enters the anterior chamber by way of the iris, and subsequent studies by Hamburger⁶ demonstrated that the contribution of the ciliary body to the intraocular diffusion of fluorescein was probably insignificant. We have studied the passage of dyes through the ciliary body directly, observing their movement in the direction from blood to aqueous and vice versa. The experiments were conducted as follows:⁷

To study the passage of dyes from the blood to the intraocular cavity, the dye (from 0.04 to 0.2 millimols per kilogram) was injected intravascularly into an albino rabbit. After the injection, the eye was enucleated with the animal under ether anesthesia, it was opened equatorially, and the vitreous and lens were removed with gentle pressure of the thumb of one hand while the inverted eyeball

3 The physical and chemical characteristics of the dyes used in all of the experiments reported in this paper are described in the Appendix.

4 Gaedertz, A., and Wittgenstein, A. *Arch f Ophth* **119** 395 and 403, 1927, **119** 755, 1928. Fischer, F. P. *Arch f Augenh* **100-101** 480, 1929.

5 Ehrlich, P. *Berl klin Wchnschr* **19** 388, 1882.

6 Hamburger, C. *Ueber die Ernährung des Auges*, Leipzig, Georg Thieme, 1924.

7 In preliminary experiments attempts were made to use the frozen section technic. It was found that freezing the tissue caused a destruction or disturbance of its normal metabolism and led to erroneous and contradictory results with the dyes. On the other hand, if the living tissues were stained with the dyes the penetration of which was to be tested, and then fixed, the dye washed out of the tissue, especially from the epithelial layers. This led to an erroneous conclusion stated in a previous communication,¹ namely, that methylene blue injected into the vitreous does not stain the ciliary body, including the epithelium. Actually, most basic dyes, including methylene blue, injected into the vitreous stain the epithelium but not the stroma. Obviously, therefore, it was necessary to use and observe the dye directly on surviving tissue. It was found that such tissue kept in Ringer's solution could be used for from two to four hours after killing the animal or enucleating the eye. Temperature variations between 20 and 40 C. seemed to have little or no effect. Changes of short duration in the oxygen tension from 0 to 150 mm. of mercury did not affect the tissue irreversibly.

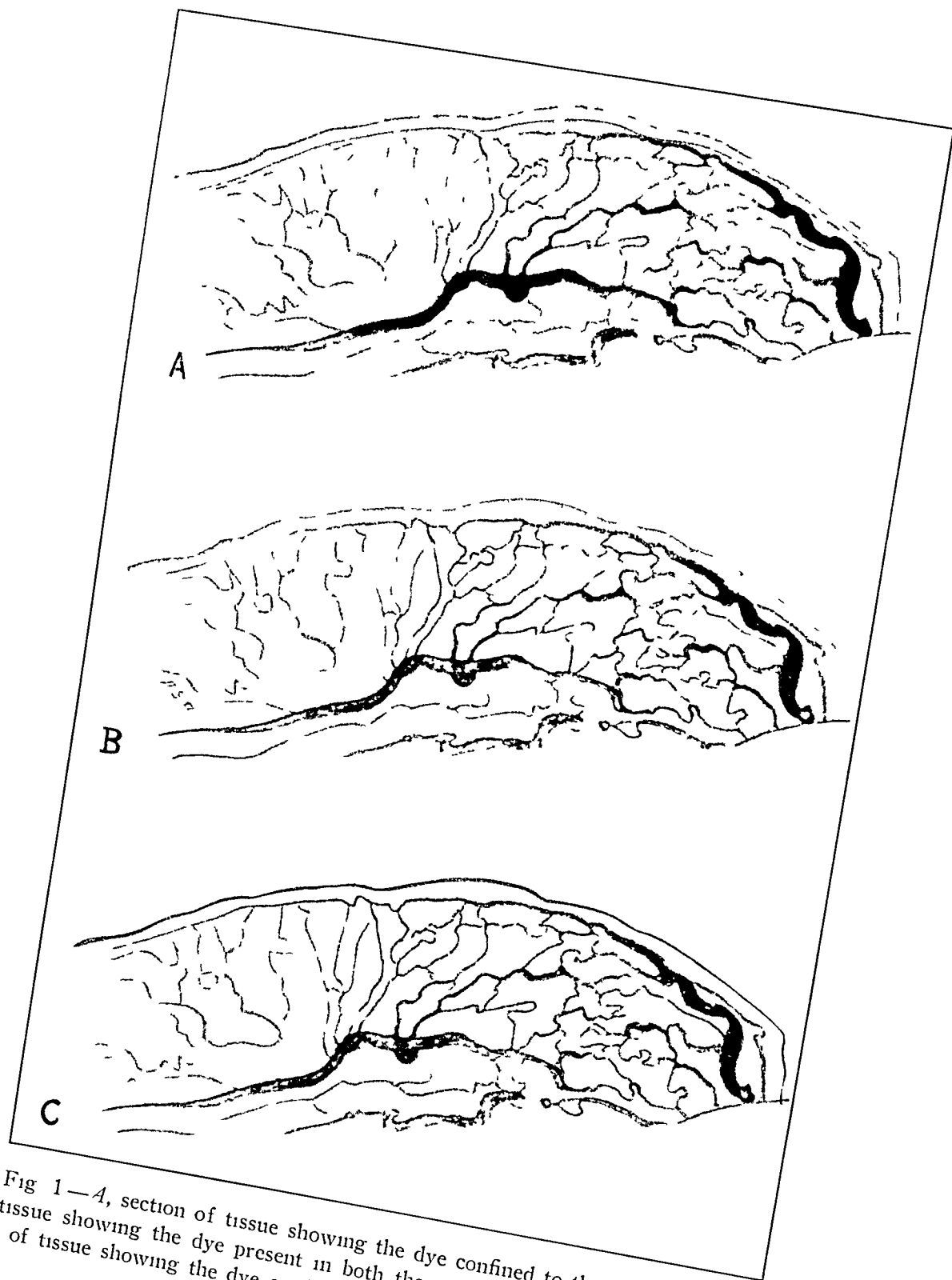


Fig 1—*A*, section of tissue showing the dye confined to the stroma *B*, section of tissue showing the dye present in both the epithelium and the stroma *C*, section of tissue showing the dye confined to the epithelium

was held between the thumb and the index finger of the other hand. The zonular fibers were examined for the presence of the dye. The iris and ciliary body were then removed in one piece with a tissue forceps and placed in a special Ringer's solution⁸. The ring of tissue was placed on a microscope slide with the ciliary processes upward and examined under a microscope (16 or 8 mm objective).

When the dye fails to penetrate from the stroma to the epithelium the picture is produced that is shown in figure 1 *A*. The interpretation is sometimes difficult because the epithelium on the anterior tips of the ciliary processes may be so thin as to escape observation with the low power of the microscope. Certain acid dyes stain the epithelium feebly even when introduced through its free surface, and when penetrating from the stroma to the epithelium they may rapidly diffuse out of the latter tissue into the surrounding fluid, leaving the epithelium relatively colorless. The distinction between the picture under these circumstances and that shown in figure 1 *A* is that the margin of the stained area in the stroma is blurred instead of being sharp. This difficulty was encountered particularly in the asphyxia and p_n experiments cited later.

To study the passage of dyes from the intraocular cavity to blood, the eye of an albino rabbit was removed and dissected in the manner previously described. The ring of tissue was cut into from ten to twenty sectors in a bath of Ringer's solution. These sectors were placed on slides (ciliary processes upward), and a ciliary process was lapped over the cut edge with the aid of a camel's hair brush (fig. 2). The excess fluid was drained off with blotting paper, and a minute drop (not more than 1 cu. mm.) of dye solution (approximately 0.001 molar) was placed on the moist surface of the glass next to the tissue. As soon as the vertical edge of epithelium was stained, the excess of dye was drained with blotting paper, and a few drops of Ringer's solution added. The tissue was observed under a microscope to determine the penetration of the dye.

One of the difficulties in determining whether or not the dye has penetrated arises from a spreading or creeping of the dye in the epithelium. This may happen either because the dye runs over the horizontal surface of the ciliary process in the moment of staining or as the result of slow diffusion of the dye from cell to cell after the staining solution has been removed. Two criteria are available to distinguish between such spreading of the stain and penetration, as illustrated in figure 1 *B*. 1. With critical focusing of the microscope, a sharp epithelium-stroma boundary, as in figure 1 *C*, is still to be recognized in case of nonpenetration.

8 The Ringer's solution referred to throughout this paper is that used by Dr. George O. Gey for tissue culture work. It contains per liter of solution: sodium chloride, 8 Gm.; potassium chloride, 0.37 Gm.; calcium chloride (anhydrous), 0.17 Gm.; magnesium chloride (anhydrous), 0.10 Gm.; disodium phosphate, 0.18 Gm.; sodium bicarbonate, 0.25 Gm.; carbon dioxide is added until the solution is completed. This solution was chosen after it had been found that experiments performed in an isotonic solution of sodium chloride gave erratic results. Tests were made to discover the effect of ions normally present in plasma. It was found that Ringer's solution, lacking only in calcium, caused the permeability of the membrane to increase, while 0.9 per cent sodium chloride plus a trace of calcium was adequate to maintain the normal permeability. The minimum amount of calcium required was found to be less than one-tenth that normally present in the body fluids. It is evident, therefore, that clinically possible variations in blood calcium cannot affect the membrane significantly. Further tests showed that even a considerable excess of magnesium could not be substituted for calcium.

2 The dye fades more rapidly in penetration than in creeping, since it diffuses into a larger volume in the former case than in the latter. In long-continued experiments dyes which fail to penetrate into the stroma are sometimes bleached out of the superficial layer of epithelium, leaving the second layer of epithelium holding most of the remaining dye. In general, basic dyes stain the tissues more intensely than acid dyes. This difference is not as marked at lower pH .

Experiments were also performed in which the dye solution was injected directly into the stroma of the ciliary body either before or after enucleation. In other experiments the dye was injected directly into the vitreous. Identical results were obtained in regard to the penetration of dyes introduced either *in vivo* or *supravivally*.

Acid dyes (dyes which are anions in solution) introduced into the stroma of the ciliary body produce the picture illustrated in figure 1 *A*. The dye fills the stroma and does not penetrate into the epithelium. Acid

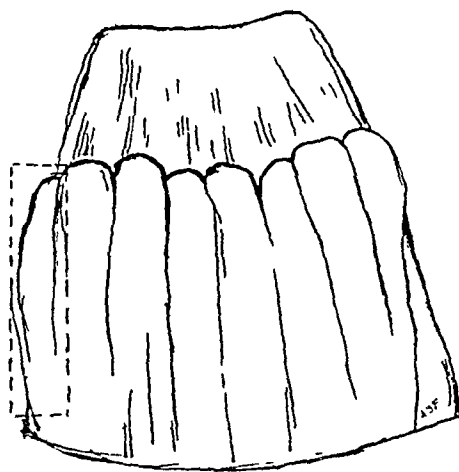


Fig 2—A segment of the iris and ciliary body viewed from behind. The overlapping ciliary process is indicated by the dotted lines.

dyes introduced into the epithelium produce the picture illustrated in figure 1 *B*. The dye penetrates into the stroma. If only a small amount of dye is used, the epithelium becomes colorless after a time, leaving a picture similar to that in figure 1 *A*. Basic dyes (dyes which are cations in solution) introduced into the epithelium fail to penetrate into the stroma, as illustrated in figure 1 *C*. Basic dyes introduced into the stroma penetrate rapidly into the epithelium, and if the concentration is not too high, the dye accumulates in the epithelium leaving the stroma relatively colorless, as in figure 1 *C*. Neutral dyes (dyes which are either uncharged or zwitterions in solution) penetrate with ease in both directions, as illustrated in figure 1 *B*.

The results with certain typical dyes which are not reduced by the tissue in air are summarized in table 1. The behavior of the other dyes which were used is reported in the Appendix. It can readily be seen

that the behavior of the dyes depends essentially on their ionic charge and not on their chemical structure. When similar experiments are performed on tissue asphyxiated either in an atmosphere of nitrogen or in the presence of 0.001 molar cyanide solution, it is found that the irreciprocal permeability to these dyestuffs disappears. Any particular dye now penetrates equally well from the epithelium to the stroma and from the stroma to the epithelium, and the phenomenon of accumulation disappears. However, a marked difference between the behavior of acid and basic dyes is to be noted, for now acid dyes penetrate rapidly and with ease, while basic dyes penetrate with great difficulty or not at all. This change in permeability produced by asphyxia was found in the case of nitrogen asphyxia to be reversible, the tissue regaining its normal

TABLE 1—*Penetration of Dyes Through Stroma-Epithelium Barrier of Ciliary Body at Physiologic p_n*

Dye	Type	Penetration Normally		Penetration with Asphyxia	
		E to S	S to E	E to S	S to E
Crystal violet	Basic	0	++++	±	±
Malachite green	Basic	0	++++	±	±
Night blue	Basic	0	+++	0	0
Safranines	Basic	0	+++	0	0*
Eosin	Acid	+++	±	++	++
Bromphenol blue	Acid	+++	±	++	++
Rose bengal	Acid	+++	0	+	+
Rhodamine B	Neutral	++	++	++	++

0 indicates no penetration, ±, a trace of penetration, +, slow diffusion, ++, rapid diffusion, +++, slow accumulation beyond diffusion, +++++, rapid accumulation beyond diffusion. E, epithelium, and S, stroma.

* No penetration under cyanide, partially reduced under nitrogen.

characteristics when restored to air. Recovery from cyanide asphyxia has also been noted under certain conditions (see section III).

We have investigated in some detail the exact position of the barrier at which acid dyes are stopped from penetrating into the epithelium and basic dyes are stopped from penetrating into the stroma and have found that this barrier lies in the same region in each case, at the junction of the epithelium and the stroma. The penetration of basic dyes under asphyxia is blocked at the same place. [Histologic study of this region reveals a forward continuation of Bruch's membrane which is visible in the flat posterior part of the ciliary body, becoming thinner and thinner as one follows it forward and being lost from view in the anterior portions of the ciliary body. We have not, however, been able to identify the stroma-epithelium barrier with this forward continuation of Bruch's membrane. Until more detailed knowledge is available, we shall refer to the barrier as the stroma-epithelium barrier. Further experiments on the nature of this barrier are reported here.]

✓ *Summary*—There is a barrier between the stroma and the epithelium of the ciliary body which under asphyxia exhibits a selective permeability in favor of acid as against basic dyes. When the normal respiratory metabolism is operating, however, additional forces are active which facilitate the transfer of acid dyes from the epithelium to the stroma and of basic dyes from the stroma to the epithelium and impede the transfer of these dyes in the reverse direction. It is evident that these additional forces could be accounted for if there were in the ciliary body an ionic electric current carrying anions from the epithelium to the stroma and cations from the stroma to the epithelium. However, in order for an electric current to exist, there must be a source of energy and a carrier of electrons to complete the circuit. Since the phenomena the cause of which we are seeking to discover disappear under asphyxia, we have sought for the source of the energy in the respiratory metabolism of the tissue, which is the next step in our study.

II OXIDATION-REDUCTION POTENTIALS OF THE CILIARY BODY

Since the behavior of dyes described and the assumptions of an electric current indicated that there must be a difference of free energy in the stroma-epithelium system, it was natural to look for this energy in the oxidative and reductive processes of the tissue. Clark and his co-workers⁹ have shown that the intensity of oxidation-reduction activity of chemical systems that are capable of reversible oxidation may be measured in terms of electrical potential and that, in fact, chemical systems of this type, if properly connected by electrodes, are capable of generating an electric current. The potential of such systems may be measured directly electrically, but it may also be measured indirectly through the use of indicators which undergo a color change on oxidation or reduction. These investigators have prepared and studied a series of such indicators which may be used to determine the oxidation-reduction potential of systems by methods analogous to those by which p_H is determined through the use of p_H indicators. These studies refer to systems that are in thermodynamic equilibrium.

In the living tissues thermodynamic equilibrium does not exist. Instead there is a continuous interaction between many chemical systems. When these reactions proceed at constant rates, a steady state is established by a balance between the rates of oxidation and of reduction, which can be measured either potentiometrically or with oxidation-reduction indicators. The objections to the electrometric method need

⁹ Studies on Oxidation-Reduction I-X, By the Staff of the Division of Chemistry, Hygienic Laboratory, United States Public Health Service, Hygienic Laboratory Bulletin 151, United States Public Health Service, 1928.

not be enumerated here¹⁰ The indicator method was used in the present study because of its directness and relative ease and because it enables one to determine the potential in all parts of the tissue at the same time This involves some lack of precision, since the degree of bleaching of the various indicators can be estimated only roughly Other sources of error with the indicator method are discussed by Cohen, Chambers and Reznikoff¹¹ Nevertheless, given appropriate indicators, we believe that the potential of tissues can be determined with an error of less than ± 0.015 volt The experiments were performed as follows

TABLE 2—Oxidation-Reduction Potentials of the Ciliary Body

Indicator	E_o' at pH 7.4 Volts	Reduction of Indicator					
		Aerobic		Cyanide S	Anoxia E	Anaerobic	
		S	E			S	L
Binderschedler's green	+0.211	++++	++++	++++	++++	+++-	++++
2,6-dichloroindophenol	+0.189	++++	++++	++++	++++	+++-	++++
Toluylene blue	+0.101	++++	++	++++	++++	+++-	++++
Lauth's violet	+0.050	++++	0	++++	++++	+++-	++++
Cresyl blue	+0.034	++++	0	++++	+++-	+++-	++++
Methylene blue	-0.002	++++	0	++++	+++-	+++-	++++
Indigo trisulfonate	-0.099	+++	0	++++	++++	+++-	++++
Indigo disulfonate	-0.143	+	0	++++	++++	+++-	++++
Cresyl violet	-0.175*	0	0	+++	++-	+++-	++++
Dimethylphenosafranine	-0.275	0	0	0	0	++-	++-
Safranine T	-0.298	0	0	0	0	—	+
Estimated potential volts		-0.130	+0.100	-0.200	-0.200	-0.250	-0.290

0 indicates no reduction, +, one third reduced, ++, one half to two thirds reduced, +++, one third to nine tenths reduced, +++++, over nine tenths reduced, S, stroma, and E, epithelium

* The question in regard to the potential of cresyl violet arises from a lack of certainty as to whether the dye which we used was identical with that studied by Rapkine, Struck and Wurmser (*J. Chim. Phys.* **26**: 340, 1929)

The test indicator is introduced into the stroma or epithelium of the ciliary body and allowed to come to equilibrium with the tissues. The degree of bleaching which takes place is observed under the microscope. Subsequently, an oxidizing agent, such as ferricyanide, is added, and the recovery of color is noted. The ratio of intensity of color before and after oxidation with ferricyanide affords a measure of the ratio of oxidant to reductant of the indicator in equilibrium with the tissue, and hence of the potential within the tissue. The recovery of color on oxidation is a necessary control demonstrating that the indicator has been reversibly reduced and not irreversibly destroyed. Experiments were conducted both aerobically and under conditions of asphyxia.

The results of these experiments are summarized in table 2. It can be seen that the potential in the epithelium in air is +0.100 volt, while that of the stroma in air is -0.130 volt, giving a difference of 0.230

¹⁰ Chambers, R. An Analysis of Oxidation and Reduction of Indicators in Living Cells, in Cold Spring Harbor Symposia on Quantitative Biology, Cold Spring Harbor, L. I., New York, The Biological Laboratory, 1933, vol. 1, p. 205.

¹¹ Cohen, B., Chambers, R., and Reznikoff, P. *J. Gen. Physiol.* **11**: 585, 1928.

volt between the potentials of the two tissues Under nitrogen both epithelium and stroma have the same potential, -0.290 volt In the presence of 0.001 molar cyanide and air, the epithelium and stroma both have a potential of -0.200 volt

Summary—There is a difference in potential between the epithelium and the stroma (disappearing under asphyxia) which is capable of supplying the free energy for the electric current mentioned previously In order to do so, the two parts of the tissue must be coupled by an electron carrier capable of accepting electrons from the stroma and transferring them to the epithelium Before considering this electron carrier, we shall report briefly experiments relating to the enzyme systems involved in maintaining the difference in potential between the epithelium and the stroma

III THE ENZYME SYSTEMS

Since the metabolites of the stroma and epithelium are in all probability identical, the cause of the difference in potential between these two tissues is to be sought for in their enzyme systems

A OXIDASES

Seidel,¹² Schmelzer¹³ and Samojloff¹⁴ have shown that the ciliary epithelium gives an intense indophenol-oxidase reaction, whereas the stroma gives no reaction We have confirmed and extended these observations The technic is as follows

A piece of tissue is placed on a microscope slide as described before and covered with about 1 cc of Ringer's or buffer solution This allows an easy access of oxygen for the reaction To the bathing solution is added an equal volume of a freshly prepared solution of dimethylparaphenylenediamine and alphanaphthol (each 0.01 molar) in Ringer's solution The development of the color of indophenol blue is observed under the microscope

In the experiments the epithelium becomes intensely stained with indophenol blue while the stroma remains unstained This indicates that indophenol-oxidase (Warburg's respiratory enzyme¹⁵) is present only in the epithelium and is absent in the stroma The test for indophenol-oxidase is not a quantitative one, but gross changes in the activity of the enzyme manifest themselves in easily detectable changes in the rate of development of the color reaction If the tissue is poisoned with cyanide, no color develops in the test though the reagent is not itself

12 Seidel Ber u d Versamml d ophth Gesellsch **45** 14, 1925

13 Schmelzer Ber u d Versamml d ophth Gesellsch **45** 259, 1925

14 Samojloff, A J Arch f Ophth **118** 391, 1927

15 Warburg, O H Ueber die katalytischen Wirkungen der lebendigen Substanz, Berlin, Julius Springer, 1928

affected by cyanide¹⁶ Since the reagent is likewise unaffected by moderate changes in p_H , it is possible to use this test as an indication of the effect of p_H on the activity of the enzyme The results of these experiments are shown in table 3 It is evident that a moderate increase in alkalinity over the normal physiologic p_H does not affect the activity of the enzyme, but that at p_H 6.9 the activity is reduced, while at p_H 6.2 it is almost entirely suppressed

In addition to the indophenol-oxidase, we must consider other respiratory systems capable of reacting directly with oxygen As noted in table 2, the potential of the tissue is 0.090 volt more negative under nitrogen asphyxia than it is under cyanide asphyxia in air This indicates the presence in the tissue of substances other than indophenol-oxidase, capable of reacting directly with oxygen However, these substances must be equal in effect in the epithelium and the stroma, since the potential under asphyxia is the same in both tissues

TABLE 3—*Variation of Indophenol-Oxidase Activity with p_H*

p_H	Pyridine-Pyridine Acetate Buffers					Ringer's Solution
	5.70	6.17	6.55	6.90	8.32	
Reaction	$\pm ?$	\pm	+	++	++++	++++

B DEHYDROGENASES

In order that the postulated electric current can do a significant amount of work, the stroma must have sufficient reducing capacity This capacity is determined by the substrates and the enzymes (dehydrogenases)¹⁷ which activate them The nature and concentration of the substrates determine the total reducing capacity of the tissue Most of the substrates, however, have reducing ability only after activation by the dehydrogenases The concentration of these enzymes, consequently, determines the active reducing capacity of the tissue An attempt was

¹⁶ If the test is performed in the presence of a small volume of 0.001 molar cyanide, the tissue will at first remain entirely colorless, while in the control without cyanide the color develops rapidly After the lapse of some time the color suddenly begins to appear in the tissue with cyanide and then develops as rapidly as it did initially in the control This appearance of color in the tissue with cyanide can be prevented if the cyanide-reagent solution is frequently renewed It is evident that the tissue is able to oxidize cyanide and recover from its toxic effects We have noted a similar recovery from cyanide in regard to its effect on the penetration of acid and basic dyes, provided an overdose of cyanide is avoided

¹⁷ Since these enzymes activate both the oxidant and the reductant, they might more properly be referred to as "metabolitases"

made, therefore, to estimate the concentration of dehydrogenases and substrates in the stroma. This was done by comparing the behavior of two basic dyes, methylene blue and crystal violet, one of which, methylene blue, can be reduced by the stroma and when reduced is electrically neutral. This comparison was made as follows.

If crystal violet in low concentration is introduced into the stroma, it is transferred rapidly and completely to the epithelium. When higher concentrations are used, the dye accumulates in the epithelium much more slowly. This difference in behavior with concentration is attributed to the fact that crystal violet is associated in concentrated solution (see Appendix). Methylene blue, on the other hand, although approximately equally associated, is transferred from the stroma to the epithelium equally well in dilute and concentrated solutions. This, in spite of the fact that when methylene blue is visible in the stroma the potential difference between the stroma and the epithelium is diminished and in spite of the further fact that under conditions when methylene blue is not reduced it is found to penetrate the tissue more slowly than crystal violet. For instance, crystal violet in the supravital technic stains the ciliary epithelium with great rapidity, while methylene blue stains slowly. This is not due to a difference in the staining ability, for with time an equal intensity of stain can be produced with either dye. Furthermore, at p_H 5.3, at which basic dyes penetrate from the epithelium to the stroma (see section IV), crystal violet passes with ease while methylene blue penetrates with great difficulty. On the other hand, when methylene blue is reduced the leuco form penetrates much more readily than does crystal violet. For instance, at physiologic p_H under asphyxia, crystal violet penetrates with difficulty or not at all, while methylene white penetrates with great ease, as can be shown by subsequent oxidation.

It was found that the oxidant of methylene blue passes through the stroma-epithelium barrier less readily than does crystal violet and the reductant penetrates much more readily than crystal violet. Since methylene blue is transferred from the stroma to the epithelium under physiologic conditions more readily than crystal violet, it is evident that it is transferred mainly in the reduced state. Since methylene blue, even in high concentration, is rapidly transferred from the stroma to the epithelium, it is evident that the rate of reduction in the stroma must be fast and, consequently, that there must be a high concentration of dehydrogenases and of substrates in the stroma. This inference is substantiated by a study of the secretory mechanism of the choroid plexus by one of us (R. D. S.) in association with L. Flexner^{17a}.

The inactivation of the dehydrogenases, by a shift in p_H , may be compared with that of the indophenol-oxidase. Whereas under nitrogen, at physiologic p_H , safranin T is partially reduced by the stroma, at p_H 6.2 methylene blue is only partially reduced, and at p_H 5.7 even toluylene blue is only partially reduced. Since the concentration of metabolites is not changed, it follows that the dehydrogenases are inactivated at these p_H .

17a Stiehler, R. D., and Flexner, L. J. Biol. Chem., to be published

Summary—We may conclude that the difference in potential between the epithelium and the stroma is due, at least in part, to the presence in the epithelium of indophenol-oxidase and the absence of this enzyme in the stroma. Other oxidative enzymes are apparently distributed in the stroma and epithelium. Dehydrogenases are present in the epithelium and stroma. The potential difference between the epithelium and the stroma can be abolished by asphyxia, which affects the oxidase system or by acid p_H , which inactivates both the oxidase and the dehydrogenases. It is interesting to note that with the disappearance of the potential difference the ionic current described in section I likewise disappears. The remaining link in the postulated electric circuit, namely, the electron carrier, will be considered next.

IV STROMA-EPITHELIUM BARRIER

Since the epithelium and the stroma are everywhere separated from one another by the stroma-epithelium barrier, it was natural to study this barrier and to see whether it could act as the electron conductor carrying electrons from the stroma to the epithelium. The first investigations were directed toward the influence of p_H on the physical chemical characteristics of this barrier.

A p_H EFFECT AND ISOELECTRIC POINT

As has already been noted, when the potential difference between the stroma and the epithelium and the ionic current are abolished by asphyxia, the stroma-epithelium barrier shows a selective permeability in favor of acid and neutral dyes and a relative impermeability to basic dyes. It has likewise been noted that a slight shift toward higher acidity will produce the same effect. If the acidity is raised still higher, a point is reached where the permeability characteristics of the barrier to acid and basic dyes are reversed. This transition point will be called the isoelectric point of the barrier. The experiments on the effect of p_H on the barrier were performed as follows.

In order to test the effect of p_H on the barrier, it was first necessary to know how effectively the p_H of the tissue could be controlled by buffer solutions. Small portions of excised ciliary body were placed on glass slides and covered with from 1 to 2 cc. of various buffer solutions. After a few minutes a drop of p_H indicator (bromocresol green or bromocresol purple) was added, and the color in the tissue as well as in the bathing solution was observed under a microscope. Under the conditions of the experiment (p_H 5.3 to 7.0) these indicators were found to penetrate well into the epithelium and stroma. As nearly as could be determined, the same p_H existed on both sides of the barrier and in the surrounding fluid when buffers were used which had one species of the buffer system in the neutral or uncharged state, e. g., acetic acid-acetate, pyridine-pyridine hydrochloride and pyridine-pyridine acetate-acetic acid buffers. When buffers were used, both species of which were charged, e. g., phosphate and citrate buffers of p_H below

7.0, the p_H of the tissues was considerably higher than that of the surrounding fluid. Pyridine-pyridine acetate and acetic acid and acetate buffers were, consequently, selected for further study. The buffers were isotonic and contained calcium chloride, potassium chloride, magnesium chloride and potassium dihydrogen phosphate in the concentrations present in Ringer's solution. Buffer solutions between p_H 5.0 and 7.0 were made and their p_H determined electrometrically with a glass electrode. Standard acetate (0.1 molar acetic acid plus 0.1 molar sodium acetate) was used as the standard and assigned a p_H value of 4.620 at 25 C.

In testing the effect of p_H on permeability, an essentially similar technique was used in one set of experiments. Small portions of excised ciliary body were placed on a glass slide and covered with from 1 to 2 cc. of buffer of the desired p_H . After five minutes the buffer solution was drained off, and a minute drop of dye solution was placed near the edge of the tissue. When the epithelium had become stained, the excess of dye was removed, and the tissue was washed with the buffer and examined under the microscope for possible penetration of the dye into the stroma. In those experiments in which the observations had to be protracted for more than a few minutes, the evaporation of the buffer was avoided by having the tissue in a hollow ground slide under a cover slip.

In order to observe the penetration of dyes from the stroma to the epithelium, a more complicated procedure was used. The chest of an albino rabbit was opened, ether anesthesia being used, a cannula was inserted into the ascending aorta and the animal was perfused with Ringer's solution until the fluid returned clear. The perfusion was then halted. Both corneas were incised, and with the aid of a blunt spoon the lens and the vitreous were delivered, care being taken to avoid injury to the ciliary body. The ocular cavity was then irrigated for five minutes with a buffer solution (different buffers being used in the two eyes). At the end of five minutes, with the buffer solutions still actively irrigating the ocular cavity, a solution of the dye to be tested was injected through the perfusing cannula into the aorta, the injection being continued until the ocular blood vessels were well colored with the dye. The ciliary body and iris were then removed from each eye and dropped into a solution of the buffer with which it had been irrigated. Small slices still immersed in the same buffer solution were then studied under a microscope.

With decreasing p_H there is in the first part of the acid region a slight but definite decrease in the permeability of the stroma-epithelium barrier for both acid and basic dyes. This is associated with a slight shrinkage of the tissue. Throughout this zone of slight acidity (p_H 7.0 to 5.5) the barrier maintains the same selectivity as at physiologic p_H under asphyxia, being relatively impermeable to basic dyes but freely permeable to acid dyes (table 4). Between p_H 5.50 and 5.47 an abrupt change occurs, for at p_H 5.47 and lower the barrier is impermeable to acid dyes and freely permeable to basic dyes. At p_H 5.485 it is equally permeable to both acid and basic dyes. The same selective permeability of the barrier was found irrespective of whether the test dye was allowed to diffuse from the epithelium toward the stroma or in the reverse direction. It follows that the isoelectric point measured is that of the stroma-epithelium barrier and not that of any other substance.

Summary—The selective permeability of the stroma-epithelium barrier for acid as against basic dyes is to be attributed to the electric charge inherent in the barrier, and the reversal of this selective permeability at p_H 5.50 to 5.47 indicates a reversal of the sign of the charge. At some point between p_H 5.50 and 5.47 the barrier must be without excess electric charge, i. e., isoelectric, a conclusion which is confirmed by the finding that selective permeability is absent at p_H 5.485. On the acid side of the isoelectric point, i. e., at p_H 5.47 and lower, the barrier is positively charged. On the alkaline side of the isoelectric point, i. e., at p_H 5.50 and higher, including physiologic p_H , the barrier is negatively charged.

B. OXIDATION-REDUCTION

The isoelectric point of the stroma-epithelium barrier was found neither to be fixed in position nor to have a definite breadth of zone. Instead it could be changed experimentally by oxidation and restored

TABLE 4—*Determination of the Isoelectric Point of the Stroma-Epithelium Barrier, Effect of p_H on Penetration of Dyes*

Acetic Acid-Acetate Buffers				
p_H	Penetration			
	Epithelium to Stroma		Stroma to Epithelium	
	Acid Dyes	Basic Dyes	Acid Dyes	Basic Dyes
5.3	0	+	0	+
5.473	0	+		
5.486	+	+		
5.496	+	0		
5.7	+	0	+	0

to the original condition by subsequent reduction. This observation indicated that the barrier is a material substance, not merely a bounding surface, and that this barrier has as one component, at least, a reversible oxidation-reduction system. The presence in the barrier of such a system would enable it to act as an electron conductor capable of receiving electrons from the stroma and transferring them to the epithelium.¹⁸ This property or action of the barrier is comparable to that of "mediators" which act normally in intracellular metabolism between two enzyme-substrate systems.

The only available method for studying the oxidation-reduction system of the stroma-epithelium barrier is to determine the shift in the isoelectric point (or the change in charge on the stroma-epithelium barrier) on oxidation. This was done by studying the selective permeability of the barrier to acid and basic dyes. In most experiments

¹⁸ The potentiality of the stroma-epithelium barrier to act as an electron conductor by virtue of a reversible oxidation-reduction system does not exclude the possibility of other, as yet unknown, mechanisms of electrical conduction.

the test dye was introduced into the epithelium. In a few experiments the dye was introduced into the stroma. The same results were obtained with both sets of experiments, proving that the effects observed were due to oxidation of the barrier and not of some other substance. By the use of this method, it was possible not only to study the effect of oxidation on the properties of the barrier but to measure roughly the characteristic potential (E'_0) of the oxidation-reduction system in the barrier. The experiments were performed as follows.

To test the effect of oxidation, three pieces of excised ciliary body were placed on slides. One piece of tissue was immersed in a buffer solution and the other two pieces in a buffer solution plus an oxidizing agent. After five minutes the oxidizing solution was removed from one of these pieces and replaced by a buffer solution containing a reducing agent. After five more minutes the oxidizing solution and the reducing solution were washed off with a buffer solution and the test stain was added to all three specimens. To test the effect of reduction, the same procedure was used except that buffer solutions containing a reducing agent were added to two of the three specimens at first, and one was subsequently oxidized. Ferricyanide, ferric chloride, iodine and quinhydrone have been used as oxidants and sodium hydrosulfate, hydroquinone and a solution saturated with both quinhydrone and hydroquinone as reductants.

The effect of oxidation on the charge of the barrier was also studied by observing the movement of dyes from the stroma to the epithelium. Acid dyes were obtained in the stroma, with the epithelium clear, by injecting the dye intravenously, removing the iris-ciliary body as described previously and placing the tissue in Ringer's solution (the ionic current prevents the epithelium from staining). Slices of the tissue were placed on slides. A buffer solution (p_H 5.7) was added to some slices and a buffer solution plus an oxidant to others. After from five to ten minutes, some of the slices containing the buffer plus an oxidant were washed with buffer solution, and buffer plus hydrosulfite was added. Basic dyes were obtained in the stroma, with the epithelium clear, by irrigating the ocular cavity with a buffer solution (p_H 5.7) while injecting the dye as described previously. The tissue was treated as with acid dyes except that it was kept in a buffer solution in place of Ringer's solution. The choice of oxidant was difficult, since the oxidants, except ferric chloride, react with basic dyes. Iodine seemed the best to use, for although it precipitated the dye, precipitation was not complete. The tissue was observed in acetate buffers (pyridine reacts with iodine) with and without iodine. Care must be used not to allow the tissue to remain in the iodine solution for more than a minute or two since the iodine tends to be concentrated in the tissue and renders the dye more insoluble. The iodine was washed off with several changes of buffer solution, and the tissue was examined. The dye which penetrated into the epithelium in the presence of iodine was largely precipitated and hence gave only a feeble color. If a reductant was then added which reduced the excess iodine, the dye became soluble and stained the tissue brilliantly. The same results were obtained whether the penetration of dyes was studied from the epithelium to the stroma or vice versa.

As can be seen in table 5, all of the oxidants employed produce a reversal in the penetration of acid and basic dyes. Since the penetration of dyes depends on the electric charge on the stroma-epithelium barrier,

the charge must be reversed, demonstrating that the isoelectric point of the barrier has been shifted. This change in the charge on the barrier on oxidation can be completely reversed on subsequent reduction in the case of all the oxidants, showing that the oxidation-reduction system of the barrier is a reversible one and that the reaction is independent of the specific reagents employed.

In table 6 it is seen that the isoelectric point of the stroma-epithelium barrier is shifted by oxidation from p_H 5.485 to about p_H 6.6, whereas

TABLE 5—*Influence of Oxidation and Reduction on the Stroma-Epithelium Barrier*

Acetate Buffer p_H 5.70				
Reagent	Penetration of Dyes After			
	Oxidation		Reduction*	
	Acid Dyes	Basic Dyes	Acid Dyes	Basic Dyes
Control (no oxidant)	+	0		
Ferricyanide	0	+	+	0
Iodine	0	+	+	0
Quinhydrone	0	+	+	0
Ferric chloride	0	+	+	0

* The tissue is first oxidized by the reagent and then reduced with hydrosulfite

TABLE 6—*Shift of Isoelectric Point of Stroma-Epithelium Barrier by Oxidation*

Pyridine-Pyridine Acetate Buffers				
p_H	Penetration of Dyes Through Membrane			
	Membrane Reduced		Membrane Oxidized	
	Acid Dyes	Basic Dyes	Acid Dyes	Basic Dyes
5.40	0	+	0	+
5.47*	0	+		
5.60	+	0	0	+
6.10	+	0	0	+
6.27	+	0	+	+
6.94	+	0	+	+
7.08	+	0	+	0
Ringer's solution	+	0	+	0

* Acetic acid-acetate buffer

with reduction, no shift is observed (compare p_H 5.473 in table 4 with p_H 5.47 in table 6). It was noted in table 4 that the isoelectric zone of the barrier normally is less than 0.03 p_H units. Table 6 shows that in the oxidized state the zone covers between 0.7 and 1.0 p_H unit. Since the extent of the isoelectric zone is a measure of the slope of the acid-base titration curve in the vicinity of the isoelectric point, as shown later, it follows that the titration curve of the barrier normally and in the reduced state has a slope at least thirty times the slope of the curve for the oxidized state. This is not unusual, because oxidants generally have stronger acid and basic groups than the reductants, and, as is well known,

the stronger the acid-base groups in an ampholyte the wider the isoelectric zone. It is also not surprising to find that reduction causes no shift in the isoelectric point under these conditions. These conclusions are derived from the following theoretic considerations.

From the acid-base titration curves for the reductant and oxidant of an ampholyte containing a reversible oxidation-reduction system, e. g., the stroma-epithelium barrier, the relation between the isoelectric point and the degree of oxidation can be determined. Unfortunately, the actual titration curves are not known. However, an approximate relation can be calculated if the acid-base titration curves for both reductant and oxidant of the ampholyte are assumed to be linear in the pH region between the isoelectric points of reductant and oxidant, as shown in figure 3 in which OX represents the titration curve of 100 per cent oxidant and RED that of 100 per cent reductant.¹⁹ At the isoelectric point (pI) for the ampholyte partially oxidized, it follows that

$$(B_o^+) = K_o (pI_o - pI) \alpha$$

$$(A_r^-) = K_r (pI - pI_r) (1 - \alpha)$$

in which

(B_o^+) equals the excess cations of the oxidant

(A_r^-) , the excess anions of the reductant

K_o , the slope of the titration curve of the oxidant

K_r , the slope of the titration curve of the reductant

pI , the isoelectric point for a mixture of oxidant and reductant

pI_o , the isoelectric point of the oxidant

pI_r , the isoelectric point of the reductant

α , the fraction in the oxidized state

At any isoelectric point $(B_o^+) = (A_r^-)$

or

$$K_o (pI_o - pI) \alpha = K_r (pI - pI_r) (1 - \alpha)$$

$$pI = \frac{K_r pI_r (1 - \alpha) + K_o pI_o \alpha}{K_r (1 - \alpha) + K_o \alpha}$$

Let $M = pI_o - pI_r$, then

$$pI = pI_r + \frac{MK_o \alpha}{K_r (1 - \alpha) + K_o \alpha}$$

or

$$\frac{pI - pI_r}{M} = \frac{\Delta pI}{M} = \frac{K_o \alpha}{K_r (1 - \alpha) + K_o \alpha} = \frac{\alpha}{\alpha + (1 - \alpha) K_r / K_o}$$

In figure 4, $\Delta pI / M \times 100$ (percentage of shift in the isoelectric point) is plotted against 100α (percentage of oxidation) for different ratios of K_r / K_o .

19 This assumption is one extreme of the possible titration curves and is approximated by substances having many acid and basic groups, e. g., proteins. The other extreme is obtained when each species has only one acid and one basic group, e. g., simple amino acids. Calculations made for both extreme cases lead to almost identical results. Since the latter type leads to an equation of the fourth degree with the attendant difficulties of calculation and presentation, only the simpler case is presented.

From the curves, it can easily be seen that when K_r is much larger than K_o , a small amount of oxidation produces little shift in the isoelectric point. The relative values of K_r to K_o can be determined from the width of the isoelectric zones as follows

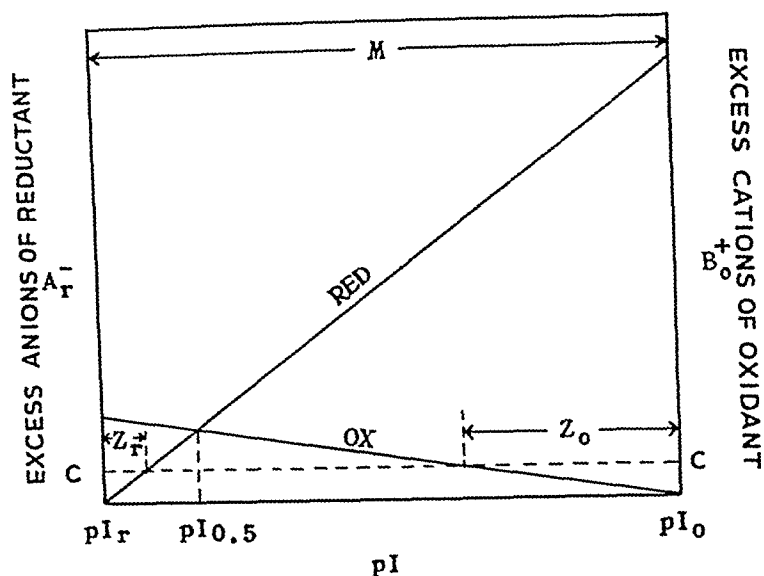


Fig 3—Diagram illustrating the relation between excess anions of reductant or excess cations of oxidant and shift in the isoelectric point

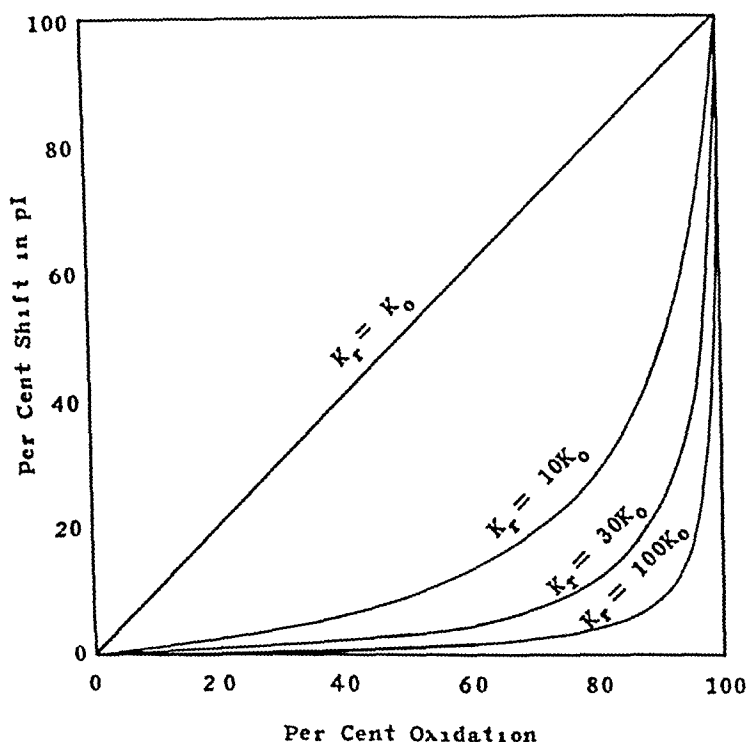


Fig 4—Chart showing a shift in the isoelectric point with oxidation

In figure 2, C is assumed to be the excess charge necessary on the stroma-epithelium barrier, in any state of oxidation, to prevent the passage of oppositely charged ions. Z_r will then be equal to one-half the width of the isoelectric zone of the reductant and Z_o to one-half that of the oxidant. Since Z_r and Z_o are

inversely proportional to K_r and K_o , the ratio K_r/K_o will be equal to the ratio of the widths of the isoelectric zones of oxidant and reductant ($2Z_o/2Z_r$). The intersection of the titration curves of oxidant and reductant is the isoelectric point (pI_o) for 50 per cent oxidant plus 50 per cent reductant. It can be seen that this point will vary from pI_o when K_o equals ∞ , through $\frac{1}{2} (pI_r + pI_o)$ when K_o equals K_r , to pI_r when K_o equals 0 (K_r remaining constant).

In the case of the stroma-epithelium barrier of the ciliary body, as stated before, the slope of the reductant is at least thirty times that of the oxidant ($K_r = 30 K_o$) and probably not more than one hundred times that of the oxidant ($K_r = 100 K_o$). If one observes these curves in figure 4, it is easily seen that it would not be possible to detect any oxidation of the stroma-epithelium barrier less than 25 per cent and, further, that 80 per cent of the shift in the isoelectric point occurs in the last 10 per cent of oxidation of the barrier. Consequently, the isoelectric point is most sensitive to changes in potential between 90 and 99 per cent oxidation of the stroma-epithelium barrier. This agrees with our observation.

TABLE 7—*Determination of E_o' of Oxidation-Reduction System of the Stroma-Epithelium Barrier*

Pyridine-Pyridine Acetate Buffer pH 6.6

Reagent	E_h at pH 6.6 Volts	Penetration of Crystal Violet
Normal		0
Ferricyanide	Over +0.420	+
Quinhydrone	+0.300	+
Saturated quinhydrone plus hydroquinone	+0.215	+
Saturated hydroquinone	+0.185	0

In table 7 it is significant to note that at pH 6.6 oxidation of the stroma-epithelium barrier by quinhydrone allowed basic dyes to pass through the barrier. Reduction of the barrier with a saturated solution of hydroquinone ($E_h = +0.185^{20}$) restored the original selective permeability, whereas, a solution saturated with both hydroquinone and quinhydrone ($E_h = +0.215$) did not. Therefore, at pH 6.6 and at some potential (E_h) of the stroma-epithelium barrier between +0.185 and +0.215, the selective permeability of the barrier to basic dyes is removed. This reversal in permeability to basic dyes occurs at the upper limit of the isoelectric zone, as shown before. It can be shown that the shift in the limit of the isoelectric zone is proportional to the shift in the isoelectric point itself. From the information derived on the relation between the isoelectric point and percentage of oxidation, the degree

20 This is the minimum value of the potential measured. It is obvious that the hydroquinone solution must have contained traces of quinone. Since this solution was used as a reductant, the potential established in the tissues cannot be less than the measured potential. If the potential in the tissues is higher, the error in measuring the potential of the stroma-epithelium barrier is correspondingly reduced.

of oxidation (α) of the stroma-epithelium barrier, corresponding to a shift in the upper limit of the isoelectric zone to p_H 6.6, is calculated to be between 98 and 99.5 per cent.

The necessary information required to calculate the E'_0 of the oxidation-reduction system of the stroma-epithelium barrier from the values of E_h and α is not available. However, if it is assumed that the system in the stroma-epithelium barrier can be treated as similar systems in aqueous solution and that neither reductant nor oxidant are associated, the value of E'_0 can be calculated from the equation

$$E_h = E'_0 + \frac{RT}{nF} \ln \frac{\alpha}{1-\alpha}$$

The validity of these assumptions must remain questionable.

On the basis of the assumption that the oxidation-reduction system of the stroma-epithelium barrier involves two electrons (a one electron system would be more favorable for the present argument), the E'_0 at p_H 6.6 is $+0.140 \pm 0.025$ volt (the error considers only the errors in the determination of E_h and α and not those inherent in the assumptions). It is not possible to determine the E'_0 of the system at physiologic p_H since the E'_0 - p_H curve is not known. It is reasonable to assume that the slope of the curve is no greater than 0.06. If a 0.03 slope is assumed, then E'_0 equals $+0.115 \pm 0.050$ volt at p_H 7.4. The additional uncertainty of ± 0.025 volts is added to allow for a 0.00 slope or a 0.06 slope. The potential of the epithelium at this p_H is estimated to be $+0.100 \pm 0.015$ volts (see section II). If the assumptions are valid, then, under physiologic conditions the stroma-epithelium barrier would be oxidized partially by the epithelium or reduced completely by the stroma.

Summary—The isoelectric point of the stroma-epithelium barrier is shifted by oxidation and restored to the original position by subsequent reduction. Therefore, the barrier has as one of its components, at least, a reversible oxidation-reduction system. The isoelectric zone is wider in the oxidized state than in the normal and reduced states. From the data on the shift of the isoelectric point and the width of the isoelectric zones, together with the potential of an incompletely oxidized stroma-epithelium barrier measured at one limit of the isoelectric zone, the E'_0 of the oxidation-reduction system is calculated with the aid of certain assumptions to be $+0.140$ volts at p_H 6.6. From this potential it is inferred that the epithelium normally tends to oxidize the membrane partially, whereas the stroma tends to reduce it. Consequently, electrons can be received by the barrier from the stroma and transferred to the epithelium by virtue of the reversible oxidation-reduction system of the barrier. All the elements necessary for an electric current to exist have been demonstrated. This current can produce useful work. One form of work is in the transfer of water, which will be considered next.

V TRANSFER OF WATER

The present investigation had its origin in the discovery that the ciliary body was irreciprocally permeable to water. Since the movement of water in the transparent and watery tissues of the eye cannot readily be observed, we have been forced to study first the movement of substances that we could see, namely, dyes. In what way can the knowledge gained regarding the mechanism of the transference of dyes in the ciliary body be applied to the problem of transfer of the water? The passage of an ionic current through the pores of an electrically charged membrane is associated with the transfer of water through the membrane. This phenomenon has been known and studied for over a century and is called electroendosmosis. It has been shown that if the membrane is positively charged, the flow of water is in the same direction as that of the anions, if the membrane is negatively charged, the flow of water is in the same direction as that of the cations. Since the ciliary stroma-epithelium barrier is negatively charged at physiologic p_H , the endosmotic flow of water through the barrier must be in the same direction as that of basic dyes, namely, from the stroma to the epithelium.

The mechanism by which water is transferred across a membrane under the influence of an electric current has not been adequately determined. The generally held theory assumes that the applied current moves the water directly. Bethe's²¹ experiments suggest that the current leads to an accumulation of ions on one side of the membrane and that the water is moved osmotically. For our present purpose it is immaterial whether one or the other of these hypotheses is correct. It is, however, possible to show that water can be transferred across the ciliary stroma-epithelium barrier in appreciable quantities by an artificially applied electric current as follows.

The experiment was performed as follows (fig 5). A glass cannula filled with Ringer's solution was introduced into the vitreous of each eye of a rabbit, ethyl carbamate anesthesia being used. Each cannula was connected by a T tube to a horizontal capillary tube containing a bubble of air and thence to a reservoir of Ringer's solution, the capillary to serve as a flow meter. The other arm of each T tube contained a zinc-zinc sulfate nonpolarizable electrode, the zinc sulfate solution being separated from the Ringer's solution by an agar plug. At the onset of the experiment the level of the reservoirs of Ringer's solution was raised to a point slightly above the normal intraocular pressure, so that a slow movement of the bubble in each capillary toward the eye was observed. An electric current was then applied to the two zinc electrodes, and any change in the direction or velocity of movement of the bubbles in the flow meters was recorded.

It was found that when sufficient potential (20 volts or more) was applied, the solution flowed into the eye from the reservoir attached to

21 Bethe, A., and Toropaff, T. *Ztschr f phys Chem* **39** 597, 1915

the positive electrode, and fluid came out of the eye attached to the negative electrode (fig 5). In the latter case the direction of the ionic current was the same as that which we have observed normally under the influence of the difference in potential which exists between the two sides of the ciliary stroma-epithelium barrier. When the direction of the current was reversed, the direction of flow in the flow meters was promptly reversed.

If the animal's eyes were removed after a short flow of current and fixed and sectioned, it was found that the ciliary body in the eye that had been attached to the positive electrode was normal in appearance,

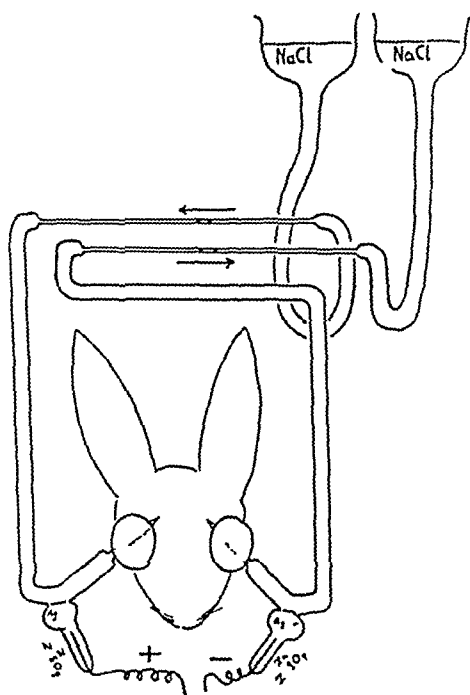


Fig 5—Schematic diagram of apparatus used in the electroendosmosis experiment

while in the eye attached to the negative electrode the ciliary capillaries were engorged and tightly packed with corpuscles. It would appear that fluid had been withdrawn from these capillaries into the interior of the eye at so rapid a rate as to result in an inspissation of the blood within the capillaries and a consequent congestion of these vessels.

Summary—These experiments show that the electrical mechanism responsible for the transfer of dyes in the ciliary body is adequate to explain also the transfer of water in this tissue. The experiments here reported are sufficient to show that this secretory mechanism may, in fact, be operative in the ciliary body. Conclusive proof that this mechanism is actually operating and that if operating it accounts quantitatively for the secretory activities of the ciliary body is lacking.

VI OTHER COMPONENTS OF THE BLOOD-AQUEOUS BARRIER

A CAPILLARY WALL

The blood-aqueous barrier in the ciliary body is composed of three different membranes—the capillary endothelium, the stroma-epithelium barrier and the epithelial cell membrane. In the course of the experiments described numerous dyes have been injected intravascularly into albino rabbits, and the iris and ciliary body were then examined under the microscope. It was observed that none of the basic dyes used penetrated the walls of the capillaries of the iris, while all entered the ciliary stroma with ease. The acid dyes penetrated the capillaries of the iris slowly and feebly, while they penetrated the ciliary stroma with ease.

Summary—The ciliary capillaries are much more permeable than either the capillaries of the iris or the stroma-epithelium barrier.

B EPITHELIAL CELL MEMBRANE

The question may be raised as to whether the structure that we have called the stroma-epithelium barrier is identical with the cell membrane on the surface of the second layer of epithelium facing the stroma. While it is plain that this part of the cell membrane must form part of the barrier between the cytoplasm of the epithelium and the reticulum of the stroma, it is evident from the experiments reported that those parts of the epithelial cell membrane that are available for separate observation show entirely different permeability characteristics from the stroma-epithelium barrier. At p_H higher than the isoelectric point of the barrier, both acid and basic dyes diffuse readily into both layers of epithelium, while only acid dyes penetrate the stroma-epithelium barrier. At p_H lower than the isoelectric point of the barrier, both acid and basic dyes continue to diffuse readily into both layers of epithelium, while only basic dyes penetrate the stroma-epithelium barrier. Also, we have found no evidence of polarity in the cell membrane of the first layer of epithelium in which both the free surface and that adjoining the second layer were adequately studied. If the basal layer of the epithelial cell membrane is identical with what we have called the stroma-epithelium barrier, it is different from either the intercellular or the superficial membrane of the epithelial cells.

Summary—The stroma-epithelium barrier is the least permeable of the three anatomic barriers between the blood and the aqueous in the ciliary body, hence the limitations in permeability of the stroma-epithelium barrier form the determining factor in the whole system.

COMMENT

The present investigation was undertaken with the aim of discovering the secretory mechanism of the ciliary body. We have found that the respiratory metabolism of the epithelium differs from that of the stroma. This difference is due to the presence of Warburg's respiratory enzyme in the epithelium and the absence of this enzyme in the stroma. Owing to this distribution of the oxidase, there is a difference in potential between the epithelium and the stroma which gives rise to an electric current carrying cations from the stroma to the epithelium and anions from the epithelium to the stroma. This ionic current will transfer water from the stroma to the epithelium. The electric circuit is completed by an electron transfer through the stroma-epithelium barrier, presumably through the reversible oxidation-reduction system of this barrier. Under conditions which cause the potential difference between the epithelium and the stroma to disappear, the electric current disappears. It is interesting to note that the reciprocal permeability of the ciliary body to water reported previously¹ disappears under conditions which abolish the potential difference and the electric current.

The secretory mechanism which we have described should not, we believe, be thought of as solely responsible for the formation of the intraocular fluid. Variations in the permeability of the ciliary capillaries, in the osmotic pressure of the plasma proteins and in the excess of pressure in the ciliary capillaries over the intraocular pressure must largely control the amount of fluid which enters the stroma of the ciliary body and is available for transfer into the ocular cavity.

SUMMARY

Between the stroma and the epithelium of the ciliary body there is a barrier which, under certain conditions, shows a selective permeability in favor of acids as against basic dyes. This barrier is negatively charged at physiologic p_H and has an isoelectric point at p_H 5.485. The permeability of the barrier is increased by the absence of calcium. The barrier is capable of reversible oxidation and reduction. Depending on the validity of certain assumptions, the E'_0 of the oxidation-reduction system in the barrier is +0.140 volt at p_H 6.6. Under physiologic conditions the barrier tends to be partially oxidized by the epithelium and to be reduced by the stroma.

The epithelium of the ciliary body contains indophenol-oxidase. The stroma does not contain this enzyme. Other oxidases are probably equally distributed in these tissues. Both tissues contain dehydrogenases. Related to the unequal distribution of the indophenol-oxidase there is a difference in potential between the epithelium and the stroma of 0.230 volt. The potential of the epithelium is +0.100 volt. The potential of the stroma is -0.130 volt.

The ciliary body shows an irreciprocal permeability to water and to certain acid and basic dyes, transferring water and basic dyes from the stroma to the epithelium, i. e., from the blood to the aqueous, and transferring acid dyes in the reverse direction.

It is postulated that the mechanism of the irreciprocal permeability of the ciliary body depends on an electric current which transfers water and cations from the stroma to the epithelium and anions from the epithelium to the stroma. The source of energy for this electric current is supplied by the differences between the oxidative and the reductive processes in the epithelium and stroma. The electric circuit is completed by an electron transfer through the stroma-epithelium barrier, presumably through the reversible oxidation-reduction system of the barrier.

APPENDIX

In the course of the investigation reported here many dyes beside those mentioned were used. Many of these were found unsuited for the purposes of our study because of poor color, poor staining quality, poor penetration, irreversible destruction in the tissue or excessive toxicity (staining of nuclei). These and certain other characteristics of the dyes used are listed in table 8. The azo dyes were not included in the table, since they are generally toxic and do not penetrate or stain the epithelium easily. The dyes that were found most satisfactory and that were, therefore, used for most of the experiments were crystal violet (basic dye), bromphenol blue and rose bengal (acid dyes) and rhodamine B (neutral dye).

In order to interpret the behavior of the dyes in the tissue it would be necessary to know their physical chemical characteristics. Unfortunately, little such information is available in the literature. Data on the molecular size or volume, molecular shape, hydration, etc., are still wanting. Some of the observations on the behavior of the dyes in the tissue are explained by the properties of these dyes, discussed later.

A IDENTIFICATION OF THE DYES

Commercial dyes sold by different manufacturers under the same name are often different dyes. Furthermore, dyes as sold often are not one chemical substance but contain impurities and isomeric substances. In order to identify the dyes used in these studies, the Color Index number and the manufacturer are listed in table 8. Dyes not obtained commercially were obtained through the courtesy of the department of physiologic chemistry or synthesized in our laboratory.

B ASSOCIATION

Holmes²² has shown that the absorption spectrums of many dyes shift with changing concentration of the dye. Cohen and Preisler²³ found in the case of the oxazine dyes that the oxidation-reduction potentials shifted with the shift of the absorption spectrums and suggested that association (polymerization) of the oxidants would explain this phenomenon. Stiehler and Clark,²⁴ by studying

²² Holmes, W. C. *Indust. & Engin. Chem.* **16** 35, 1924.

²³ Cohen, B., and Preisler, P. W. *Studies on Oxidation-Reduction. XVI. The Oxazines. Nile Blue, Brilliant Cresyl Blue, Methyl Capri Blue, and Ethyl Capri Blue*, *Pub. Health Rep.*, 1931, supp. 92.

²⁴ Stiehler, R. D., and Clark, W. M. *J. Am. Chem. Soc.* **55** 4097, 1933.

TABLE 8—Data on Dyes Used in Experiments

Dye*	Color Index No	Chemical Type	Associated in Concentrated Solution†	Comment
Cations in Solutions				
Crystal violet (NAO)	681	T P M †	Yes	Described in text and appendix
Malachite green (NAC)	657	T P M	?§	Like crystal violet but penetrates stroma epithelium barrier more readily
Basic fuchsin (NAC)	677	T P M	No?	Poor stain, poor color, low solubility
Night blue (Gr)	731	T P M	Yes}	Extremely low solubility in salt solutions, otherwise like crystal violet
Victoria blue (NAC)	729	T P M	Yes{	
Methyl green (Gr)	685	T P M	No?	Filled stroma when injected intravenously, stained epithelium a feeble violet instead of green
Toluylene blue (Lab)	820	Indamine	? }	Penetrated stroma epithelium barrier in both directions aerobically, probably as reductant
Bindschedler's green (BDH)	819	Indamine	? {	
Methylene blue (HWD)	922	Thiazine	Yes	Described in text
Lauth's violet (NAC)	920	Thiazine	Yes	Like methylene blue
Cresyl blue (NAC)	877	Oxazine	Yes	Like methylene blue
Cresyl violet (NAC)		Oxazine	?	Oxidant like crystal violet, reductant like leucomethylene blue
Dimethylphenosafranine (NAC)	842	Azine	Yes	Like methylene blue except not reduced in stroma in air accumulated in epithelium slowly
Safranine T (?)	841	Azine	Yes	Like dimethylphenosafranine, poor color
Anions in Solution				
Bromphenol blue (HWD)		Sulfonphthalein	No	Described in text, feeble stain
Eosin Y (?)	763	Fluorone	No	
Erythrosine (?)	773	Fluorone	No	
Phloxine (NAC)	778	Fluorone	No	
Rose bengal (POL)	779	Fluorone	No	
Uranin (?)	766	Fluorone	Yes	Poor stain, poor color, penetrates stroma-epithelium barrier easily
Acid fuchsin (?)	692	T P M	?	Poor stain, poor color, irreversibly destroyed in tissue
Fast green F O F (NAC)		T P M	?	Poor penetration into epithelium, poor stain
Bromeresol green (HWD)		Sulfonphthalein	No }	Like bromphenol blue, partially as free acid in acid solutions see appendix
Bromeresol purple (HWD)		Sulfonphthalein	No {	
2,6 dichlorophenolindophenol (Lab)		Indophenol	No?	Easily reduced— in acid solutions, free acid present and irreversibly destroyed
Indigo disulfonate	1180	Indigo	No ??	Poor stain like bromphenol blue except penetrates stroma-epithelium barrier more slowly
Indigo trisulfonate (Lab)		Indigo	Yes?{	
Neutral in Solution				
Rhodamine B (NAC)	749	Fluorone	Yes	Described in text and appendix, penetrated stroma epithelium barrier rapidly under all conditions
Pyocyanine (HLAR)		Azine	?	Poor stain, low solubility, reduced in stroma

* (NAC), National Aniline & Chemical Co., Inc., (Gr) Groubler, (BDH), British Drug Houses Ltd. (HWD), Hynson, Westcott & Dunning, Inc., (POL), Providence Chemical Laboratories, (HLAR), Hoffmann-La Roche, Inc., (Lab) laboratory sample, (?), manufacturer unknown

† T P M indicates triphenyl methane dye

‡ The data are taken mainly from Holmes²²

§ The question mark means that no information is available or that data are not conclusive

the distribution of the dye between two immiscible solvents, were able to show in the case of one of the aposafranines that association of the oxidant occurred

The absorption spectrum of crystal violet in aqueous solution shifts with concentration,²² but there is no shift with concentration in amyl acetate solutions. We have, therefore, studied the distribution of crystal violet between water and amyl acetate. The results show that in amyl acetate only one species of the dye exists, whereas in water this species and its dimer are present. Determinations of the freezing point on 0.01 molar solution of crystal violet in water indicate a molecular weight of about 1100 (molecular weight of $C_{25}H_{30}N_3Cl + 9H_2O = 570$)

The distribution experiments show that in 0.01 molar solution over 99 per cent of the dye exists as dimer. Consequently, in concentrated aqueous solutions two molecules of the dye associate and in the amyl acetate solutions only the monomer exists. The association constant was found to be about 2×10^8 in water, about 2×10^{-6} in 0.15 molar sodium chloride solution. The effect of ionic strength on the association constant is similar to that found with sulfonated rosindone.²⁴

This phenomenon of association makes possible the interpretation of the following observations. When the ionic current in the ciliary body is abolished (asphyxia or p_H 6.0), crystal violet in 0.001 molar solution fails to penetrate the membrane. If 0.00001 molar solution is used, penetration slowly takes place. When the ionic current is not abolished, the dye is transferred from the stroma to the epithelium. If low concentrations are used, the accumulation of the dye in the epithelium is rapid. When high concentrations are used, the accumulation is much slower. With varying concentrations pictures intermediate between these extremes are obtained. Since even in concentrated solutions of the dye a small fraction is present as monomer, it is possible that only the monomer can pass through the membrane. Many of the other dyes that we have used also show this phenomenon of association (table 8).

C IONIC CHARGE AND RELATIVE MOBILITY

The ionic charge and the relative mobilities of the dyes were determined by observing the movement of the dyes under an electric current. The dyes were dissolved in 0.01 molar buffers. The dye concentration was 0.001 molar. The resulting classification of anions, cations and neutral particles is shown in table 8. The behavior of all of the dyes was the same as predicted from their chemical structure except rhodamine B. This dye is generally classified as basic and would, therefore, form cations in solution. The sample, obtained from the National Aniline & Chemical Co., was alleged to be the chloride. The sample did not contain chloride, and the dye did not migrate under the influence of an electric current. The dye is, therefore, electrically neutral and is probably a zwitterion in solution. The mobilities of all of the other dyes did not differ from one another by more than a factor of two. Consequently, the differences in penetration of these dyes in the tissue cannot be explained on the basis of their mobilities.

In buffers of low p_H some of the acid dyes exist partially as the free undissociated acid and hence behave as neutral substances. At physiologic p_H these dyes exist only as the anion or dissociated acid in solution.

COLOBOMA OF THE OPTIC NERVE AND OF THE MACULA

A MICROSCOPIC STUDY

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AND

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Colobomas of the optic nerve and of the macula are among the more interesting of ocular anomalies. The literature contains many clinical descriptions but relatively few anatomic studies of these defects. Caspar¹ in 1887 classified colobomas of the nerve clinically on the basis of the arrangement of the blood vessels as they emerged from, and distributed themselves on, the disk. It was soon learned, however, that there was little correlation between the microscopic structure and the appearance of the fundus and that wide differences existed in the cases in which anatomic study was possible.

Most authors agree that colobomas are due to faulty development in the elements comprising the tissue "anlage." Often these tissues are modified or replaced by other tissues, and alterations may occur in tissues surrounding the coloboma as well. Coloboma of the nerve is due to anomalous closure of the fetal cleft and occurs in its upper end. The defect has been ascribed by some to primary aberrations in the epiblastic layer of the optic cup and by others to alterations primarily in the mesoblastic tissue. Faulty development of the inner and outer layers of the optic cup and stalk, infolding of the primary layers, as well as the interpolation of vascular or other mesodermal tissue, and the alterations that may be present as the result of the pluripotentiality of the cells in the margin of the everted secondary optic vesicle are mentioned as factors influencing the formation of the immediate tissue about the nerve as well as of the nerve itself. Other factors, such as ingrowth of the nerve fibers of the retina into the primordially optic nerve stalk and the influence of one tissue develop-

Read before the New York Academy of Medicine, Section of Ophthalmology, April 18, 1938

From the Laboratories and Ophthalmological Division of the Mount Sinai Hospital

¹ Caspar, L. Ueber das Colobom des Sehnerven, Dissert., Bonn, J. Bach Wwe, 1887, Zur Kenntnis der angeborenen Anomalien der Sehnervpapille, Arch f. Augenheilk. **32** 12, 1896

ment on another, are further explanations offered for the structural peculiarities seen in cases of coloboma of the optic nerve

Colobomas of the nerve vary from almost complete absence of the nerve to modified degrees of excavations and "filled-in" ectasias of the

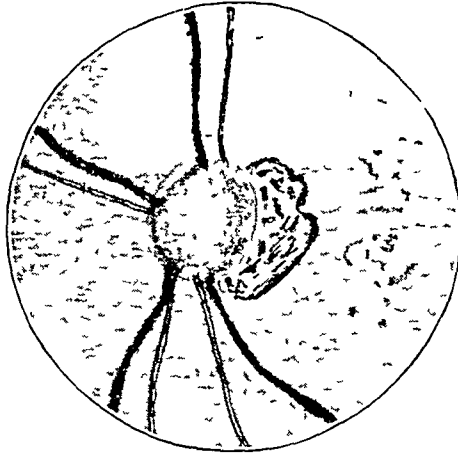


Fig 1—Drawing of the left fundus showing the coloboma of disk, temporal myopic crescent and macular retinal atrophy

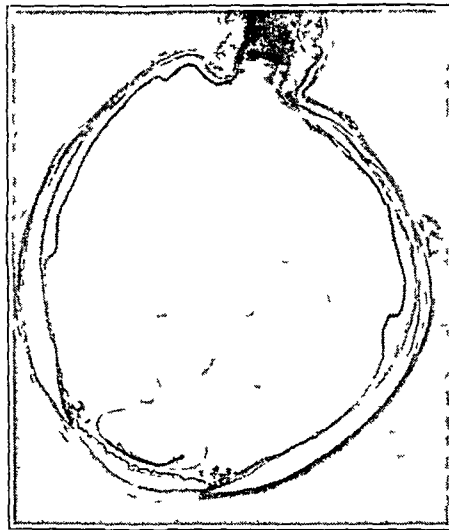


Fig 2—Section through the disk, indicating ectasia of the dura and sclera at the posterior pole and enlargement of the diameters of the globe

nerve head The peripheral portion of the nerve structure may be involved Included, perhaps, are the crater-like holes of the optic disk and disks with the type of inferior conus described by Fuchs²

² Fuchs, E Ueber die Lamina Cribrosa, Arch f Ophth 91 435, 1916

Coats³ in 1908 reviewed the literature dealing with instances of congenital defects of the nerve and concluded that only a few were true colobomas. Coats,³ Parsons⁴ and Duke-Elder,⁵ although of the opinion that the relative differences may be a question of didactic interest only, have defined the criteria for such congenital anomalies of the optic nerve. According to these authors a true coloboma of the optic nerve is one in which there is absence of a communication between the inter-vaginal space and the coloboma, i. e., the ectasia must lie wholly within the sheaths of the optic nerve. Also, a definite boundary must exist between the choroid and the colobomatous area. Coloboma of the choroid adjacent to the optic nerve and involving the latter is therefore not included within this grouping. Furthermore, in a true coloboma the central artery of the retina is found to course through the nerve head itself.

Colobomas of the macula have been described. Microscopically, the macula may be almost entirely replaced by unorganized tissue or consist of either cystic cavities or be represented by a hole. Although the macula is not derived from the primary fetal fissure, some authors consider macular coloboma to be an associated growth anomaly accompanying abnormal closure of the fetal cleft. Subsidiary fissures continuous with the primary cleft have been described. When not connected with the primary fetal fissure, the coloboma is considered atypical. According to Mann⁶ all colobomas of the macula are acquired. She expressed the belief that the maculas in those cases considered as colobomatous were injured during fetal life by trauma or inflammation. Sorsby,⁷ among others, however, has described cases of congenital macular coloboma in which the condition not only was familial but was often present bilaterally.

REPORT OF A CASE

History—Mrs. S. J., aged 40, was admitted to the medical ward of the Mount Sinai Hospital with a diagnosis of hypertensive renal disease. The fundus of the right eye revealed hypertensive retinitis. The left eye was highly myopic, with vision reduced to perception of light. The media were clear. The disk was

3 Coats, C. The Pathology of Coloboma of the Nerve Entrance, Roy London Ophth. Hosp. Rep. **17** 178, 1908.

4 Parsons, J. H. Pathology of the Eye, London, Hodder & Stoughton, 1906, vol. 2, p. 824.

5 Duke-Elder, W. S. Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, pp. 1256 and 1261.

6 Mann, I. C. Certain Abnormal Conditions of the Macular Region Usually Classed as Colobomata, Brit. J. Ophth. **11** 99 (March) 1927.

7 Sorsby, A. Congenital Coloboma of the Macula, Together with an Account of the Familial Occurrence of Bilateral Macular Coloboma in Association with Apical Dystrophy of the Hands and Feet, Brit. J. Ophth. **19** 65 (Feb.) 1935.

roughly oval, uniformly gray and from two to three times normal size. Its surface was slightly depressed, but further details of depth and markings could not be ascertained. All of the vessels sprang from the edge of the disk and turned sharply into the retina much in the manner of cilioretinal arteries and opticociliary veins. A large crescent bordered the disk temporally. The macular area was the seat of discrete areas of atrophy, probably of myopic origin. The

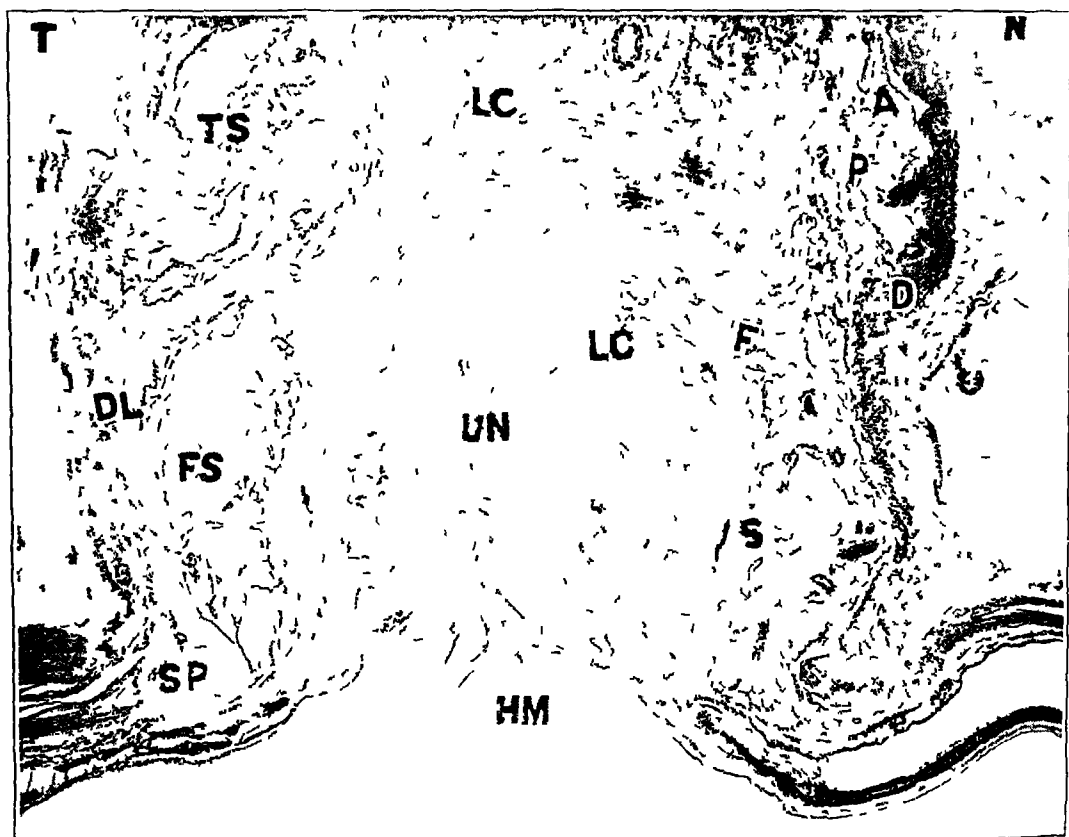


Fig 3—Section of the nerve through the approximate center of the disk. *T* is the temporal and *N* the nasal side of the nerve. Above is the normal nerve with the central retinal artery. The points in the nerve indicated by *LC* correspond to the upper and lower limits of the lamina cribrosa. The pia, *P*, and arachnoid, *A*, terminate midway between these levels and merge with the dura, *D*. There are thus a true intervaginal space, *TS*, and a false intervaginal space, *FS*. The upper fibers of the lamina cribrosa arch abruptly downward, especially temporally. They emerge as strands into the false space and can be traced down toward the scleral promontory, *SP*. Some are attached to the dura, *DL*, others are attached as fragments to the edge of the nerve, *F*. The false space contains, in addition to fragments of the lamina cribrosa, a loose syncytium of dural fibers and blood vessels derived from the central retinal artery. In front of the lamina cribrosa is the undifferentiated portion of the nerve, *UN*, consisting of central fibrillar and outer cellular zones, bordered on either side by a dural sheath, *S*, rich in elastic fibers. The central zone is continuous with the remnants of the hyaloid membrane, *HM*. The choroid is poorly developed temporally. Nasally, vessels in the false space enter the choroid, which is well developed on this side. No retinal vessels are seen in the disk at this level.

IVEXLER-LAST—COLOBOMA OF OPTIC NERVE AND OF MACULA 791

diagnosis was coloboma of the optic nerve in a highly myopic eye. Study of the visual fields could not be done on account of the condition of the patient. Death occurred ten days after her admission to the hospital.

Ocular Findings—The left eye, together with 15 mm of the nerve, was removed six hours after death. The cut end of the nerve was normal in size, but the vaginal space was considerably wider than normal. At the junction of the nerve and the sclera was a prominent ectasia of sclera, especially nasally. The measurement from this point to the corneal limbus was 33.5 mm; the circumference of the outer nerve sheath at the sclera was 26 mm, 6 mm larger than this circumference in the average normal globe.

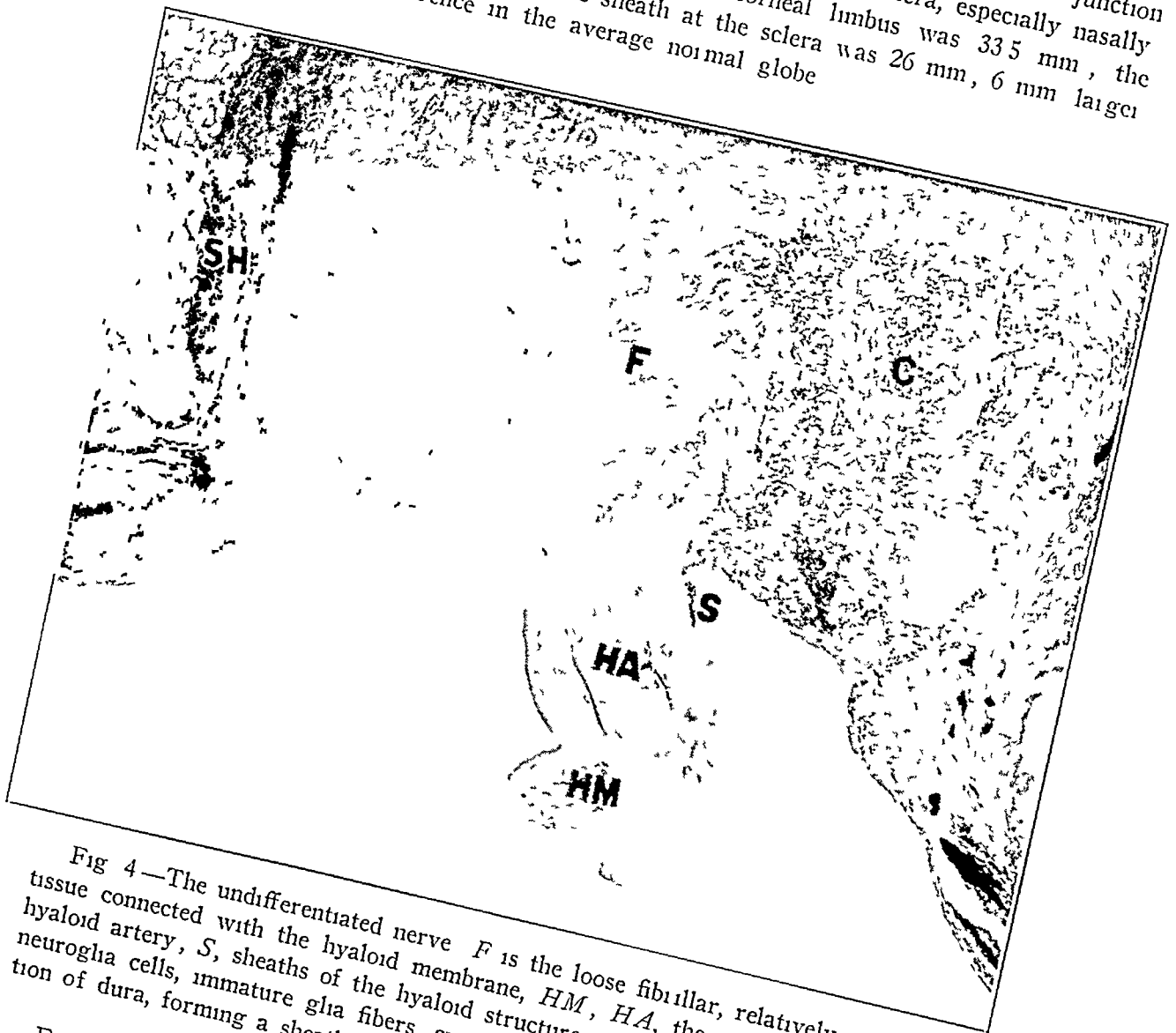


Fig 4—The undifferentiated nerve *F* is the loose fibrillar, relatively acellular tissue connected with the hyaloid membrane, *HM*, *HA*, the endothelium of the hyaloid artery, *S*, sheaths of the hyaloid structure. The tissue, *C*, contains large neuroglia cells, immature glia fibers, cystic spaces and pigment. *SH* is a reflection of dura, forming a sheath for the undifferentiated nerve.

Fixation of the globe was done in Bouin's solution, and serial sections were prepared in pyroxylin and paraffin and were stained with hematoxylin and eosin, Van Gieson's stain and Weigert's elastica.

Microscopic Structure—Globe. The anterior portion of the globe was normal except for a moderate degree of myopic stretching. In the retina, temporal to the macula, there were a few cystic spaces in the outer layers.

Colobomatous Area The scleral canal was considerably widened, the distance between the scleral promontories measuring 3.5 mm. The section through the approximate center of the nerve revealed a large defect at its bulbar end. Here the nerve was replaced by a poorly developed undifferentiated tissue. This tissue was arranged in three zones as follows: 1. Centrally there was a loose fibrillated syncytium, which stained poorly with eosin and was practically avascular and



Fig. 5—Section stained with Weigert's elastica to show splitting of the elastic lamina, LC, as it crosses upward toward the definitive nerve. Some of the fibers are attached to the scleral promontory, S, others can be seen in the dural sheath, lining the undifferentiated nerve.

acellular. 2. Extending from this central tissue, and for 1.5 mm laterally and backward, was a poorly organized mass of nerve tissue, the periphery of which consisted of irregular bundles of nerve fibers intermingled with poorly stained nerve septum fibers and large glial cells. Among the nerve bundles were irregular cystic spaces of various sizes. The poorly developed nerve tissue was continuous with the retina anteriorly and with the true nerve posteriorly. 3. The peripheral

zone consisted of a broad sheath which was continuous with the pia posteriorly and merged at its bulbar attachment with the chorioretinal tissue. The inner aspect of this sheath was lined with masses of pigment resembling pigment epithelial tissue.

Behind this mass of undifferentiated tissue there was an abrupt transformation of the definitive nerve at the lower border of the lamina cribrosa. The central acellular tissue was continuous with a membranous mesenchymal structure, which projected for about 15 mm into the vitreous, stained somewhat lighter and was more homogeneous than the structure in the disk. In the center of this tissue was an endothelial lining, undoubtedly a remnant of the embryologic hyaloid vascular system. For a short distance in the vitreous this structure was bordered by a sheath of glial tissue.

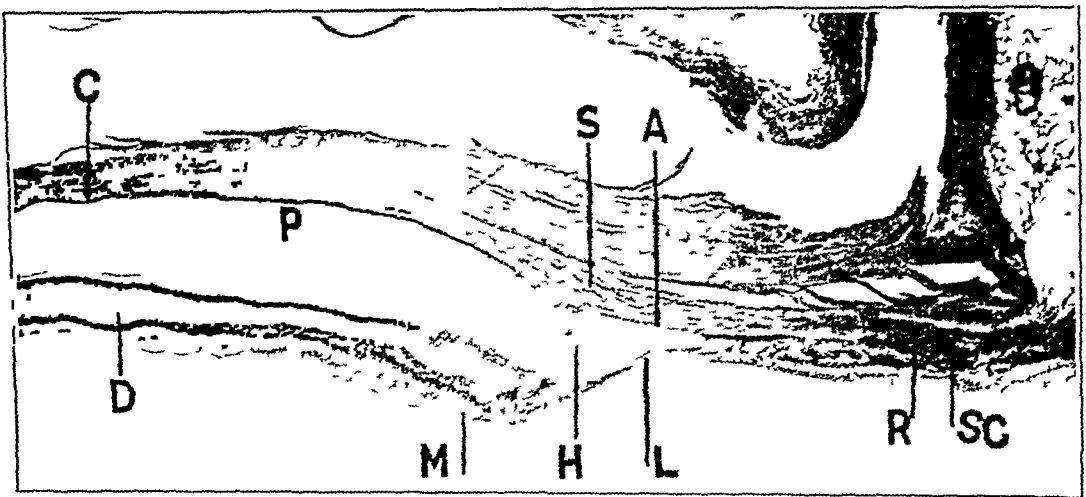


Fig 6—Partial choroidal-macular coloboma. *C* indicates the normal choroid with pigment epithelium, *P*, *S*, absence of the choroidal structure with replacement by elastic fibers, *A*, absence of pigment epithelium, *D*, cystic degeneration of the retina adjoining the macula, *M*, the macula, *H*, a hole in the macula, *L*, the internal limiting membrane, and *SC*, a portion of sclera enclosing layers of retinal pigment, *R*.

Lamina Cribrosa The most anterior limit of the laminated fibers was situated 2 mm behind the point where the hyaloid artery entered the hyaloid sheath and formed the upper limit of the poorly developed nerve. Centrally, the lamina was widened due to an abnormal separation of its elements. Its uppermost fibers arched abruptly downward, especially on the temporal side, and were continued in the outer portion of the thick fibrous sheath which enveloped the poorly differentiated nerve below. Nasally, the laminated fibers turned outward, but less abruptly than temporally, and projected as finger-like processes into an intervaginal space (fig 3). At some points these projections actually crossed the space and could be traced as far forward as the scleral promontory. Most of the fibers lay free in the space, however, and mingled with a meshwork of fibrillar tissue. The bulk of the middle and lower fibers deviated sharply forward into tissue, forming a sheath for the undifferentiated nerve. The exact termination of the lamina cribrosa could not be ascertained, but it appeared that the greater bulk arose from the region of the scleral promontory.

Nerve Sheaths The intradural space about the malformed nerve was considerably wider than about the normal nerve above. The dural sheath on the temporal side was inclined outwardly, at the disk it formed an angle of 30 degrees with the nerve and bulged farther from the nerve as it continued backward. At the point of junction of the coloboma with the true nerve, it inclined nasally. On the nasal side, the dural sheath showed only a slight outward displacement.

The sheaths about the true nerve were normal in structure. At a point 2.5 mm from the entrance of the nerve the arachnoid and the pia turned sharply outward and downward and merged with the dura. There was thus formed an artificial

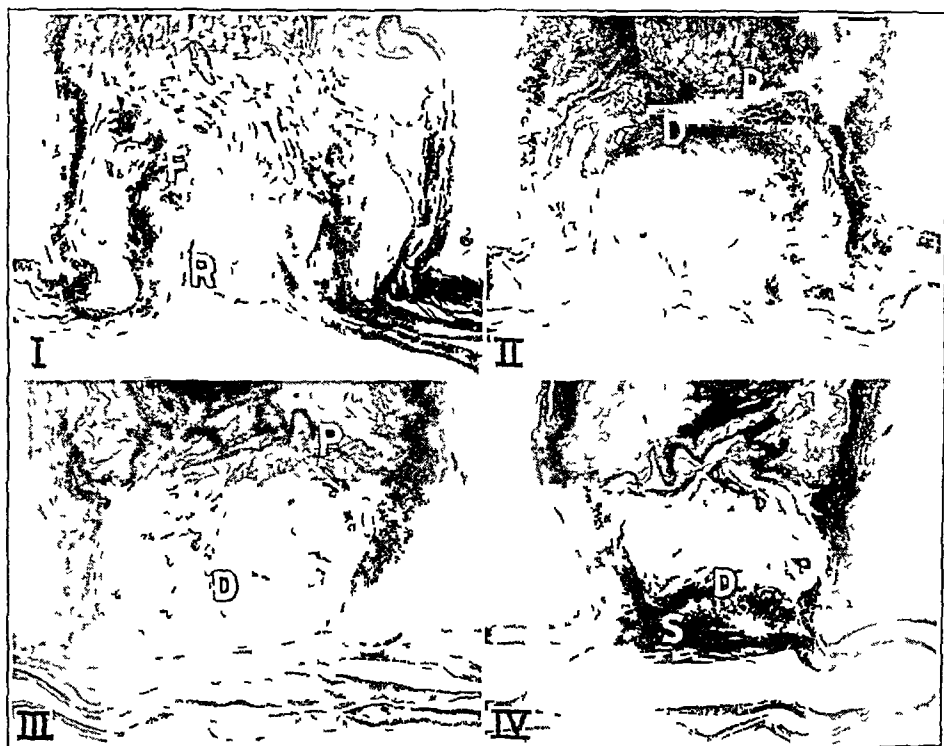


Fig 7—Section through the intermediate, upper and lower levels of the disk (The levels are indicated in figure 8) I is the intermediate level above or below the central hyaloid area (Van Gieson's stain) *F* indicates the nerve fibers in the undifferentiated nerve, *R*, the retinal vessels at the periphery of the nerve. II is the level near the edge of the disk. *P*, the pia of the normal nerve and *D*, the dural sheath of the undifferentiated nerve, merge. Retinal vessels emerge at this level. III is the section at the edge of the disk. *D* indicates the sheath of the undifferentiated nerve surrounded by a meshwork of dural fibers containing blood vessels derived from the central retinal artery in the normal nerve, and *P*, the pia of the nerve above. IV is the section at the outer edge of the disk, indicating fusion of the dural meshwork, *D* with the sclera at the margin of the disk, *S*.

or false vaginal space anterior to this point of junction. The outer edge of this space consisted of perceptibly thinned dura, while its inner side was made up of an irregular fibrous sheath which lined the undifferentiated nerve. This sheath resembled closely the outer dural sheath of the nerve. Its outer portion,

in addition to its fibrous structure, was rich in elastic fibers. The bulbar or anterior end of the space was formed by the union of the scleral promontory with the sheath of the undifferentiated nerve. This portion of the space was considerably wider nasally than temporally. The space itself was filled with a syncytium resembling areolar tissue but which in reality consisted of thin fibro-cellular septums rich in capillaries, and which, on account of its elastic structure and its intimate connection with the outer dural sheath membrane, was undoubtedly

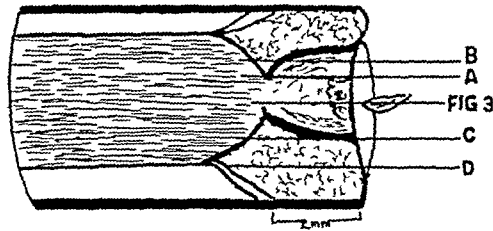


Fig 8—Diagram of the coloboma. A, B, C and D, respectively, indicate the levels at which sections I, II, III and IV in figure 7 are taken. The coloboma comprises a central undifferentiated nerve tissue containing remnants of the hyaloid system and a space surrounding it on all sides. In figure 3 this space is termed the "false intervaginal space."

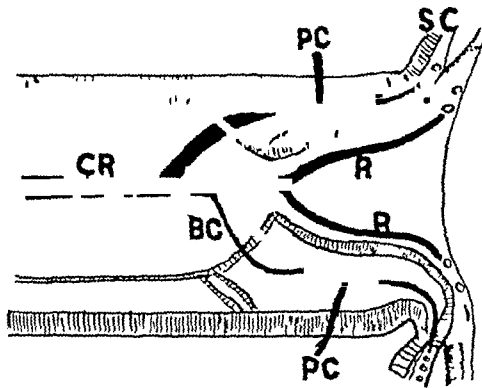


Fig 9—Diagram of the vascular supply of the undifferentiated nerve and surrounding space. S indicates the sclera, C, the choroid, R, the retina, and CR, the central retinal vessel in a normal nerve, BC, the branches of the central vessel, pierce the pial sheath and enter the space about the undifferentiated nerve and communicate with short posterior ciliary vessels, PC. These enter the choroid. Retinal vessels, R, occupy the periphery of the undifferentiated nerve and emerge into the retina at the edge of the disk.

dural tissue intermingled with fragments of the lamina cribrosa, torn from their dural attachment. Large blood vessels, each with a dura-like tissue about them, were also present in the spaces. These blood vessels communicated with the short posterior ciliary vessels which entered the artificial vaginal space through the dura. Anteriorly they communicated with the vessels of the circle of Zinn.

Macular Area. The temporal edge of the macula (artificially detached from the choroid) was normal except for disarrangement of Henle's layer. The nasal portion was thin, and its elements were irregularly arranged. The foveal region was represented by a clear space lined in front by the anterior limiting membrane. The cone layer, the pigment epithelium and the lamina vitrea were absent in the

macular region Beginning at the temporal edge of the macula, the choroid was also maldeveloped Only an occasional vessel was seen Pigment cells were sparse, and the stroma was replaced, as it were, by an overdevelopment of the laminated fibers of the suprachoroidea which appeared to have replaced the choroid for the most part Above and below the macular area the choroid was well developed and normal in all respects Nasal to the fovea, the pigment layer of the retina reformed and near the edge of the optic canal became reduplicated and enveloped bundles of sclera-like tissue

Peripapillary Zone This area was distorted due to the enlargement of the optic canal, the interposition of a pseudovaginal space on either side and the presence of a separate dural sheath about the undifferentiated nerve Temporally, the retina was fused with the thin undifferentiated choroid, and opposite the scleral promontory it was continuous with the dural sheath of the false nerve Nasally, the retina was normal The choroid was well formed and jutted forward and turned at right angles to join the sheath on this side It thus formed the anterior boundary of the false vaginal space nasally

COMMENT

The anomaly described was essentially a coloboma of the nerve at its entrance, the nerve being replaced by rudimentary retinal tissue Associated with this defect were the persistence of remnants of the hyaloid system, widening of the scleral canal, dislocation of the lamina cribrosa and a choroidomacular coloboma

It is possible, of course, to ascribe the presence of rudimentary nerve tissue to the ingrowth of primary retinal layers into the optic stalk In the presence of this abnormal circumstance, persistence of the hyaloid system might well result This explanation, in which the hyaloid system assumes a secondary rather than a primary rôle in the formation of the coloboma, would appear to be more attractive What cannot be explained from the microscopic study, however, is the relation of the undifferentiated nerve to the widening of the optic canal and distortion of the associated structures, unless it is explained on the basis of disturbed growth relationship

The inclination of the nerve nasally suggests that a detachment began on the temporal side, and a continuation of this mechanical force produced not only separation of the lamina cribrosa from its scleral attachments but a dehiscence of a portion of the dura as well Only in this manner can one explain the dura-like sheath of the undifferentiated nerve The intervening space thus produced would appear to consist of fragments of dura and lamina However, whether this deviation of the nerve was a primary force or whether it ensued as a result of the outward displacement of the dura and widening of the optic canal cannot be definitely stated The enlargement of the globe would follow on the weakening and consequent ectasia of the sclera at the edges of the canal

ACUTE ALCOHOLIC AMAUROSIS

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AND

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Six cases of total but temporary blindness associated with acute poisoning due to ethyl alcohol have been observed by us during the past two years. Five of these were seen in the wards of the medical service of the psychiatric division of the Bellevue Hospital during the year ending Dec 31, 1937. The history was that during or soon after the ingestion of alcoholic liquor the patient suddenly became blind. By the time these patients reached us, usually within a few hours, examination revealed total blindness in both eyes, normal pupillary reactions to light and convergence (as in the amaurosis associated with uremia) and normal fundi. Complete recovery, as a rule, followed within twenty-four hours, in 1 case, however, improvement was more gradual.

We believe that this condition develops with sufficient frequency to merit consideration whenever sudden blindness occurs in association with alcoholism. We are, therefore, presenting the findings in 4 typical cases.

REPORT OF CASES

CASE 1—S C, a 33 year old man, was admitted to the medical service of the psychiatric division of the Bellevue Hospital on Sept 15, 1937, because of the sudden onset of total blindness twenty minutes before admission. His history included addiction to alcohol of at least ten years' duration. During the past six months he had been consuming about 1 quart of whisky daily and eating little. During the past year he had had four similar attacks of blindness, each of which cleared up completely and spontaneously within twenty-four hours. Shortly before the onset of the present attack of blindness he had taken several drinks of whisky.

On admission he was confused, disoriented, confabulating and totally blind. There was complete paralysis of all the extraocular muscles. The pupils were equal and reacted to light and convergence. The fundi were normal. The patient had pellagra, as manifested by the characteristic glossitis, stomatitis, dermatitis and diarrhea. There was no peripheral neuritis.

Within twelve hours the external ophthalmoplegia had disappeared, and vision had returned. Marked nystagmus was present by this time. During the following

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two days the patient became more severely ill. The picture of acute alcoholic encephalopathy with convulsions and paresis of both external rectus muscles with esotropia developed. Vision was 10/200 in each eye. During this period the patient had received a diet low in the pellagra-preventive factor. He was then given, in addition, 18 Gm of *vege*¹ by mouth and 10 mg of synthetic crystalline vitamin B₁² by parenteral administration daily. Four days after the institution of this regimen the paresis of the external rectus muscles and the acute encephalopathic picture had disappeared, the glossitis, stomatitis and diarrhea had cleared, and the dermatitis was considerably improved. The patient continued to manifest a Korsakoff psychosis. Twelve days after admission the vision was 16/30 in the right eye and 16/40 in the left eye. Because of the psychosis it was not possible to test the visual fields satisfactorily.

CASE 2—L S., a 35 year old taxi driver, was admitted to the medical service of the psychiatric division of the Bellevue Hospital on Dec 11, 1937, because of the sudden onset of total blindness. His wife stated that two years previously he had been hospitalized for eight days because of an injury to the skull. One and one-half years previously he had been hospitalized again because of trauma to his arm and chest and perhaps to the head. He usually took alcohol only in moderation. At about 3 a m on the day of admission he began drinking "corn whisky." He had imbibed from 1 to 2 pints of this when, at 7 30 a m, he stumbled over a chair, fell to the floor and realized that he was totally blind.

Examination when he was admitted showed a well developed, obese man, who did not appear ill but complained of headache and blindness. He was unable to perceive light. The right pupil was slightly larger than the left, but both were regular in outline and reacted promptly to light. There was a coarse bilateral nystagmus. Bilateral subconjunctival hemorrhages were present. The fundi were normal. There were no signs of peripheral neuritis. The blood pressure was 106 systolic and 76 diastolic. The patient was afebrile and clear mentally.

Analysis of a sample of the corn whisky consumed by the patient showed absence of methyl alcohol.

Two days after his admission there was perception of light in the right eye, but the left eye was still totally blind. His vision did not improve within the next eight days. Ten days after admission 50 mg of thiamin (crystalline vitamin B₁)² was administered once daily by intravenous injection. This was continued until a total of 350 mg was given. Three days after the institution of this therapy the patient was able to discern moving objects with the right eye, five days later vision in the right eye was 20/15, and the visual field was normal. The left eye was first able to see light seventeen days after the onset of the amaurosis, two days later vision in that eye was limited to counting fingers at 1 foot (30 cm). The vision in the left eye remained the same for five days, and then the patient claimed that the eye was totally blind. The vision in this eye continued to be nil for two weeks. During this time the patient was having many financial worries. All tests for hysterical amblyopia gave negative results, but the pupils were equal in size and reacted to direct and consensual light equally. He complained of headaches radiating from the frontal to the occipital region and said that they were severe in the latter region. At the end of two weeks, while the examiner was testing the patient with complementary colors in another effort to detect hysterical amblyopia in the left eye, the patient admitted

1 The *vege* was supplied by *Vege*, Inc.

2 The vitamin B₁ was supplied by Merck & Co.

seeing very blurred objects. At that moment he was told that he would rapidly recover his vision. A solution of silver nitrate strong enough to produce pain was applied to the lids of the left eye. The vision became 20/70 in ten minutes. In thirty minutes it was 20/40, and the visual field was normal. The patient stated that vision in the left eye had never been as good as that in the right, but on the following day it was 20/30. He has retained vision of 20/15 in the right eye and of 20/30 in the left eye.

CASE 3—L. N., a 39 year old white man, was admitted to the medical service of the psychiatric division of the Bellevue Hospital on the night of Dec. 15, 1937, because of the sudden onset of total blindness four hours prior to admission. The history was that at about 4 p. m. he had drunk three or four bottles of beer with his supper. While playing with his children after supper "something snapped," and he became totally blind. He was brought to the hospital in an ambulance. He had never had a similar attack, but three days previously he had noticed that his eyes watered and that his vision was slightly blurred when he tried to read.

He had a chancre in 1918, which was treated by cautery alone, meningitis in 1936 and frequent traumatic injuries to his head, none of which was so serious as to necessitate hospitalization. He was employed as a sheet metal worker for many years and recently had been doing a great deal of soldering in a poorly ventilated room. He and his fellow workers were often forced to take rest periods because of the irritating fumes, but he had never heard of attacks of blindness in any of the other employees. He admitted that it was his habit to drink three or four bottles of beer daily with his evening meal, but no further history indicative of addiction to alcohol could be elicited. He was supported in this history by his wife. Two weeks after he was discharged, however, he presented himself for examination because of pain in his chest. He then appeared to be acutely intoxicated, and he admitted imbibing 2 jiggers of gin before coming to the clinic. He again stated that he was not addicted to the use of alcohol.

When admitted to the hospital on December 15 he appeared well developed and well nourished and not acutely or chronically ill. He was totally blind. The right pupil was larger than the left, both were regular and reacted promptly to light. There was no palsy of the extraocular muscles and no nystagmus. Ophthalmoscopic examination showed a normal fundus. There was an indentation in his forehead caused by an old injury. All the superficial and deep reflexes were intact, and there were no abnormal reflexes. There was no evidence of peripheral neuritis. The blood pressure was 130 systolic and 90 diastolic, the pulse rate was 80, and the temperature was normal. The patient was clear mentally.

The following morning, December 16, he stated that he had awakened at about 4 a. m. and found his vision to be normal (about twelve hours after the onset of blindness). Examination at 10 a. m. revealed vision of 20/20 in each eye. The optic disks and the fundi were normal. The right pupil was larger than the left, but both reacted to light and in convergence. The visual fields were found to be normal, and there were no scotomas. At no time did the patient show any other neurologic abnormalities or encephalopathic manifestations.

Analysis of the urine failed to reveal the presence of lead. Examination of the stained blood smear revealed no stippling or other abnormalities. The Wassermann reactions of the blood and spinal fluid were negative, and the colloidal gold curve of the spinal fluid was normal. The vision has remained normal.

CASE 4—J. B., a 30 year old Jewish man, was brought to the Institute of Ophthalmology of the Columbia Presbyterian Medical Center on April 5, 1936, totally blind and mildly intoxicated. He had been addicted to the use of alcohol

for at least ten years, consuming an average of from 1 to 2 pints of whisky daily. His parents had hospitalized him in various institutions for the treatment of alcohol addiction, without effect. The history as obtained from the patient and his parents was that he had drunk about 2 quarts of whisky during the preceding two days. His father then confined him to a locked room to prevent him from drinking. The patient, however, found some "Whiz," an antifreeze mixture for automobile radiators, in a closet of the room and drank a quart of this as well as 10 cc of perfume. Two hours later he became totally blind. He had no headache and did not vomit but acted intoxicated. An oculist who was consulted told the family that the patient would never see again. Eight hours after the consumption of the "Whiz" he entered the hospital.

Examination revealed a hematoma behind the left ear, the result of a fist fight on the previous day. Both lids of the right eye showed marked ecchymosis due to a fall against a bedpost that day. Vision was nil in each eye, but the pupils were round, about 3 to 4 mm in size, and reacted promptly to light and in convergence. The possibility of hysteria or malingering was considered. The patient's answers and actions at all times, however, were consistent with those of a man totally blind. He failed to blink when an object was suddenly passed in front of his eyes, he blundered into a wall when permitted to walk unaided, he pointed toward the examiner when asked where the latter was in the room, because he could tell from what direction the voice came.

An infusion of saline solution was given, and sedatives were administered because of his great anxiety over his blindness. Inhalations of carbon dioxide and oxygen were given for ten minutes every four hours, and fluid intake was forced. Twelve hours after entering the hospital the patient had perception of light in each eye, three hours later he could see large objects, and in another three hours the vision was 20/15 in each eye, and the fields of vision were normal.

Analysis of the antifreeze mixture left in the bottle from which the patient had drunk showed that it consisted of from 50 to 60 per cent ethyl alcohol, water and small traces of a denaturing agent, pyridine. Methyl alcohol was not found by chemical examination. The manufacturer of the antifreeze mixture confirmed that it contained no methyl alcohol as then made. Analysis of the perfume, also, revealed absence of methyl alcohol.

At the urgent request of the patient's parents he was told that if he ever drank again he would become blind, and this was emphasized on several occasions. Three weeks after his recovery from the sudden amaurosis he returned, stating that his vision had rapidly failed in the past few hours. He denied that he had been drinking. The vision was approximately 20/200 in each eye, but with encouragement this improved to 20/30 in a few minutes. He finally admitted drinking whisky. The amblyopia was apparently of the hysterical type. The following morning his vision was 20/15. For the past two years the patient has had no further ocular complaints.

Two additional patients with acute alcoholic amaurosis have been seen in the medical service of the psychiatric division of the Bellevue Hospital within the past year. The cases are not reported as they are similar to case 3. In these patients, too, there was total blindness, with active pupils, normal fundi and spontaneous recovery within twelve hours.

COMMENT

In an analysis of 8,800 specimens of illicit liquor, reported by Lythgoe³ in 1928, only 8 were found to contain methyl alcohol. In a similar analysis of 100 specimens of illicit liquor, Hunt⁴ found that only 6 contained small amounts of methyl alcohol. He stated "The often expressed fear of injury to the eyes from such small percentages of methyl alcohol is probably not well founded. To get even 15 cc of methyl alcohol in the samples tested in this study would involve the taking of a surely fatal dose of ethyl alcohol." Hunt concluded that the only poisonous substance of significance in the samples examined by him was ethyl alcohol. Another point of interest was his finding that a sample of genuine bottled-in-bond whisky was slightly more toxic than all but three of the samples of illicit liquor, in which the slightly higher toxicity was due to the higher content of ethyl alcohol. Harger,⁵ also, concluded that the poison in "poison liquor" is usually ethyl alcohol.

During 1937 9,667 persons were admitted to the psychiatric division of the Bellevue Hospital because of alcoholism. Only 2 suffered from methyl alcohol poisoning, both patients had attempted suicide, and had recovered from the effects of the acute poisoning due to the methyl alcohol within twenty-four hours. Neither patient showed any residual pathologic process or any immediate or delayed impairment of vision. Chemical examination of the liquors consumed by our patients with amaurosis was made in the 2 instances in which a sample could be obtained. Both samples showed nothing significant except ethyl alcohol. Methyl alcohol was definitely not present. In 1 other instance the beverage imbibed was of a nationally advertised and widely consumed brand.

From the foregoing facts, it would appear that methyl alcohol poisoning among persons addicted to alcohol is rare and that amaurosis due to methyl alcohol poisoning is a still greater rarity. Temporary total blindness, however, occurred in 0.05 per cent (5 of 9,665) of instances of ethyl alcohol poisoning. The incidence may actually have been considerably larger as many of these patients, owing to the transient nature of the blindness, were probably not seen by the physician while blind, and their history is not recorded.

That ethyl alcohol poisoning may cause temporary total blindness has not been generally recognized. De Schweinitz⁶ made the following

3 Lythgoe, H. C. Character of Illicit Liquor upon Massachusetts Market, *New England J. Med.* **198** 228 (March 22) 1928.

4 Hunt, R. An Examination of the Toxicity of One Hundred Samples of Illicit Liquor, *New England J. Med.* **198** 230 (March 22) 1928.

5 Harger, R. N. What Is the Poison in Poison Liquor? *J. Indiana M. A.* **23** 137 (March 15) 1930.

6 de Schweinitz, G. E. *The Toxic Amblyopias: Their Classification, History, Symptoms, Pathology, and Treatment*, Philadelphia, Lea Brothers & Co., 1896 p. 26.

statement "Now and then acute alcoholism seems to have been responsible for almost complete blindness without ophthalmoscopic change rapidly disappearing under antiphlogistic treatment and total abstinence." He referred to 1 such instance.

Strader⁷ in 1927 reported a case which may represent another instance of acute amaurosis associated with ethyl alcohol poisoning.

A man 24 years of age began drinking "moonshine" at about 2:30 p. m. on Oct. 9, 1926. He had imbibed about 1½ pints by 11 p. m., when he noticed that his vision was failing. Four hours later he was completely blind. He was first seen at 9:30 a. m. on October 10. His vision was reduced to perception of light in each eye. There was moderate congestion of the retinal vessels, otherwise the fundi were normal. He complained of supraorbital headache and was treated by lumbar puncture. By October 12 he was able to count fingers at 6 inches (15 cm.), and one week after the onset his vision was 20/20 in the right eye and 20/20 in the left eye with a —0.50 cvl, axis 90, with normal visual fields and no scotoma. He was seen again on November 29, at which time there was no change in vision or in the fundi.

Strader stated in his report that he assumed that the "moonshine" probably contained methyl alcohol. From the facts cited concerning the toxicity of illicit liquors and because of the resemblance of this case to those observed by us, it is not unlikely that the amaurosis was due to acute poisoning with ethyl alcohol.

The nature or location of the lesion responsible for the transient blindness in these patients cannot be described with any degree of certainty. We believe, however, that the location is most likely central and that it may be classified in that protean and loosely defined group of disease complexes collectively described as "alcoholic encephalopathy."

While hysterical amblyopia subsequently developed in 2 of our patients, we do not believe that this was the initial responsible factor in any of our cases. In the cases reported the onset of blindness occurred while the patients were suffering from acute alcoholic intoxication. Apparently acute poisoning with ethyl alcohol is necessary for the development of this disease. We believe, however, that a background of addiction to alcohol is probably a prerequisite. It is of interest also to note that in all of our cases there was a previous history of trauma to the head. The poor tolerance of ethyl alcohol shown by persons who have had injuries of the skull is well known.

All our subjects recovered, the only constant factor in their therapy being the withdrawal of alcohol. We believe that the therapeutic measures employed in these subjects probably in no way influenced the outcome and that recovery is spontaneous in this disease.

⁷ Strader, G. L. Toxic Amblyopia from Drinking, *Am J Ophth* 10:359 (May) 1927.

SUMMARY AND CONCLUSIONS

We have reported 4 cases of acute alcoholic amaurosis, characterized by total blindness in both eyes, normal pupillary reactions to light and convergence, normal fundi and rapid improvement. We have presented evidence and discussed our reasons for considering this disease a manifestation of acute poisoning with ethyl alcohol. We have pointed out that methyl alcohol poisoning among persons addicted to the use of alcohol appears to be of rare occurrence.

In conclusion, we believe that acute alcoholic amaurosis, although a disease of infrequent occurrence, demands a correct etiologic diagnosis because of the excellent prognosis which can be offered in this disease as compared with the poor prognosis which is generally given in the cases of blindness due to methyl alcohol poisoning.

PANOPHTHALMITIS AND SYMPATHETIC OPHTHALMIA

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It is an old saying that once in a while panophthalmitis leads to sympathetic ophthalmia, whether or not the injured globe has been eviscerated. This is the reason why in Vienna evisceration has been given up, and whenever it is necessary to incise a panophthalmic globe it is customary later to excise the stump. In textbooks it is almost classic to state that in panophthalmitis the purulent process destroys the entire uveal tract and leaves no place for the development of sympathetic ophthalmia. So convinced were the older ophthalmologists of the efficacy of purulent inflammation that, taking the hint from farriers, they introduced setons into injured eyes to produce pus as a prophylactic measure. It is common knowledge in the laboratory that in practically every phthisic globe more or less of the ciliary body and the choroid and also those extensions which the uvea sends into the emissaria of the sclera remain. Apparently the extensive, but at the same time never complete, destruction of the uveal tract is not the sole cause of the degree of protection supposed to be offered by panophthalmitis.

The pathogenesis of sympathetic ophthalmitis has served for decades as a favorite field of speculative research. Most of the known facts concerning this disease are explained by the assumption that the exciting agent is a virus which enters the eye by way of the wound, where it produces the so-called "inoculation chance" of Redslob, from which it spreads to other parts of the uvea of the same eye and from thence by way of the blood stream to the uvea of the fellow eye. In a panophthalmic eye it is believed that the virus may live in a state of symbiosis in combination with whatever germ may be the cause of the purulence.

EXPLANATION OF VARYING AMOUNTS OF UVEA REMAINING IN PHTHISIC GLOBES FOLLOWING PANOPHTHALMITIS

Closed Panophthalmitis—In cases in which the opening in the cornea or sclera is small, the purulent process within the globe goes on under high pressure, destroying in succession the retina

Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 9, 1938

and the choroid, the latter almost never entirely. The sclera finally becomes infected, and being distended by the high intraocular pressure, ruptures at the wound or at the equator. It would seem that the longer the perforation was delayed, the more thorough would be the destruction of the uveal tract. It may be argued that the less uvea left, the less likelihood there is of sympathetic infection. Furthermore, whatever uvea is spared may be so damaged by toxins and high pressure that the virus may not find it a favorable medium for propagation. Closed panophthalmitis is no less a clinical than a pathologic entity. It may manifest itself within twenty-four hours after the injury. The excruciating pain increases with the increase in intraocular pressure. The edema of the bulbar conjunctiva and the protrusion and immobility of the globe indicate scleral and orbital involvement. With the reduction of pressure by incision or spontaneous rupture, the pain promptly abates.

Open Panophthalmitis—In cases in which the opening in the cornea or sclera is large, the formation of pus goes on under no great pressure, and consequently abscesses and necroses in the choroid are likely to be less extensive, much of the toxins being eliminated through the wide opening. The sclera in cases of open panophthalmitis is usually little affected, so that protrusion and immobility are not prominent clinical features.

More of the uveal tract is saved in the open than in the closed type of panophthalmitis, so that of the two types one would consider the open type to be the more dangerous. Under this classification come also cases in which the eyes have gone on to phthisis after destruction of the cornea due to a serpiginous or gonorrheal ulcer, the opening thus brought about having the same effect as a perforating wound.

To be sure the kind of germ that produces the pus and the degree of toxicity come into consideration in accounting for the destruction of the uvea, but the greatest role perhaps is that played by the size of the wound, that is, the extent of destruction of the uvea depends on whether an "open" or a "closed" type of panophthalmitis is present.

INCIDENCE OF PANOPHTHALMITIS IN SYMPATHETIC OPHTHALMIA

Panophthalmitis is of relatively rare occurrence today, not only because severely injured eyes are excised before the infection is far advanced but because the usual infecting germs do not lead to a widespread purulence. However, in regions with warm climates, such as Egypt, panophthalmitis is still relatively frequent, whereas in the United States it probably represents not more than 2 or 3 per cent of all intraocular infections following injuries.

In regard to the incidence of panophthalmitis in cases of sympathetic ophthalmitis, one may glean an idea from the fact that of 101 globes showing characteristic sympathetic tissue, reported in a previous paper,¹ there were only 3 which had been affected with panophthalmitis. In this same material there were 9 instances of typical sympathetic infiltration of the choroid without involvement of the fellow eye. Of these 9, 7 followed common endophthalmitis and 2 followed panophthalmitis. Thus 3 cases of panophthalmitis in a series of 101 cases of sympathetic ophthalmia, that is about 3 per cent, is surely not an

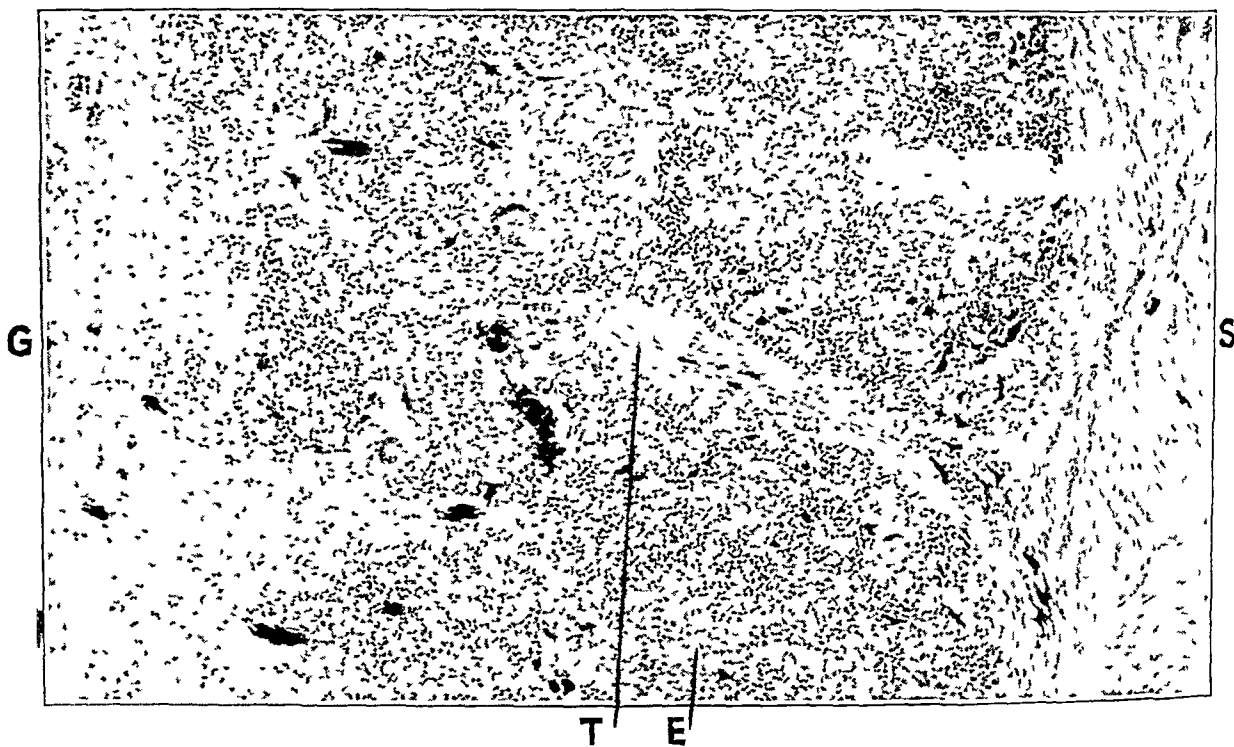


Fig 1—Section of a phthisic globe of a boy aged 14 years with sympathetic ophthalmitis. The cornea was perforated with a knife and the lens extruded. Typical sympathetic nodules were seen in the ciliary body. The section from the choroid is not well outlined because of the panophthalmitis. A large number of nests of epithelioid cells (*E*) lie in the midst of a dense lymphocytic infiltration. Along the internal surface granulation tissue (*G*) mingles with the specific infiltration of the choroid. Along the external surface the sclera (*S*) is inflamed. Extending from the sclera into the specific infiltration, there is a long band of connective tissue (*T*) caused by panophthalmitis.

insignificant figure, this suggests that a panophthalmitic eye may not be so harmless as has been commonly thought.

¹ Samuels, B. Significance of Specific Infiltration at the Site of Injury in Sympathetic Ophthalmia, *Arch Ophth* 9:540 (April) 1933.

DIAGNOSIS OF SYMPATHETIC TISSUE IN PANOPHTHALMITIS

The diagnosis of sympathetic tissue in a phthisic globe following panophthalmitis is beset with difficulties, principally because of the damage done to the choroid by its having participated in the purulent inflammation

To demonstrate difficulties of diagnosing sympathetic ophthalmia in panophthalmic globes, the following 2 cases are reported

CASE 1—*Open panophthalmitis*

A patient aged 66 had spontaneous iridocyclitis with an increase in tension, later an ulcer of the cornea developed, with perforation, followed by panophthalmitis

The cornea was not included in the preparation examined microscopically. The iris and ciliary processes were destroyed. The choroid was widely detached and was enormously thickened. The retina was necrotic. No perforation of the sclera was found, and neither should one have been expected in accordance with the history of destruction of the cornea. A wide detachment of the choroid indicated that some of the contents of the globe had been expelled through the corneal defect, thus accounting for the missing lens.

The stumbling block encountered in the study of this phthisic globe lay in the presence of irregular islands of a reddish color in the midst of a dark blue background of densely packed lymphocytes in a greatly thickened choroid. These islands gave the impression of being islands of epithelioid cells, such as are common in sympathetic ophthalmitis, under which heading the microscopic section was originally filed. In reality, an enormous mass of granulation tissue was being dealt with. What was so peculiar was its presence in the choroid, where granulation tissue is seldom found, and its great extent. This was made possible by the natural looseness of the choroidal stroma allowing a large abscess to develop which organized into granulation tissue. A factor contributing to the expansion of the choroid as the abscess developed was doubtlessly the low intraocular pressure of a panophthalmic eye. The organized mass contained few pus cells in comparison with a vast quantity of lymphocytes. Above all else, there were numerous distended capillaries and solid endothelial buds. These succulent looking vascular elements, taking a reddish stain and occurring in groups, so simulated islands of epithelioid cells as to raise the question of sympathetic ophthalmitis.

In this connection it was recalled that sympathetic tissue is composed mainly of two kinds of cells, namely, lymphocytes and epithelioid cells, with or without giant cells. All other cells, such as plasma cells and eosinophils, are merely accidental, being an expression of another disease, for it should be remembered that sympathetic ophthalmitis is always combined with some other condition. Furthermore, sympathetic tissue, like tuberculous tissue, is remarkable for the sparsity of its blood vessels.

Finally, the granulation showed an unusually small number of fibroblasts, in spite of its great vascularity. In explanation, it may be that with so many blood vessels of all sizes to spring from, endothelial buds and capillaries came into being before there was time for fibroblasts to develop.

CASE 2—*Open panophthalmitis*

A boy aged 14 years two weeks before enucleation of his eye received a stab in the eye from a knife, causing prolapse of the iris, which was excised. Panophthalmitis developed from the infected wound.

Microscopic examination showed the iris to be heavily inflamed, but there were no typical changes. The ciliary body contained numerous pus cells and a number of plasma cells. The appearance of the choroid was so similar to that in case 1 that it came in the same category. The choroid was thickened, and small reddish islands were observed standing out against a bluish background. However, under high magnification the choroid was found to be packed with plasma cells, with relatively few lymphocytes, and the reddish islands turned out to be capillaries and solid endothelial buds. The reaction, as in the former case, was excited directly by toxins from the infected area in the vitreous cavity.

It is interesting that the predominant cells in the granulation tissue in case 1 were lymphocytes and in case 2 plasma cells. Indeed, the almost solid array of plasma cells in case 2 told at once that the condition was not sympathetic ophthalmitis, for however numerous plasma cells may be in sympathetic tissue they never predominate, and the same is true for eosinophils.

CLINICAL IMPORTANCE OF DIAGNOSIS OF SYMPATHETIC OPHTHALMITIS FROM MICROSCOPIC PICTURE

Clinically, the diagnosis of sympathetic ophthalmitis on the basis of microscopic observations plays an important role because it is not always possible to ascertain whether an iritis in the uninjured eye is a genuine sympathetic ophthalmitis or is due to syphilitic or tuberculous iridocyclitis. It is well known that the clinical picture of these types of iridocyclitis often differs but little from that of sympathetic ophthalmitis. The only way to clear this up beyond dispute is to excise the injured blind eye and examine it microscopically. The diagnosis of sympathetic ophthalmitis is of special importance from the standpoint of treatment. In the treatment of ordinary iridocyclitis comparatively mild remedies are usually employed, but in the treatment of sympathetic inflammation the measures may be heroic, for example, when the patient is young and vigorous the malarial cure may be advised. Much may depend on the report from the laboratory.

ORDINARY LYMPHOCYTIC INFILTRATION AND SYMPATHETIC LYMPHOCYTIC INFILTRATION IN THE CHOROID

In the usual cases of perforation complicated by infection in the vitreous the choroid is so well protected from toxins by the retina that

it does not participate in the reaction. There are, however, exceptions to this rule, in which toxins in the vitreous travel to the choroid by a roundabout route. These exceptions follow:

1. When the infection in the vitreous is severe, the toxins diffuse into the papilla and from thence into the choroid, setting up a narrow circumpapillary zone of infiltration composed purely of lymphocytes, because the toxins arrive in a diluted state. The retina seems to offer a greater resistance to the transference of toxins than the papilla, although

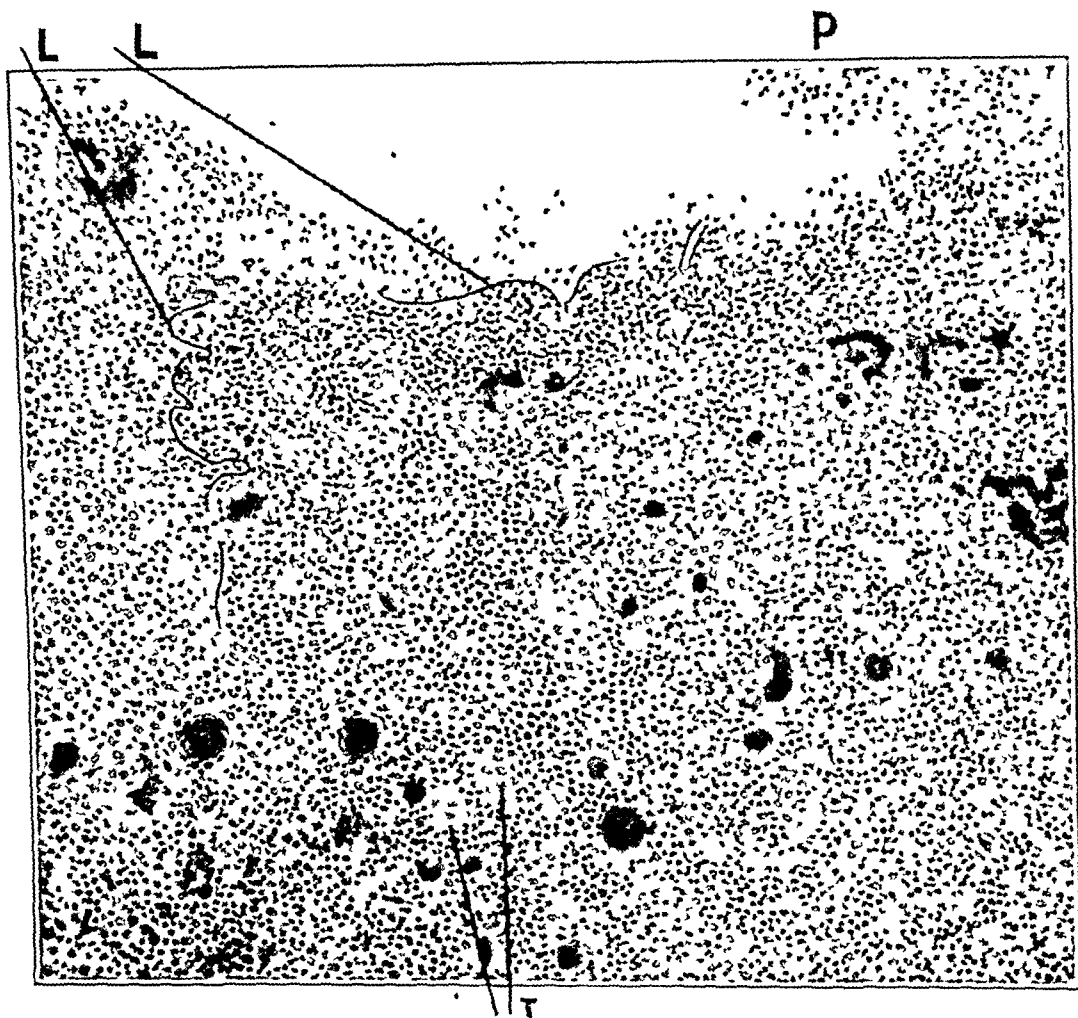


Fig 2 (case 2) —Section from a phthisic globe with granulation tissue in the choroid, simulating sympathetic tissue following panophthalmitis. The choroid can be identified by the presence of a number of wavy remnants of the lamina vitrea (*L*). Above these is the subretinal space, filled with an albuminous fluid containing pus cells (*P*). The thickness of the choroid is greatly increased by the presence of typical granulation tissue. A large number of distended and engorged blood vessels stand out prominently. Scattered about are many irregularly outlined islands of solid vascular endothelial buds simulating islands (*I*) of epithelioid cells characteristic of sympathetic ophthalmia. A great quantity of lymphocytes with relatively few leukocytes is distributed in the granulation tissue, adding to the similarity with sympathetic tissue.

the tissue of the papilla is much thicker. Apparently a more active exchange of fluid goes on between papilla and choroid within the choroidal canal than between the general layout of retina and choroid. It is true the outer layers of the retina are nourished by the chorio-capillaris, and there must be an active exchange of fluid between the tissues, yet a kind of barrier has to be supposed to exist between the inner and the outer layers of the retina along the line where the area of the retinal system of blood vessels touches the domain of the choroidal system. Whatever this barrier is, apparently it is more effective than the lamina vitrea, which is interposed between the chorio-capillaris and the retina.

2 At the same time that toxins may diffuse backward from an infection in the vitreous they may also diffuse forward, producing a reaction in the flat part of the ciliary body, and from thence they diffuse into the choroid, producing a circumscribed lymphocytic infiltration of the choroid at the ora serrata analogous to the circumcapillary infiltration.

3 Whenever the choroid is directly injured through the retina, as by an intraocular foreign body, it is just at the place of impact that a lymphocytic infiltration takes place. Such a reaction gradually abates. This localized lymphocytic infiltration can be readily recognized as an irritation caused by direct injury or secondarily by toxins diffused from the vitreous by way of the damaged retina.

Of paramount importance in deciding whether or not a case of endophthalmitis or panophthalmitis is accompanied by sympathetic infiltration is a study of the condition of the retina. To reiterate, when the retina is intact the choroid should be practically free of reaction in ordinary endophthalmitis, with the three exceptions mentioned. Lymphocytic foci scattered here and there in the choroid under an intact retina, whether the retina is detached or not, after a perforating wound suggest at once the initial stage of sympathetic ophthalmitis, particularly if the cells are typically distributed along the walls of the larger veins. The picture is clearcut, the infiltrated choroid being bounded by an intact retina on the one side and an intact sclera on the other. In comparison, when the retina is destroyed and granulation tissue in the vitreous space mingles with granulation tissue in the choroid or even with genuine sympathetic infiltration, the picture is complicated and calls for special interpretation.

CONCERNING EXPERIMENTAL WORK IN SYMPATHETIC OPHTHALMITIS

Although it may seem a digression from the main subject, I should like to point out some bearing that the material in this paper may have on experimental work in sympathetic ophthalmia that is done on ani-

mals. Infiltrations in the choroid of rabbits have been reported in which after injections of all kinds of substances into the vitreous cavity, including the virus of herpes simplex, the reaction found has been considered almost if not exactly identical with that of sympathetic ophthalmitis in human beings. However, the necrosing effect that these injections may have on the delicate retina seems to have been widely disregarded. There never can be any ground for discussion as to a similarity to sympathetic ophthalmitis if after an injection into the vitreous of a rabbit the retina becomes necrotic, because an infiltration so produced has an absolutely different meaning from that of sympathetic ophthalmitis. It belongs to the realm of panophthalmitis and is the result of a toxic influence directly exerted on the choroid.

Adding more difficulty to a statement regarding the relationship of the condition produced experimentally to sympathetic ophthalmitis is the fact that it is almost impossible by any form of irritation to cause pus cells to be poured out in a rabbit's choroid. Only lymphocytes appear, probably because the choroid of a rabbit is constitutionally more resistant and the circulation more active in carrying away toxins than is the human choroid. Consequently, when a heavy lymphocytic infiltration is present in which groups of capillaries and endothelial buds may imitate nests of epithelioid cells, one may be tempted to make a wrong diagnosis of sympathetic ophthalmitis.

EXPERIMENTAL HYPERTENSION

VIII VASCULAR CHANGES IN THE EYES

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AND

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In healthy dogs and monkeys (macaques) persistent elevation of the systolic and the diastolic pressure, with or without a decrease of renal excretory function, followed permanent partial constriction of the main renal arteries ¹

The eyes of these hypertensive animals have been studied during a period of more than five years. A preliminary report ² and an interim report ³ have been made

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Read before the Section on Ophthalmology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 16, 1938

This study was supported by the Beaumont-Richman-Kohn Fund, and by special grants-in-aid from the Josiah Macy Jr Foundation and Mr N L Dauby, Mr Alex S Winter and associates, Cleveland

1 (a) Goldblatt, H, Lynch, J, Hanzal, R F, and Summerville, W W. Experimental Hypertension Due to Renal Ischemia, *Bull Acad Med Cleveland* **16** 6, 1932, (b) Studies on Experimental Hypertension I The Production of Persistent Elevation of Systolic Blood Pressure by Means of Renal Ischemia, *J Exper Med* **59** 347, 1934 (c) Goldblatt, H, Gross, J, and Hanzal, R F. Studies on Experimental Hypertension II The Effect of Resection of Splanchnic Nerves on Experimental Renal Hypertension, *ibid* **65** 233, 1937 (d) Goldblatt, H. Studies on Experimental Hypertension III The Production of Persistent Hypertension in Monkeys (Macaque) by Renal Ischemia, *ibid* **65** 671, 1937, (e) Studies on Experimental Hypertension V The Pathogenesis of Experimental Hypertension Due to Renal Ischemia, *Ann Int Med* **11** 69, 1937 (f) Goldblatt, H, and Wartman, W B. Studies on Experimental Hypertension VI The Effect of Section of Anterior Spinal Nerve Roots on Experimental Hypertension Due to Renal Ischemia, *J Exper Med* **66** 527, 1937 (g) Goldblatt, H. Studies on Experimental Hypertension VII The Production of the Malignant Phase of Hypertension, *ibid* **67** 809, 1938, (h) Experimental Hypertension Induced by Renal Ischemia, in *Harvey Lectures, 1937-1938*, Baltimore, Williams & Wilkins Company, 1938, p 237

2 Keyes, J E L, and Goldblatt, H. Experimental Hypertension IV Clinical and Pathologic Studies of the Eyes, a Preliminary Report, *Arch Ophthalm* **17** 1040 (June) 1937

3 Keyes, J E L, and Goldblatt, H. Experimental Hypertension Studies of the Eyes, Interim Report, *Tr Internat Ophthalm Cong*, 1937, to be published

These dogs exhibited two definite types of hypertension—a benign phase, without a decrease of renal excretory function, and an acute malignant phase, with very high blood pressure and severe damage of the renal excretory function. These two phases correspond to the benign and malignant phases of essential hypertension in man. The acute malignant phase of hypertension occurred, or was produced, in dogs already hypertensive, owing to constriction of the main renal arteries when the arteries were reconstituted to a great degree, or in healthy dogs in which the main renal arteries were markedly constricted from the beginning.

The group with benign hypertension without a decrease of renal excretory function (by far the larger), after a variable period, showed two types of clinical ocular change. A small number showed early retinal hemorrhages and edema, with early moderate and late severe retinal vascular disease. A larger number showed slowly progressive retinal vascular disease with an occasional small retinal hemorrhage and more rarely, small areas of retinal edema. None of these showed papilledema (figs 1 to 5).

Visible changes in the eyes of benign hypertensive animals were a late manifestation. The earliest and only constant change noted ophthalmoscopically in these dogs was usually an increase in the tortuosity of the retinal arteries. Owing to an anteroposterior, as well as a lateral, tortuosity of these arteries, the light reflex became interrupted, and the vessels presented a mottled appearance. In the monkey tortuosity of the arteries has not been much in evidence. Increase in the intensity and width of the arterial light reflex was, however, an early and persistent sign. Lessening of the caliber of the arteries was easily noticed in the monkey. In the dog, owing to the relatively small size of the retinal arteries, many of which are of the cilioretinal type, small variations in caliber were difficult to visualize. Common to the eyes of the two animals was the occurrence of localized retinal hemorrhages and areas of retinal edema. In the monkey, coincident with or succeeding these changes, increasing evidence of arterial disease was observed. The arteries and arterioles became narrower, the light reflex became wider and more brilliant, and gradually many vessels assumed a copper tint. Several small terminal arterioles resembled silver streaks. Vascular tortuosity and irregularity of outline were noticeably absent. In none of the animals have the retinal veins appeared diseased. In the eyes of hypertensive dogs no arteries resembling straight copper or silver wires or threads have been observed. Other changes seen in the eyes of the dogs have been preretinal, superficial and deep retinal hemorrhages, localized and general edema and detachment of the retina, perivasculitis,

EXPLANATION OF COLOR PLATE

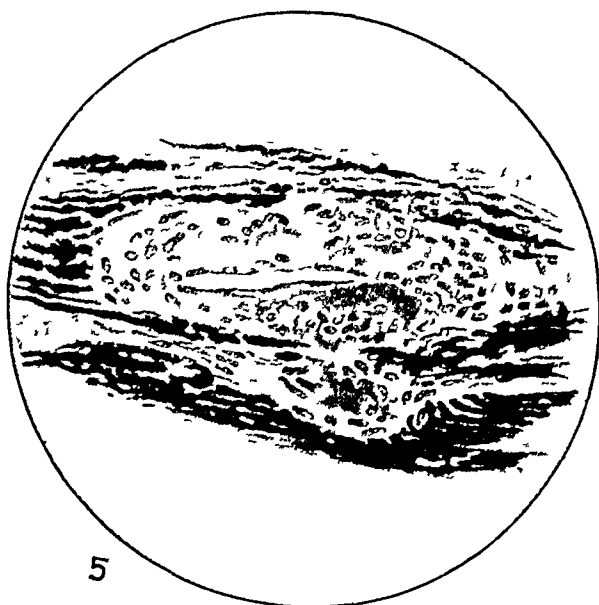
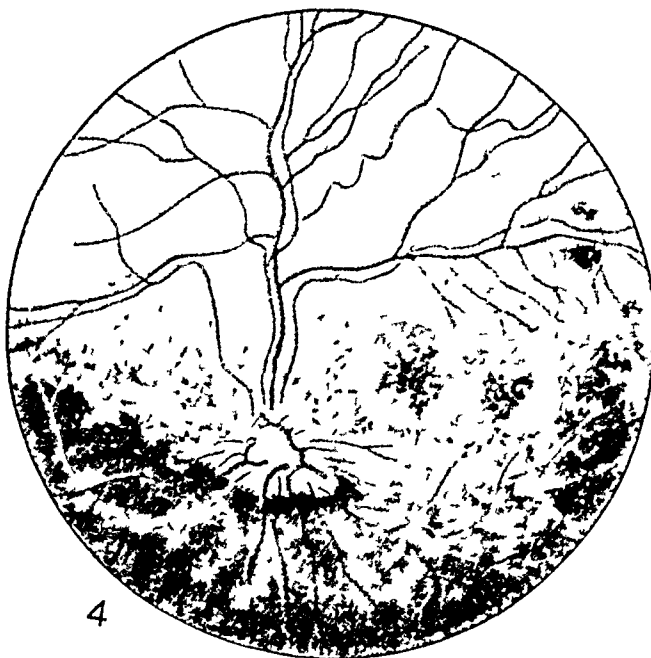
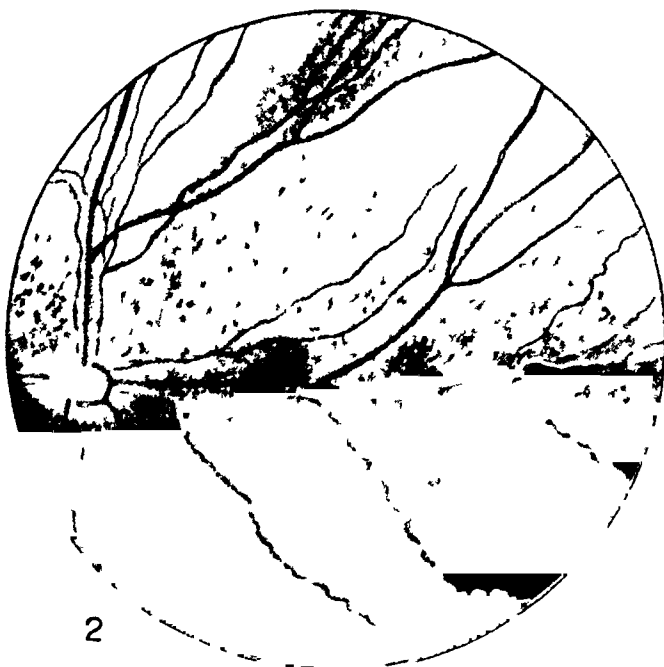
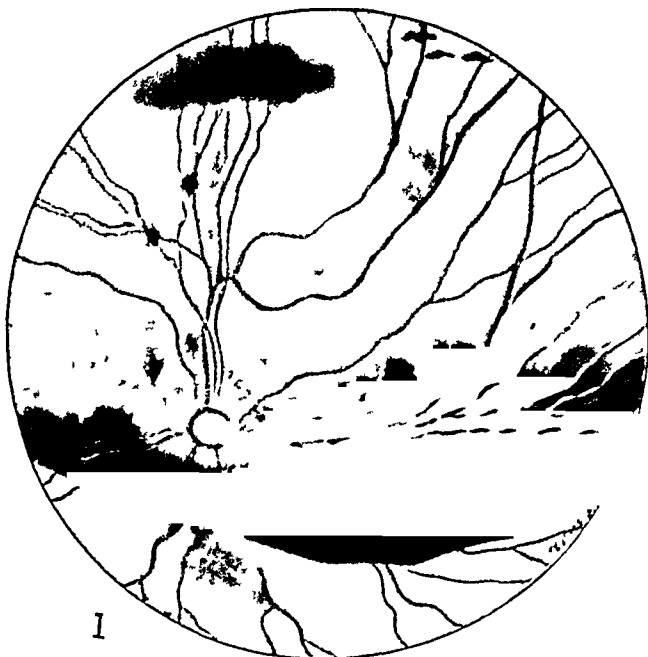
Fig 1 (dog 3-8 R)—Drawing of the fundus of a hypertensive dog, three years after the blood pressure became elevated. Attention is directed to scattered hemorrhages, detachment of the retina and the white perivascular covering on the arterioles. A description of the eyes of this animal has been published.²

Fig 2 (dog 5-9 R)—Drawing of the fundus of hypertensive dog, nearly five years after the final constriction of the renal arteries. Extensive periarteritis was present. Bilateral section of the thoracic portion of the splanchnic nerves and the lower four dorsal sympathetic ganglions was performed four years after the systolic blood pressure became elevated. After this operation the blood pressure remained at its previous high level. The animal lived more than five years after hypertension was established.

Fig 3 (dog 2-10 R)—Drawing of the fundus oculi of a dog with benign hypertension of great degree and of thirty-four months' duration. The brown area is a scar following a previous hemorrhage. Both main renal arteries were successively greatly constricted. The systolic blood pressure rapidly became elevated and is usually in the neighborhood of 300 mm of mercury. Bilateral section of the intrathoracic portion of the splanchnic nerves and the lower four thoracic sympathetic ganglions was without permanent influence on the blood pressure. At the time of writing the dog is still alive.

Fig 4 (dog 2-35)—Drawing of the fundus of the right eye. Brown scars on the tapetum followed retinal hemorrhages. An arteriole on the temporal side of the nerve head had had a perivascular sheath of increasing density for twenty-six months. A gray haze had developed in the retina over diseased nasal arteries. At the time of writing the dog is alive and has a systolic blood pressure in the neighborhood of 230 mm of mercury. After bilateral section of the splanchnic nerves and the lower four thoracic sympathetic ganglions, both main renal arteries were successively constricted. A moderate persistent elevation of systolic blood pressure ensued. A year later laminectomy was performed, and the anterior nerve roots, from the tenth dorsal to the second lumbar inclusive, were severed. There was a temporary lowering of blood pressure, which soon returned to the previous high level.

Fig 5 (dog 2-50 L 46)—Microscopic drawing of a choroidal arteriole. A portion of the wall is necrotic. There are a dissecting hemorrhage through the wall and extravascular hemorrhage.



pigmented and nonpigmented retinal scars, opacities of the vitreous, including synchysis scintillans, and cataract

While it has not yet been encountered in monkeys, perivasculitis similar to that seen in the eyes of hypertensive persons has been frequently noticed in the eyes of hypertensive dogs (figs 1 to 5). An irregular white patch appeared on a retinal artery or arteriole, usually between the origin of the blood vessel at the nerve head and the visible periphery of the retina. Once established, the white area never disappeared, but new areas appeared, coalesced and gradually spread along the artery and its branches. In the earlier stages of sheath formation, the affected blood vessel could be seen through the white covering as a narrow pink streak. Later, when the arterial covering was more dense, the vessel disappeared and was represented by an irregular white streak slightly wider than the original blood vessel. Ophthalmoscopically, the portions of the blood vessel visible through the white covering had a pink color and apparently carried blood. When an artery was completely covered, it was not possible to evaluate its functional condition. The ophthalmoscopic picture presented by perivasculitis was quite distinct from that of the silver wire artery or arteriole. The outline of the silver wire vessel was clean-cut, and the vessel was smaller than it was originally. An arteriole with perivasculitis presented a slightly irregular outline, had a soft moth-eaten appearance and was wider than it was originally.

No monkeys have been observed with malignant hypertension. The eyes of the dogs with acute or malignant hypertension presented a picture of rapid and extensive visual damage. In dog 3-8, coincident with damaged renal excretory function, complete detachment of the retina, massive intraocular hemorrhages and secondary glaucoma with ulceration of the cornea occurred. This dog already had evidence of extensive retinal vascular disease and had been hypertensive for about three years (fig 1).

An outstanding example of the malignant phase of hypertension occurring in an animal with previous hypertension was observed in dog 2-50. The right main renal artery was constricted, and the blood pressure rose to a maximum direct ^{3a} pressure of 240 mm of mercury. The right kidney was then removed. The blood pressure promptly returned to normal and remained normal. The main renal artery of the remaining kidney was greatly constricted. The mean blood pressure rose again and in the course of about one month exceeded 300 mm of mercury. Evidence of disturbed renal excretory power appeared. The animal became uremic. The urea nitrogen content of the blood was 83 mg per

^{3a} By direct or mean blood pressure is meant the pressure determined by inserting a needle connected with a mercury manometer directly into the femoral artery.

hundred cubic centimeters just before death. Blood appeared at the anus, and within a short period before death both eyes became filled with blood. Previous to the malignant phase of the hypertension, the only visible change ophthalmoscopically was doubtful tortuosity of the nasal retinal arteries (fig 6)

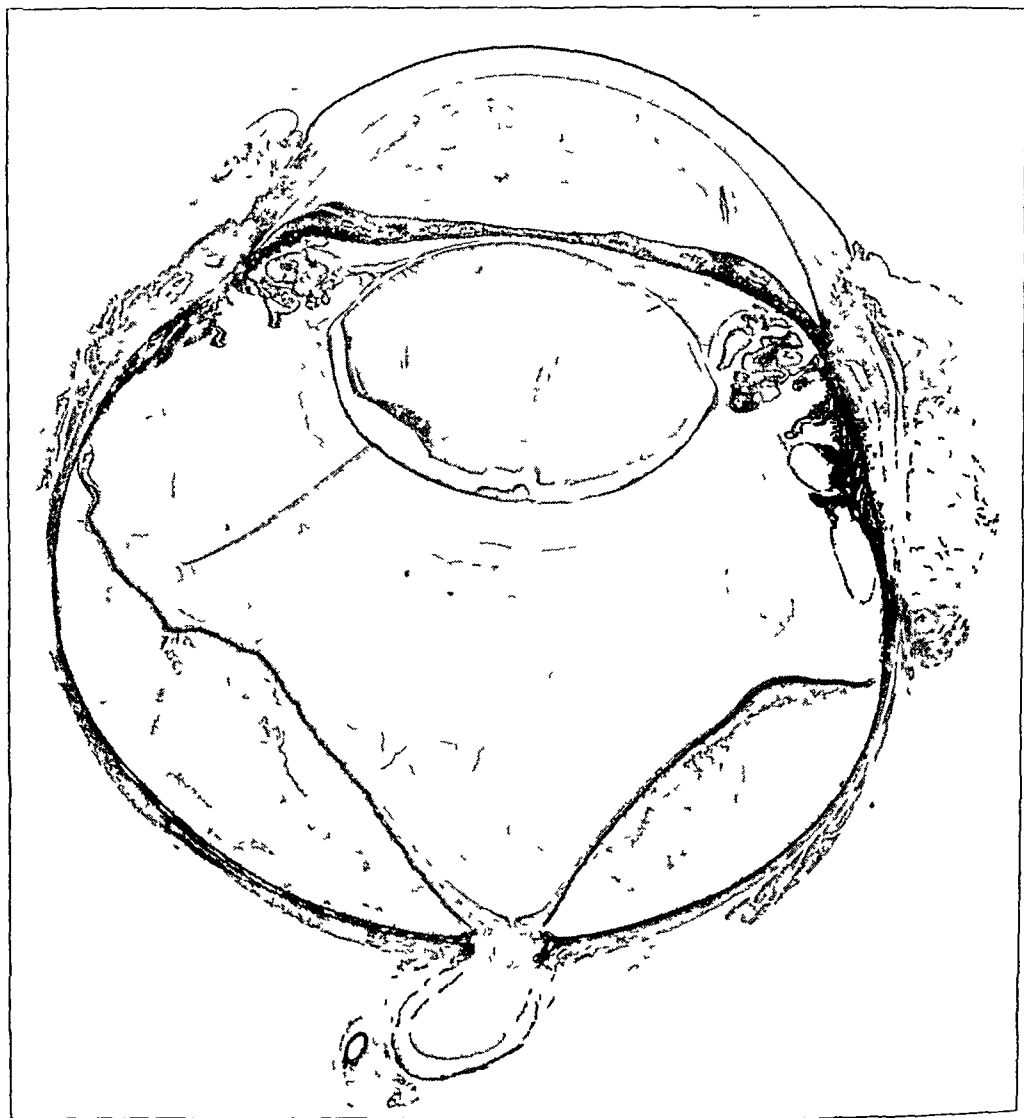


Fig 6 (dog 2-50 L 45) —A section of the left eye of a dog with malignant hypertension following prolonged benign hypertension. The eye contained bloody fluid rich in fibrin. The retina was detached. There was marked edema of the ciliary processes.

An example of the acute malignant phase of hypertension produced purposely by the same sequence of procedures employed in dog 2-50 was observed in dog 3-39. The details of the experimental history of

this animal will be given here because they were omitted accidentally from a previous publication,¹⁸ in which this should have appeared

The dog was a young virgin, black and white mongrel collie, weighing 10 Kg on April 20, 1937, when the determination of the normal pressure was begun. On July 8 the left main renal artery was clamped slightly. The mean blood pressure became elevated, reaching a maximum of 165 mm of mercury. This persisted until July 30, at which time the blood pressure was 160 mm of mercury. On this day the left kidney was removed. The blood pressure on the next day was 150 mm of mercury. With minor variations the blood pressure persisted at the normal level for this dog until September 22, on which day the right main renal artery was greatly constricted. The blood pressure became promptly elevated and rose gradually to 255 mm of mercury, the maximum pressure which was reached on October 25. On this day the blood urea nitrogen was 18 mg and the creatinine 15 mg per hundred cubic centimeters. The next day the urea nitrogen increased to 32 mg and on October 29 it was 150 mg. At this time the animal appeared extremely ill. It died on October 31.

On October 26 the eyes, which previous to the final operation were normal, showed a rapidly increasing edema of the papilla and retina. The tapetum lost its bronze appearance. It appeared gray, owing to diffuse edema. In addition, it was observed that the pupils were unequal, irregular and small. In the right eye small round retinal hemorrhages were scattered along the nasal vessels. On the temporal side of the optic nerve and high up in the retina were small sheets of hemorrhage. The left eye had a few small scattered hemorrhages along blood vessels. Four days later both pupils were widely dilated and inactive to light. In the left eye there were streaks of pale brown bloody fluid on the anterior lens capsule and in the anterior chamber. The details of the fundus were indistinct. The fundus of the right eye was faintly seen. A few new hemorrhages were present. The vitreous was hazy, and the retina was edematous and detached. Tension to touch was normal to minus (fig 7).

The acute malignant phase of hypertension has been produced in several dogs without a previous period of the benign phase by marked constriction of both renal arteries at the same time or with an interval between constrictions and also by great constriction of the aorta just above the origin of both renal arteries. The eyes of these animals have not yet been examined histologically, but the vessels of the rest of the body showed the same changes as in dogs 2-50 and 3-39.

The histologic examination of the eyes of dogs with malignant acute hypertension revealed severe degenerative disease of the arterioles of the uvea, together with extravasations of blood into the uvea and retina, papilledema, edema and detachment of the retina and subretinal and intraocular serofibrinous exudate.

The walls of some arterioles in the retina showed marked edema (fig 8). The arteries and arterioles of the choroid, ciliary body and iris, however, exhibited widespread degenerative changes. There was evidence of collagenous and hyaline degeneration of the wall of the vessel (fig 9), as well as necrosis of the wall, intramural edema and hemorrhage, perivascular hemorrhage and localized areas of lymphocytic perivascular infiltration (figs 5 and 10).

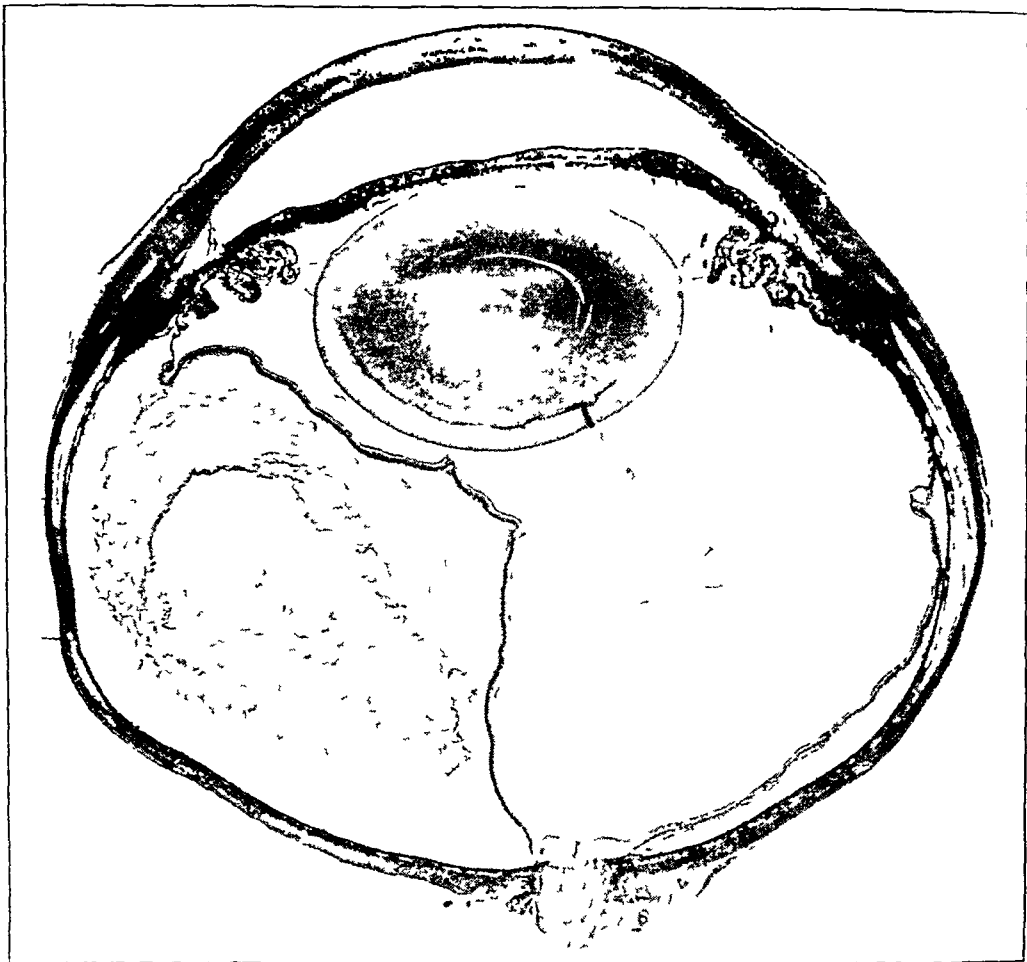


Fig 7 (dog 3-39 L 86) —A section of the left eye of a dog with acute malignant hypertension. There are swelling of the papilla and edema and detachment of the retina and choroid. The eye was filled by a bloody serofibrinous fluid.

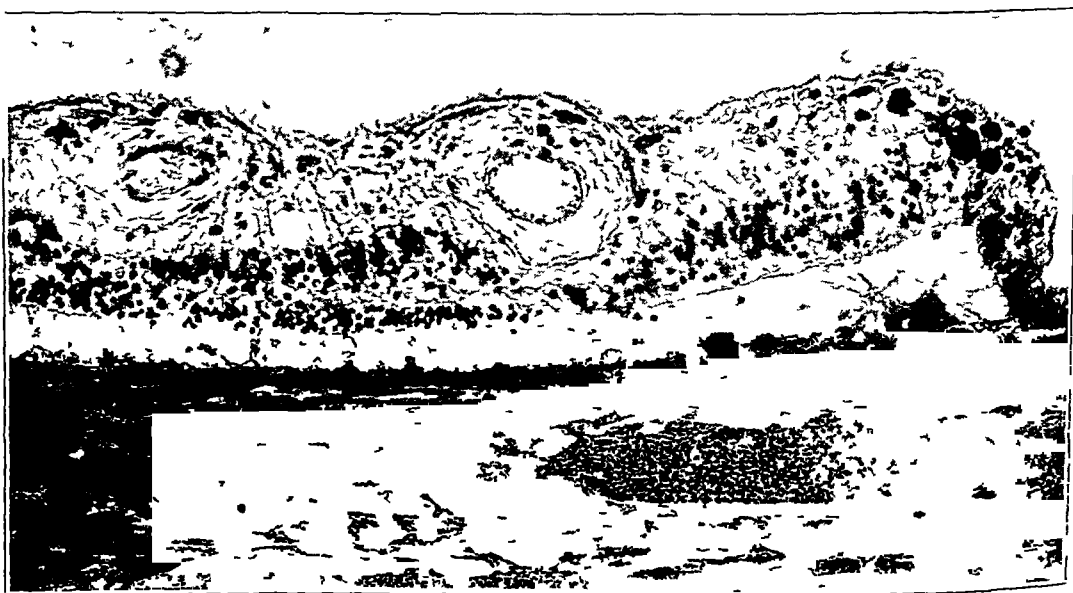


Fig 8 (dog 3-39 R 40) —Two edematous retinal arterioles at the ora serrata, $\times 200$



Fig 9 (dog 3-39 L 40)—Choroidal arteriole with swollen, partly hyalinized wall and collagenous degeneration, $\times 200$

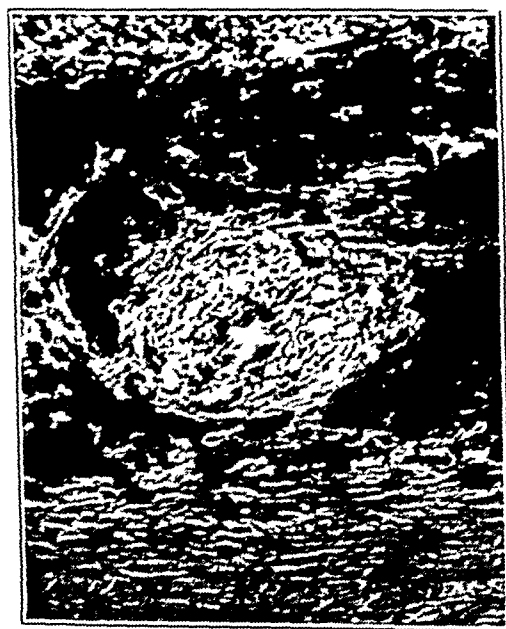


Fig 10 (dog 3-39 R 45)—Choroidal blood vessels showing edema and fibrinoid degeneration of the wall of the vessel, mainly subintimal, $\times 200$

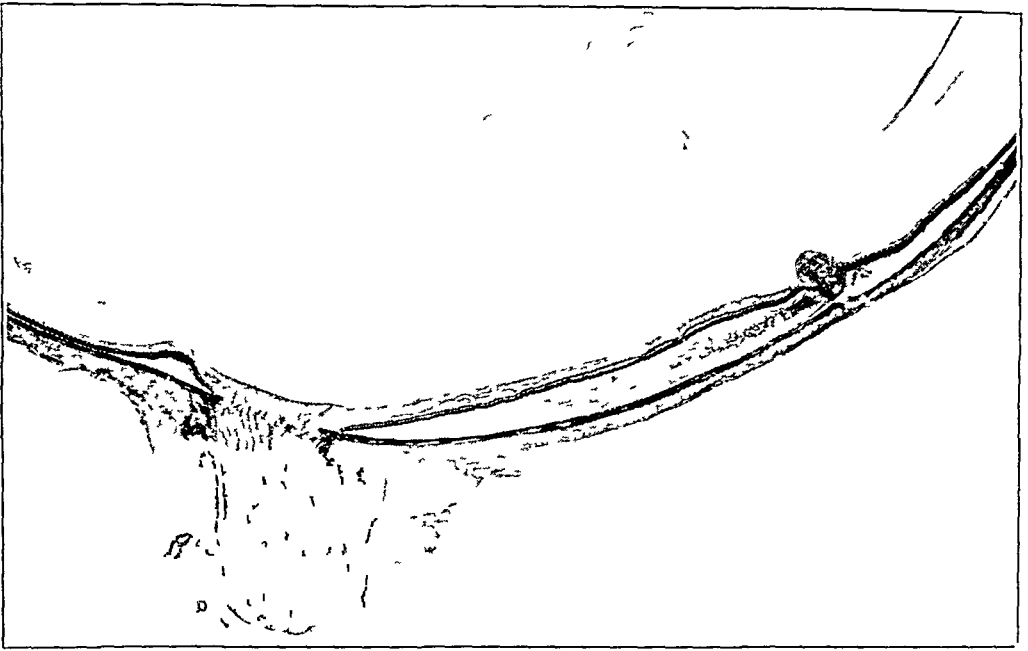


Fig 11 (dog 5-9 R 45-A) —A low magnification of a section of the back of the eye The retina is detached and undergoing degeneration A nodule of unusual appearance is located in the retina



Fig 12 (dog 5-9 R 45-B) —A higher magnification of the nodule shown in the retina in figure 11, $\times 116$ The upper portion of the nodule shows an area of hyalin containing vascular spaces In the lower portion of the nodule is a thin-walled blood vessel, the lumen of which is divided by many partitions

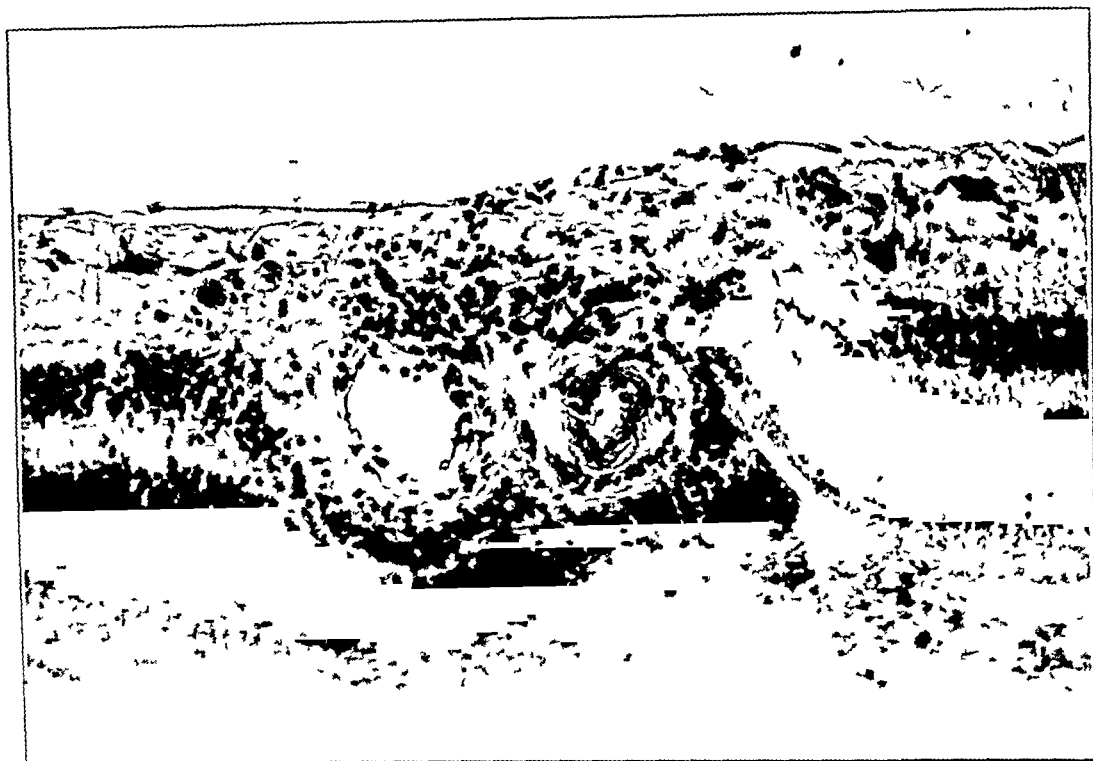


Fig 13 (dog 5-9 R 105)—An arteriole and a vein in a portion of degenerated detached retina, $\times 179$ The wall of the arteriole is unusually thick The quantity of perivascular tissue is also abnormally great

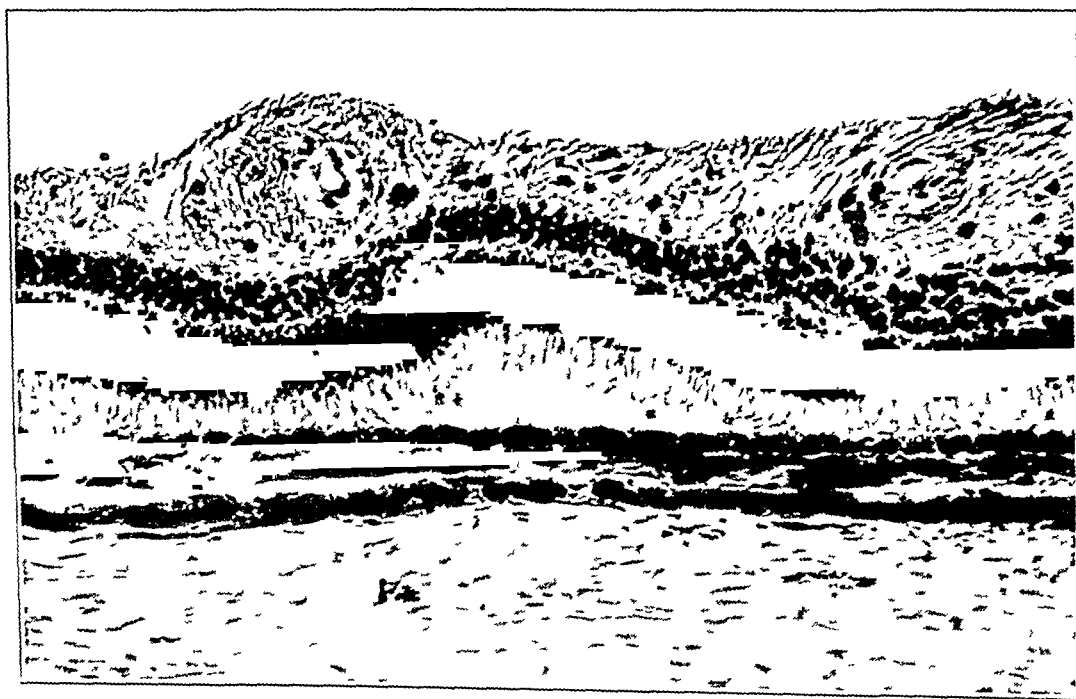


Fig 14 (dog 6 R 18)—An arteriole in a slightly edematous retina Surrounding this diseased arteriole is a large quantity of slightly edematous adventitia Hematoxylin and eosin stain, $\times 173$

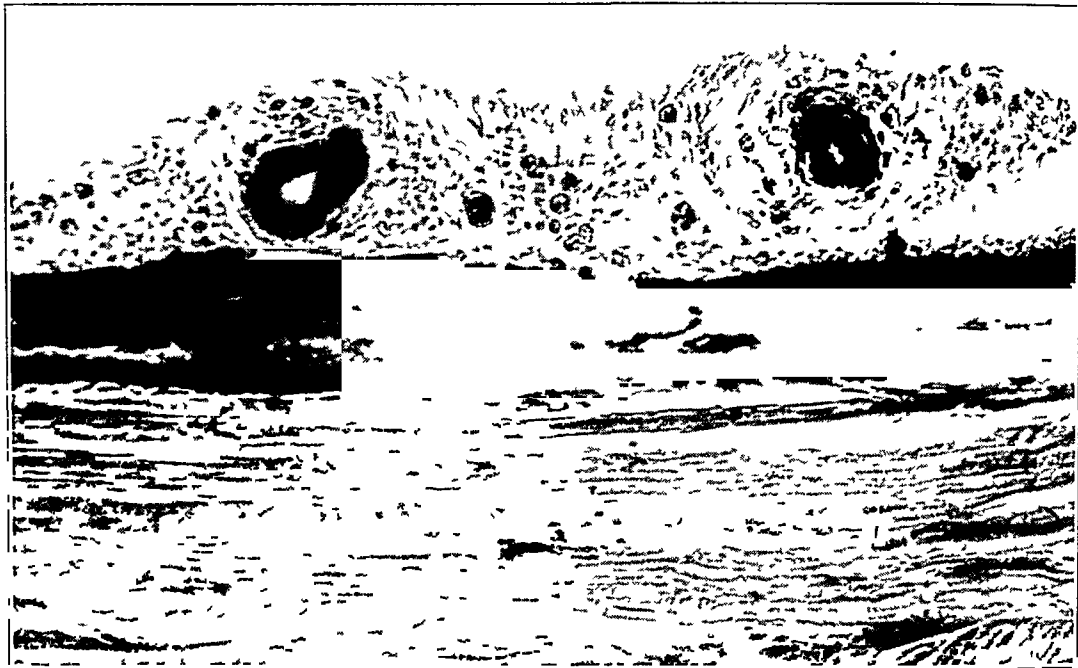


Fig 15 (dog 6 R 17)—A portion of degenerated retina containing two diseased arterioles. Marked proliferation of elastic tissue and perivascular tissue are shown. Weigert's elastic method, $\times 173$.

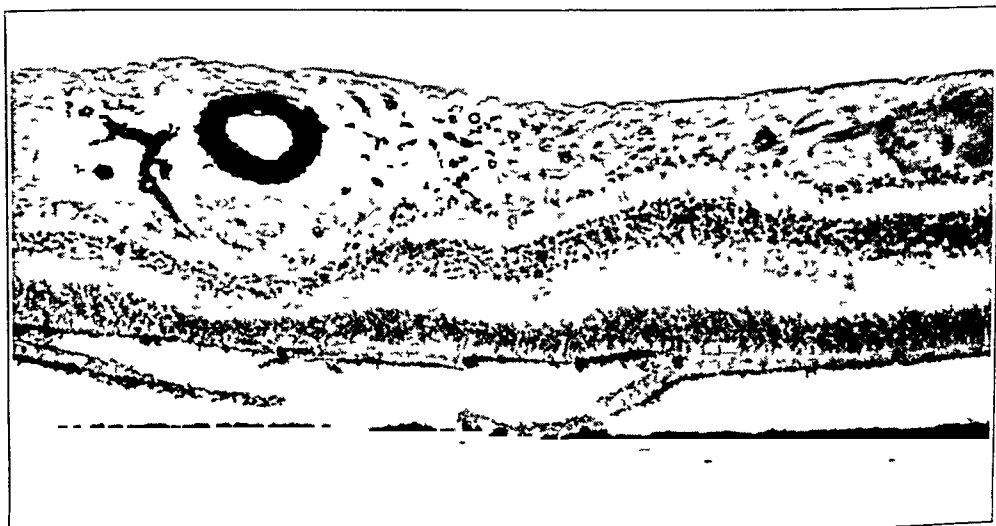


Fig 16 (monkey 5 L 81)—A retinal arteriole showing multiplication of elastic tissue and a thick adventitia. There is marked multiplication of capillaries in this region. Weigert's elastic tissue stain.

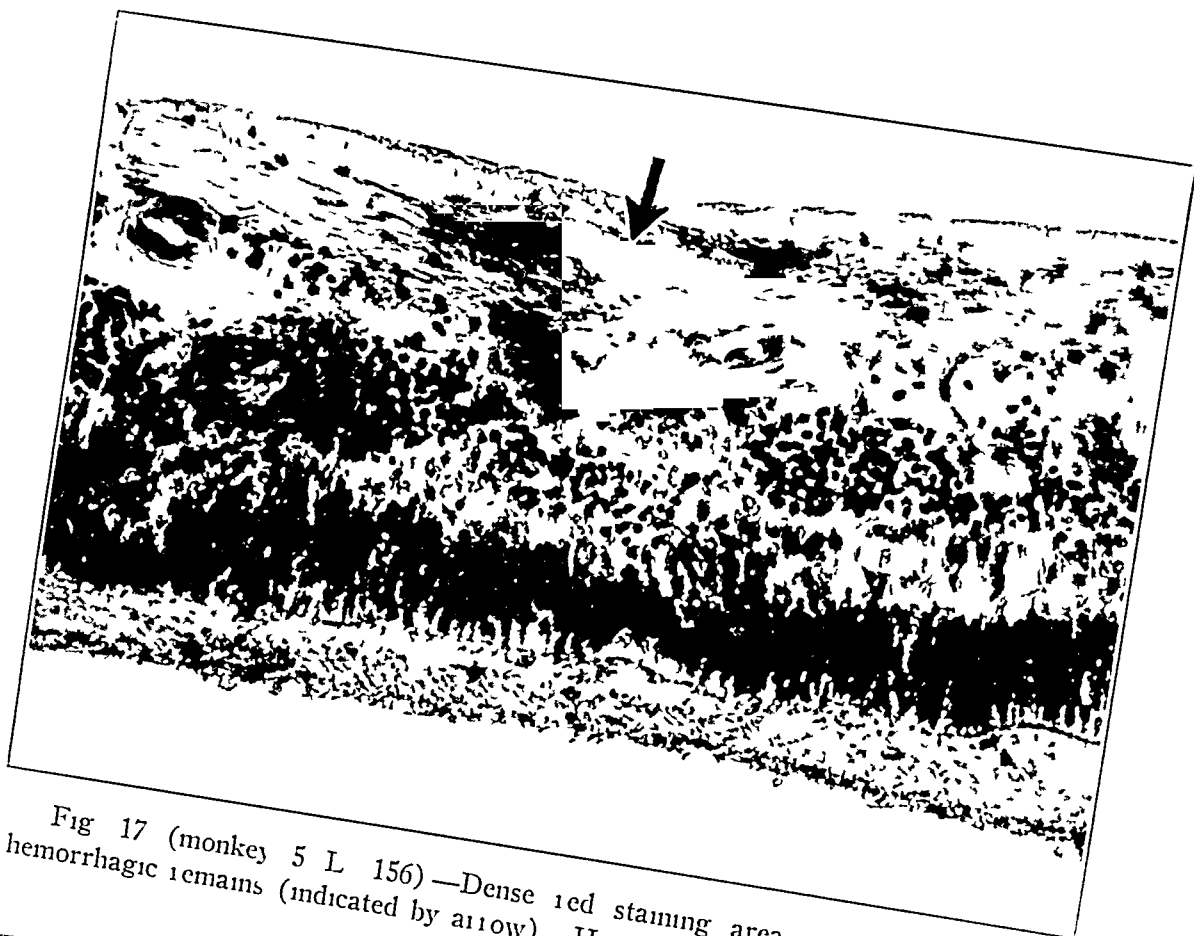


Fig 17 (monkey 5 L 156) —Dense red staining area in retina, probably hemorrhagic remains (indicated by arrow) Hematoxylin and eosin stain, $\times 215$

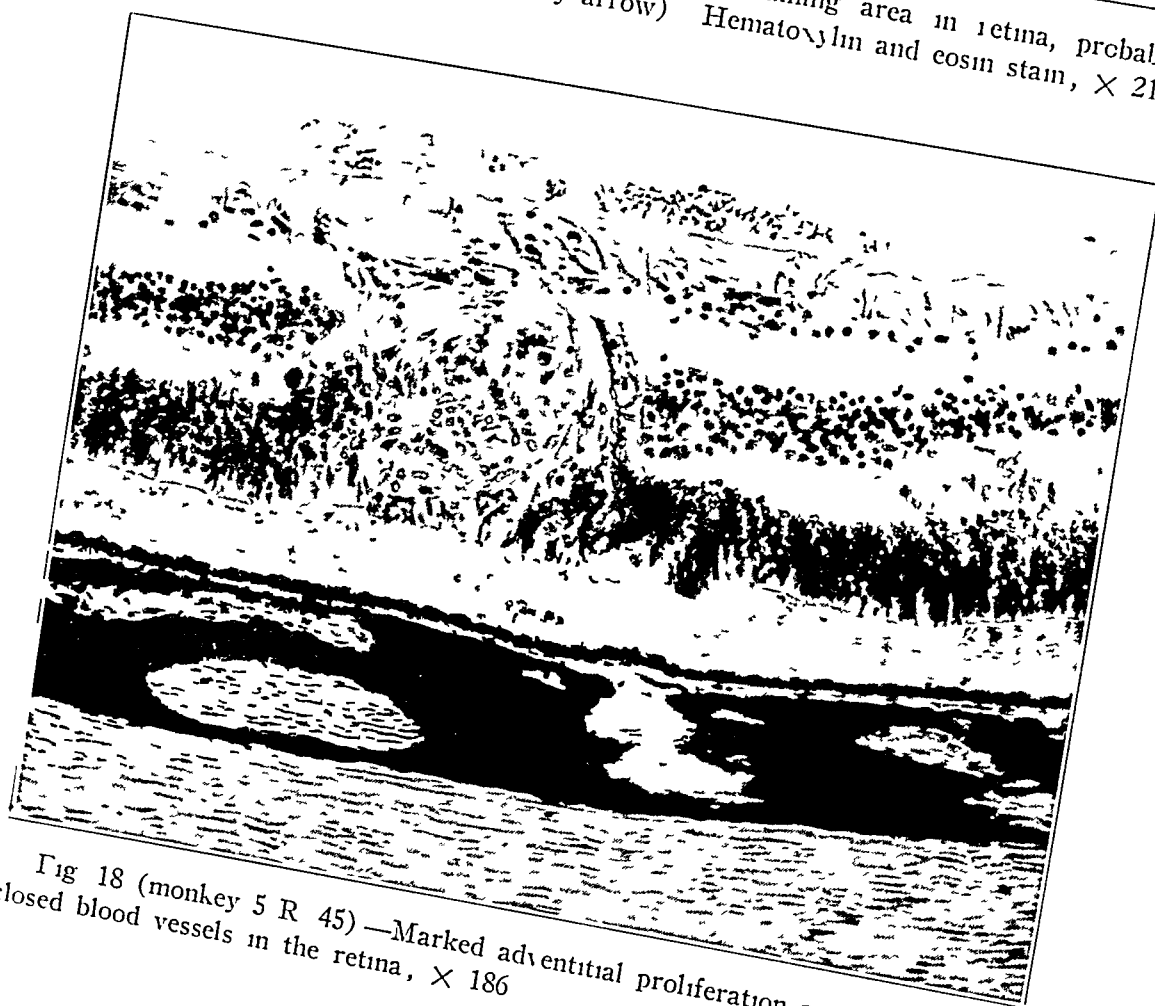


Fig 18 (monkey 5 R 45) —Marked adventitial proliferation around two small closed blood vessels in the retina, $\times 186$

In animals that had a period of benign hypertension before the malignant phase acute degenerative necrotizing lesions were superimposed on chronic arteriolar disease. Arterioles with similar degenerative and necrotic changes were present elsewhere in the bodies of these animals.¹⁸

In dogs with persistent hypertension with moderate or no disturbance of renal excretory function (the group with benign hypertension) no instance of macroscopic or microscopic papilledema has occurred. Localized areas of retinal hemorrhage and edema were present, but only in hypertensive animals with terminal renal damage was widespread



Fig 19 (monkey 5 R 25)—Choroidal arteriole showing a thick wall and hyaline degeneration of the intima. Weigert's elastic tissue stain showed that the elastica of this vessel was destroyed. Hematoxylin and eosin stain, $\times 130$

edema of the retina or choroid present. Retinal scars, in some of which there was iron-containing pigment, were present in the eyes of several animals (figs 11 and 12). Partial atrophy of the choroid was noted. Diseased retinal arteries with thick walls were seen. The thickening of the wall was most frequently caused by a thick media, although in some animals the intima showed definite evidence of thickening and degeneration, particularly hyalinization. The blood vessels in the portion of the retina where perivasculitis had been seen ophthalmoscopically frequently showed a thickening of the adventitia and perivascular cellular elements (figs 13 and 14). Some retinal arterioles showed evidence of disease of all three coats. With special stains for elastic tissue, large

quantities of elastic fibers were shown to be present in many of the small arteries or arterioles (fig 15). The choroidal arteries and arterioles of benign hypertensive dogs, beyond a rather general thickening of the wall of the vessel because of a thick media, did not show evidence of involvement of the adventitia or, except in a few instances, disease of the intima. The posterior and anterior ciliary arteries and the arterial vessels of the ciliary body and iris did not show much evidence of disease, neither was there much, if any, increase in the number of their elastic fibers.

A monkey with benign hypertension had ophthalmoscopic evidence of severe disease of the retinal vascular tree. The retinal arteries were small, and many resembled copper wires or silver threads.

Histologically, the retinal arteries showed a moderate degree of disease mostly confined to the media (fig 16). The choroidal arteries, on the contrary, had widespread and advanced disease of the intima and media (fig 19). The arteriolar disease in this monkey was similar to that found by Moritz and Oldt⁴ in hypertensive persons with arteriolar sclerosis.

COMMENT

Papilledema did not occur in dogs with the benign type of experimental hypertension as long as the excretory function of the kidney remained normal. Decreased renal excretory power usually ushered in ocular changes characterized by edema and detachment of the retina, papilledema and hemorrhages.

The ophthalmoscopic picture was a guide to the nature and extent of the vascular changes in the eyes and bodies of dogs with acute malignant hypertension. Changes seen ophthalmoscopically in dogs and monkeys with benign hypertension were of only relative diagnostic and prognostic value. The degree of apparent involvement of the retinal arteries was not always confirmed histologically. The type and extent of disease in the arterioles of the eye frequently differed from that found in the body. It was impossible during life to evaluate the condition of the uveal blood vessels. The distribution of ocular and systemic arteriolar disease was segmental and comparable to arteriolar sclerosis in man.

SUMMARY

The eyes of dogs and monkeys with persistent hypertension following constriction of the main renal arteries have been observed for more than five years. Changes similar to those seen in man with benign and malignant essential hypertension occurred in the eyes of these animals.

⁴ Moritz, A. R., and Oldt, M. R. Arteriolar Sclerosis in Hypertensive and Non-Hypertensive Individuals, *Am J Path* **13** 679, 1937.

ABSTRACT OF DISCUSSION

DR ARTHUR J BEDELL, Albany, N Y It seems proved that sex heredity, environment and infections all play a part in the terminal composite picture of hypertension in human beings These factors cannot be appraised in animals Then, too, there is the relationship of the control mechanism of the sympathetic nervous system which cannot be considered similar in animals and in human beings This suggests the question of operative intervention for the hypertension, but it is outside of the scope of either the thesis or this discussion

The authors' group of animals with benign hypertension, without a renal lesion, is the largest There seems to be no hard and fast rule which determines whether in a given case hypertension will be of the first type, with early retinal hemorrhage and edema and early moderate and later severe retinal vascular disease, or of the second type, with slowly progressive vascular changes, hemorrhages and small areas of edema

Patients with benign hypertension are observed for decades with little or no change in their condition from year to year, and then suddenly often without any discoverable cause, these patients become worse and die Furthermore, death may supervene when the retinal exudates and hemorrhages have almost disappeared, when they are increasing or when they seem to be stationary Therefore, until the general condition of the patient is known, it is impossible to determine far in advance when any benign hypertension will end As yet animal experiments have not solved this riddle

A number of women with a high diastolic pressure and a systolic pressure of 250 mm of mercury did not show the same fundus picture This is understood by those who know the infinite variety of fundus patterns, and yet it offers an insurmountable hazard against an ex cathedra prognosis An acute toxic fundus in a patient with evidence of general distress exhibits alarming signs such as papilledema, extensive and increasing exudates with hemorrhages These changes are comparable to those found by the authors in their animals with malignant hypertension The practical point is that high blood pressure should not be considered the only essential in prognosis

Persons with malignant hypertension are a detached group Unmistakable signs are seen in some, while in others they are less evident, and the disease is diagnosed correctly only after an observation period of variable time, although those with frank, serious involvement may on their first visit show edema of the retina with fulness of the vessels This type of hypertension is sometimes difficult to diagnose and usually discouraging to treat Even if the patient has high blood pressure and a severe intolerable headache, the changes in the fundus may be few These symptoms in the presence of a typical papilledema have led to intracranial explorations for suspected tumor of the brain, and sometimes the pressure has overshadowed a definite cerebral neoplasm The retinal edema may be general, and exudates and hemorrhages may be absent or present

The malignant hypertension observed by the authors in dogs resembles more nearly the human form, but differs in three ways First, the authors, at least in the abstract which they read, did not refer to the massive exudates which are a conspicuous part of the fundi of many persons with long-standing hypertension These exudates may be about

the macula, the so-called circinate retinitis, they may be large, flat, thick, less definitely outlined areas at the posterior pole, which frequently produce glistening nidescent plaques, linear exudates like spokes from the hub of a wheel, or isolated collections without regular outline. Secondly, the authors say nothing regarding gross vascular accidents, such as embolism, endarteritis, thrombosis or phlebitis, which are not unusual expressions of hypertension. Thirdly, the terminal stage, in which they found the animals' eyes filled with bloody fluid, does not seem to be duplicated in man. The nearest approach to it is in the cases of enormous retinal and vitreous hemorrhages caused by excessively high pressure complicated with diabetes, thrombosis or embolism. Perhaps the duration of the process accounts for these differences. Manifestations of hypertension develop rather rapidly in the laboratory within a few months. This gives little time for the establishment of collateral circulation and the formation of certain exudates.

The authors constricted the renal arteries and noted subsequent changes in the fundus. This is not analogous to any human illness, and their deductions considered from that restricted field are suggestive but not conclusive. The changes were too rapid, not similar to the slow induction of most hypertension, and are frequently more like the acute changes in some toxemias.

They have advanced one more proof of the effect of high blood pressure on the ocular fundus. Their work was carried on in a most scientific manner, their conclusions were justified by their observations, and they merit commendation and deserve praise.

DR WILLIAM L. BENEDICT, Rochester, Minn. It has been definitely established that elevation of systolic and diastolic arterial pressure is associated with the renal ischemia resulting from constriction of the main renal arteries in experimental animals. However, the mechanism of this elevation of blood pressure has not been proved as yet. Probably the most logical theory is that a vasopressor substance of hormonal type, produced or no longer inhibited in the ischemic kidney, initiates and maintains in the peripheral arterioles throughout the body a functional constriction which may be followed later by organic changes in the walls of the vessels. If this is true, it might be assumed that constriction of the retinal arterioles would occur as a part of the generalized vasoconstriction. Page stated that this narrowing of the retinal arterioles does occur in dogs. Apparently, however, the authors have noted this initial constriction only in monkeys. It might be possible to determine whether or not this actually occurs by measurements of the retinal arterioles before and shortly after the development of the hypertension. Apparently, the blood pressure rises rapidly after the production of bilateral renal ischemia and reaches its maximum within a few days. Subsequently it remains at this level or, in a few cases, it may drop somewhat, perhaps owing to the establishment of collateral circulation to the kidneys. If the induced hypertension were directly comparable to acute primary hypertension in man, one might expect the early development of retinitis. This has recently been found to be the case by Dr. Keyes and Dr. Goldblatt in dogs in which there developed extreme hypertension after removal of one kidney and marked restriction of the renal artery in the remaining kidney.

As in all previous experiments whereby hypertension and nephritis are produced in animals, the retinal lesions observed are not identical

with those seen in man. That this may be due to a species rather than to a disease difference is suggested by the difference in the ophthalmoscopic pictures observed by the authors in dogs and in monkeys. In particular, from the histologic standpoint, the cytoid bodies and the serofibinous exudates in the internuclear layer of the retina have not been described.

In general, one forms the impression that the changes which the authors have found in the dogs' eyes and which they characterize as late manifestations of the syndrome are of the inflammatory type. The heavy and progressive periarterial sheathing has no exact counterpart in the hypertensive retinal picture in man, and the hemorrhages into the vitreous and the accompanying scarring and proliferative changes in the retina are not seen in human beings with hypertension uncomplicated by infection or venous thrombotic disease.

It would seem certainly that the lesions observed by the authors are not associated with retained nitrogenous waste products. And further, no toxic product of disturbed renal metabolism has been demonstrated in the blood of these animals. In monkeys, in which the retinal lesions more clearly simulated those seen in man, visible lesions in the arterioles of the retina apparently preceded the development of retinitis. The retinitis healed, while the vascular lesions appeared to progress, as is the case at times in human beings with hypertensive disease. These facts, along with the histologic changes of less marked organic vascular disease than would be expected from the retinal picture, suggest at least that a part of the ophthalmoscopically observed changes in the arterioles were of functional or angiospastic type and that the preliminary functional changes which are generally accepted as the precursors of organic vascular disease in the rest of the body may have been responsible for the transitory retinitis. If so interpreted, the retinal lesions in this case might well be compared to the sequence of events in the hypertensive toxemia of pregnancy.

On histologic examination of the eyes of these experimental animals the authors found changes of varying type and degree in the media and intima of the retinal and choroidal arteries and arterioles. Similar variations are to be seen in the retinal vessels of human beings with hypertensive disease.

It seems justifiable to conclude from Dr. Keyes and Dr. Goldblatt's observations at the very least, irrespective of questions regarding the pathogenesis of the retinitis, that the retinal and choroidal arteries and arterioles are involved in a similar manner and in a comparable degree with those of the rest of the body in a generalized arteriolar disease induced by prolonged vasoconstriction in experimental animals as they have seemed to be in man.

DR. JOHN E. L. KEYES, Cleveland. The retinal arteries of the dog are smaller than the retinal arteries of man. Ophthalmoscopically, it is difficult to see spasm of the retinal arteries of the dog. The ocular fundi and the retinal blood vessels of the monkey (macaque) closely resemble those of man. Spasm of the retinal arteries is more readily recognized in monkeys than in dogs.

I have seen perivascular sheaths in the eyes of persons with essential hypertension. The changes noted clinically and histologically in these experimental animals compare very closely with changes seen in the eyes of persons with benign and acute essential hypertension.

READING DIFFICULTIES IN CHILDREN

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AND

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Educators are today making every possible effort to teach children to read well. Formerly children who failed to learn to read lived out their school lives in classes between the third and the fifth grade and were dismissed to the industrial world at the age of 14. Now every child is required to remain in school in Pennsylvania until he is 18, and he must be placed in classes beyond these low grades in order to complete his social development. The use of general testing for the intelligence quotient of each child weeds out and places in special classes those who cannot learn. There remains, however, a group of children with intelligence quotients of normal or better than normal and with a normal social and vocabulary development who fail to learn to read as well as the average of their class. The ophthalmologist is being asked with increasing frequency to examine these children to see if any visual difficulty is associated with the failure to learn to read.

Another factor of modern educational procedure sends more children than formerly to the ophthalmologist's office. Methods of teaching reading have changed. The newer method, as shall be shown, requires a greater span of visual attention but results in ability to read rapidly. Beyond the third grade the amount of supplementary reading required is based on this ability to read rapidly, and the child who reads slowly, even though accurately, tends to fall behind his grade. Symptoms of eyestrain are a frequent concomitant of the effort of such children to keep up with the required reading, and they also are brought to the ophthalmologist for consultation.

A review of the methods of teaching reading is necessary if the ophthalmologist who studied in the first grade from twenty to forty years ago is to understand the problems of the present day child. Some will remember being taught the letters of the alphabet by their sounds and then being taught how to combine the individual letters into words. Later it became evident that a child learned to read more quickly if he read whole words already in his vocabulary. After learning these words, he learned the family of words and derived the essential phonic sounds which he might use in analyzing new vocabulary. Still later, the word

method was expanded into a phrase method, but in most classes of today it has been further expanded into a sentence method. The child immediately on entering school learns to read whole sentences which express facts and actions familiar to his experience. The analysis of the sentence into individual words and the analysis of the individual word into phonic sounds are taught as a secondary matter when the child is already reading an extensive vocabulary based on visual memory rather than on synthesis of sounds.

It has long been a custom to consider a child ready to learn to read when he is 6 years old. Today many educators are making studies of so-called "reading readiness" and are inclined to believe that certain children are not prepared to read although they have reached their sixth birthday. This conclusion may be fallacious in that it is based on the fact that certain children with apparently normal equipment fail to learn to read at this age. The final conclusion on the subject of "reading readiness" must await more research, in the meantime, the vast majority of the children will be required to try to learn to read when they are 6.

It is at once apparent that a child with gross visual defects will probably fail to learn to read. However, in many schools there is no ocular examination for visual acuity until the third grade or beyond, so the child who fails in reading because of gross visual errors is not detected unless his parents take him to an ophthalmologist. Ophthalmologists who contact public school systems should urge immediate examination for all children entering school by means of illiterate charts to detect and correct major visual errors. At the end of the first year all children who are unable to read should be given a thorough examination, including refraction under atropine.

In this paper we shall not discuss further the question of visual acuity but will concern ourselves with the less obvious problems of the children who cannot read. The modern sentence method of teaching reading, while it produces rapid and intelligent readers, tests to the limit the child's powers of attention and concentration. Many of the problems of the child who is unable to read are matters more for the teacher and the psychologist than for the physician, but the doctor is directly interested in the various factors affecting visual attention. Because of the high degree of visual attention required, certain minor defects that were of less importance under older methods of teaching have now become significant.

VISUAL IMMATURITY

The first of these factors is immaturity of the child's visual apparatus. This is expressed in one way by poor resolution. If all children entering the first grade were tested for visual acuity, it would be found that the majority of them do not have full 6/6 vision. A second test

at the end of the year, or at the beginning of the second year, would show that most of them have normal vision. This is simply due to the fact that the child's resolving power has not yet been fully developed because of lack of visual experience. He may have a perfectly sharp retinal image but has never been called on to resolve and interpret that image into a symbol with meaning. The condition is of no importance and may be regarded as normal. All teaching, however, should be done by means of large charts and blackboard demonstration rather than by the use of printed books, so that the power of resolution may develop before it is necessary for the child to resolve small print.

A second evidence of immaturity is expressed in the reversal phenomenon. This may be apparent as reversal of words, letters or entire sentences. The reversal of letters is common to almost all children of from 4 to 6 years, and any one who tests visual acuity of children has noticed that they frequently reverse the position of two letters or numbers in the test line. Reversal of short words, such as reading "saw" for "was," is fairly common during the first few months of reading. A variation of the same condition is the perception of the end syllable for the first part of the word. In reading the word "running" the child may pick up the last syllable, substitute a word already in his vocabulary, and read "walking." The highest degree of reversal is reached in mirror writers, who reverse entire sentences and write them backward.

The cause of reversal may be simple immaturity or some confusion in the eye association pathways. This confusion may be due to poor interpretation of images as a result of confusion in eye-hand association. This last condition may be found in a left-handed child who is being taught to use his right hand or in a right-handed child from a family with a strain of left-handedness or in an ambidextrous child. A child sensitive to low hypermetropia will frequently show reversals.

The treatment of reversal is more a problem for the teacher and the psychologist than for the ophthalmologist, but the oculist should recognize the condition and oversee the training, as the child may be caused considerable difficulty if he is not handled properly. Reversal due to simple immaturity will disappear spontaneously as the child develops, and the only treatment necessary is the planning of the reading program in such a way that the child is given as few reversible words as possible. The child with a crossed eye-hand dominance requires more careful attention. This is particularly true of the right-handed child with a family history of left-handedness, as the hereditary situation may be overlooked. Treatment consists in determining which is the dominant eye and in having the child use the hand on the same side. Proper eye-hand coordination can be developed by tracing, drawing or mechan-

ical activities. An excellent exercise consists of tracing in a stereoscope, as in the training of amblyopia, with the pencil in the hand corresponding to the dominant eye. In the case of mirror writing it must be impressed on parents and teacher that the condition should not be drawn to the child's attention. If the child becomes aware that he is an object of interest, he will continue mirror writing deliberately and indefinitely. When a sentence is written backward the child should trace the corrected sentence and then write it properly free-hand.

The entire treatment of reversal is concerned with the clearing of the eye-hand association pathways to avoid confusion. Occasionally a situation arises in which it is necessary because of the demand on the part of the parents, the teacher or the child himself to train a left-handed child to use the right hand. If the left eye is strongly dominant, this will result in poor muscular coordination, poor reading and possibly speech defects. The best way to handle such a situation is to occlude the left eye and train the right hand and the right eye to work together in an attempt to change the ocular dominance to the right eye. Parsons stated that once ocular dominance is established, it cannot be changed by training. The age at which it is acquired is not exactly known, but it probably begins at about 3 years and is firmly fixed at the age of puberty. Even if ocular dominance is not changed the right eye-right hand coordination in left-handed children can be improved with consequent improvement in reading and writing.

By no means all left-handed children show reading or speech difficulties if they are taught to use the right hand, but many of them do have these difficulties, and the possibility must always be kept in mind in the case of such children. An excellent review of the subject of ocular dominance has recently been published by Fink.¹

LOW HYPERMETROPIA

Another cause of poor reading ability in children in the first grade is low hypermetropia. This is somewhat difficult to explain, but probably is another evidence of immaturity. A child of 6 has an accommodative power of 15 diopters, and simple hypermetropia up to 1 diopter may be regarded as normal at this age. With this power of accommodation, it does not seem possible that simple hypermetropia between 1 and 2 diopters could cause any difficulty, but the clinical fact remains that we have seen many children progress from near the bottom of their class in reading to near the top simply by the correction of hypermetropia of between 1 and 2 diopters. Since this seemed so illogical, we checked carefully with the teachers and parents to make sure there had been no

¹ Fink, W. A. The Dominant Eye. Its Clinical Significance, *Arch Ophth* 19 555 (April) 1938.

change in teaching methods or special tutoring. We do not believe that the improvement in reading was due to the psychologic effect of wearing glasses, as several of the children objected strenuously to them.

The great majority of children show no signs or symptoms from low simple hypermetropia, and our only explanation of the fact that a few of them do is based on the theory that these children have not learned to use their accommodation in a perfectly automatic, subconscious manner. Apparently some few children with low refractive errors find it necessary to pay a certain amount of attention to the mechanics of accommodation, which in other children more visually mature is perfectly automatic. This calling of attention to the act of accommodation and the necessity for concentration on the retinal image in order to keep it clear distract the child's attention from the sentence which he is supposed to be reading. We have already stated that the sentence method of teaching requires all the visual attention of which a 6 year old child is capable. By this distracting of attention the act of accommodation probably is responsible for inability to learn to read by the sentence method. We find that by the time the child has reached the third or fourth grade, after correction in early reading, he is able to read with ease and comfort without glasses even though the hypermetropia remains unchanged. This leads us to believe that in this particular type of child we are dealing with another type of visual immaturity. The child has a superabundance of accommodative power, but because of lack of practice has not learned to use it with perfect ease and lack of conscious effort. As he progresses in age and visual experience, the function of accommodation becomes automatic and requires no attention on his part. This is one of the conditions which has become important because of modern methods of teaching reading. Under the word or phonic systems, low hyperopic errors were of no significance because the child could pay attention to the act of accommodation and still have sufficient span of visual attention to read single letters or words. Many such children never learned to read rapidly, although "sentence reading" was introduced in the upper grades. The methods now used in the first grade produce more excellent readers and fewer "word" readers, but it is evident that more children will need visual aids.

DEFICIENT FUSION

All the foregoing factors in deficient reading ability may be regarded as various types of visual immaturity. Another cause of poor reading is deficient binocular fusion. In the examination of several hundred children in the first grade we found no significant fusion difficulties except in those children who had a squint, those who had had a squint and those whose vision was markedly better in one eye than in the

other. The children with squint or with squint which is partly corrected may be divided into four groups: those with uncorrected monocular squint, those with partly corrected monocular squint, those with uncorrected alternating squint and those with partly corrected alternating squint. The phrase "partly corrected" may be interpreted to indicate any stage in the recovery of the squint after refraction, operation or orthoptic training in which full binocular vision with complete ranges of fusion has not yet been established.

Uncorrected monocular squint will cause no reading difficulties, as the child's suppression is complete and there is no binocular confusion. Orthoptic training may be continued during the school term. Occlusion for amblyopia is best done outside the school, and the most satisfactory training consists of drawing and tracing rather than reading of print so that confusion in reading as amblyopia improves may be avoided as far as possible.

Partly corrected monocular squint is a frequent source of reading difficulty. This situation may be expected in a child whose squint has been cosmetically cured by refraction or by operation without adequate training in fusion. The remaining suppression or alternation produces binocular confusion, which makes it impossible for the child to read by the sentence method. It must be remembered that during the course of orthoptic training monocular squint passes through a period of alternating squint, which is confusing to a child if he is learning to read during this period of his training.

In such a situation the best course is to have the child occlude one eye while he is in school to avoid the binocular confusion which results from the rapid alternation. Occlusion is applied to the right and the left eye on alternate days. Orthoptic training should be pushed as rapidly as possible.

There is another factor in cases of cosmetically corrected squint which causes reading difficulty and which is frequently overlooked. This is poor fusional convergence. It seems paradoxical that a child who has had convergent strabismus is unable to converge but the situation is explained by the fact that a child with squint does not use true binocular or fusional convergence. This will develop spontaneously in many cases after correction of the squint by refraction, orthoptics or operations. But in many more cases the fusional convergence remains weak and causes excessive tiring of the eyes or confusion, perhaps even alternation developing at the near point. This should be kept in mind in the treatment of all children with convergent squint, and the training should be directed not only at the turning of the eyes but at the development of proper fusional convergence.

The treatment is, of course, orthoptic training. During the school year the child can be made much more comfortable either by occluding

one eye to avoid the necessity for binocular vision at the near point or by the use of base-in prisms to give binocular vision at the reading distance without the necessity for convergence. Usually orthoptic training over one summer's vacation will develop fusional convergence to nearly normal.

In cases of alternating squint we believe that it is best not to start training until the child has learned to read. The period of training is long and tedious and always produces visual confusion, which makes it difficult for the child to learn to read during the training period. A careful history should be taken in all cases of apparently alternating squint to make sure that the condition is not an original monocular squint which is in the stage of alternating in the process of spontaneous recovery.

If this is true, or if it seems advisable to correct the squint while the child is in the process of learning to read, the condition should be handled as a monocular squint in the stage of alternating. Careful testing should be done for alternation, diplopia and blurring of the print at the near point. Patients with alternating squint, particularly of the divergent type, will frequently show poor binocular vision when they have first learned to hold their eyes straight. In several of our cases monocular vision was 6/6 in each eye, and binocular vision, a blurred 6/60. This naturally tends to discourage the child in his efforts at binocular vision, and the condition must be looked for and trained. Training is best done with a reduced Snellen chart in the stereoscope, and the patient should be encouraged constantly to use both eyes together, even if vision is blurred, and to make constant attempts to get the images clear without allowing one eye to drift out or in.

These are the factors which we have found to be detrimental to children in the first and second grades who are just learning to read. Occasionally a child will learn to read well and rapidly by the sentence method, but somewhere between the third and the sixth grade his reading ability will decrease, and he will drop back to reading by the word method. In these cases the cause is almost always fusional convergence deficiency.

DEFICIENT FUSIONAL CONVERGENCE

The first and most common result of fusional convergence deficiency is occasional monocular suppression. The left eye is the one usually suppressed in a right-handed child. Reading becomes slow and difficult because the dominant right eye reads the words first, and the left eye, usually suppressed, will have occasional flashes of vision, which cause confusion. This is particularly true when the child is reading long words or sentences. The concentration necessary to interpret the word stimu-

lates the vision of the left eye, binocular vision is momentarily restored and the print changes in appearance or position. This necessitates rereading of the word or sentence. Children tell us that long words will frequently jump or the print become confused, and it is difficult for them to pick up the beginning of each new line of print.

The condition can be diagnosed easily and is frequently discovered by seeing one eye waver slightly during the test for the near point of convergence or by having the red line of the Maddox rod disappear occasionally during the phoria test. On the stereoscope it is easy to find by the use of the small Keystone dots, the Wells F-L or some similar test object. Watching the child read when a tiny light is reflected in the pupils gives a clear indication of frequent regressions. The ophthalmograph, which photographs the actual movements of the eyes during reading, gives an accurate record of such movements but will not indicate momentary suppression during which coordination of the eyes is not lost.

The second difficulty caused by poor fusional convergence is constant alternating at the near point. This alternating is frequently present without any measurable phoria. The eyes remain perfectly straight, but at the near point vision alternates rapidly between the two eyes. Sometimes the fusion at distance may be perfect, but usually there is occasional alternation. The condition is easy to diagnose with the stereoscope if small test objects are used at the near point.

Another type of suppression which makes a child unable to read rapidly is suppression of the periphery at the reading distance. This can be found by using the small three dot Keystone fusion test cards or the Wells ONE at the near point. As the child looks at the three dots, the center will remain fused, but the upper and lower dots will disappear suddenly and simultaneously, to reappear and disappear at intervals of a few seconds. Occasionally, with concentration on the middle dot, the upper and lower dots will disappear permanently until concentration is relaxed. Sometimes a child will volunteer the information that when he concentrates on a new or difficult word the rest of the page becomes blank or blurred. With so small a field of vision, naturally it is impossible for a child to read rapidly by scanning a sentence. He must pay close attention to each particular word or syllable.

The cause of these types of reading difficulty seems to be an improper distribution of binocular attention. It probably starts in the first or second grade as a result of refractive error, poor convergence, weak fusion or a strongly dominant eye. These factors result in an over-concentration on macular vision for the purpose of mastering print, and the attention is improperly distributed between the maculas of the two eyes or between the macula and the periphery. It is interesting to

speculate on the role of the bilateral cortical representation of the macula in these types of fusion deficiency

It is important to point out that in testing for these fusion deficiencies the work must be done at the reading distance with small test objects, as convergence and accommodation enter into the causes, and if the testing is done at distance or with large targets the conditions will be completely overlooked

METHOD OF CORRECTION

The first step in the correction of the difficulties of older children is careful refraction under atropine. The same low errors which cause some children in the first grade to fail to learn to read will cause older children to become poor readers when the visual demands of increased study tire or strain the eyes. The most frequent condition found in the children I have seen is low astigmatism of one eye with no astigmatism in the other or low astigmatism of both eyes at oblique axes.

The second step in remedial training is to restore normal binocular habits. This can be done most efficiently by orthoptic training. Special emphasis should be placed on near point fusional convergence, elimination of all suppression and maintenance of clear images for print. The Snellen chart reduced for the stereoscope, the Wells G series of printed cards and the Wells system of bar reading are excellent aids. The span of attention should be gradually increased by the use of more complex targets and larger lines of print and special attention should be given to complete binocular attention during coordinate movements to the left and right.

The remaining training is in the actual process of reading and should be in the hands of a skilled teacher. Children may be divided into two main groups: children of from 6 to 8 who are failing to learn to read and children from 10 to 18 who read too slowly or with too little comprehension for their required school work.

There is a wealth of available literature on the technics of remedial training in reading, and it is not the purpose of this paper to discuss such technics except from the point of view of establishing normal ocular habits.² If the child has not learned to read and ocular difficulties are present, it is wise to repeat the steps used in the early teaching of reading. He should read first from charts placed from 6 to 8 feet (183 to 244 cm) in front of the eyes (the material in such charts being adapted to his age and experience) and should advance from charts to primer size

² Betts, E. A. *The Prevention and Correction of Reading Difficulties*, Evanston, Ill., Row, Peterson, & Company, 1936. This book should be read by every one interested in reading difficulties. It contains an excellent bibliography.

print for his first efforts at book reading. Gradual reduction in the size of print should be made only when good reading habits are established for the larger types. Whether to reinforce visual images by kinesthetic images through tracing the sentences to be read is a decision which rests with the teacher. We have found it an excellent aid in cases of frequent reversals and minor reading or writing.

In the older group of children the correction of ocular difficulties frequently leads to immediate comfort and swift reading. If habits of word reading are too firmly established, remedial training in sentence reading is necessary. In either case the remedial steps should be followed by an assignment of much easy and interesting supplementary reading. This serves the twofold purpose of establishing the corrected ocular habits permanently and of building up a reading vocabulary made deficient by the years of slow reading.

The ophthalmologist in his supervision of a child with a reading problem, should not lose sight of the psychologic factors which quickly enter the picture when a child fails in normal scholastic achievement. Emotional "sets" against teachers and parents, the process of reading itself and school in general are probable complications. Children who fail to read when the ocular difficulties have been corrected and there are no language or hearing complications should be referred to a psychiatrist for analysis and solution of emotional difficulties which are probably barring his progress.

Ophthalmologic Review

CARBON DISULFIDE POISONING

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PHILADELPHIA

One of the results of the increasing application of chemicals in industry is a widening of the field of industrial risks. Although improved methods of manufacture have eliminated many of the occupational hazards previously encountered, new ones have appeared, and some previously considered eliminated have reappeared. Such is the case with regard to carbon disulfide.

Sixty years ago the literature contained many reports of carbon disulfide poisoning occurring among those who worked with rubber. Articles to be vulcanized were passed through a solution of sulfur chloride and carbon disulfide. Changes in the method of manufacture and adequate protection of the worker have practically eliminated this source of intoxication from the rubber industry.

Today carbon disulfide is being used extensively in this country in the rayon and the transparent paper industry and to a much lesser extent in the manufacture of dipped rubber goods and water-proof cements and in certain extractive and pharmaceutical processes. Considering the tremendous increase in the production of rayon and transparent paper within the past few years, it is evident that a new source of intoxication has appeared. Carbon disulfide is absorbed chiefly through the respiratory system, though the alimentary tract and the skin are capable of some absorption. When applied to the skin in the liquid state, it causes anesthesia and then a first or second degree burn. Delpach,¹ in 1856, was the first to recognize that the signs and symptoms of carbon disulfide poisoning present a definite clinical entity. His reports are still classic, and little has been added to the knowledge of this disease since publication of his original papers. Many reports have subsequently come out in the foreign literature since then, and one report, by Gordy and Trumper,² was recently published in this country.

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1 Delpach, A. Accidents que développe chez les ouvriers en caoutchouc l'inhalation du sulfure de carbone en vapeur, *Union méd* **10**.265, 1856

2 Gordy, S. T., and Trumper, M. Carbon Disulphide Poisoning, with a Report of Six Cases, *J. A. M. A* **110** 1543 (May 7) 1938

However only one report has previously appeared in the American ophthalmologic literature that of Heath,³ in 1902. The frequent mention of ocular complications in all previous reports and the opportunity to examine 120 workers who had been exposed to carbon disulfide over a number of years prompted the study reported here.

In cases of severe carbon disulfide poisoning, failing vision has been reported as one of the earliest and most common symptoms. Delpech reported 15 cases of amblyopia in his series of 34 patients, Desmarres, an ophthalmologist who examined some of his patients, stated that there were no visible changes in the fundus to explain the marked reduction in vision. Shortly after this it was determined that the amblyopia was due to a central scotoma. The cases became so common that in 1885 the Ophthalmological Society of the United Kingdom appointed Frost, Gunn and Nettleship⁴ to report on the ocular complications of carbon disulfide poisoning.

The scotoma is reported to be small, bilateral and central, though it frequently extends slightly to the temporal side of fixation. It varies but slightly in intensity and is practically always absolute. Terrien⁵ stated that the scotoma may be large (about 30 degrees) and that it may be unocular, but few have agreed with him. Uhthoff,⁶ in his classification of toxic amblyopias, placed carbon disulfide in that group of exogenous toxins frequently associated with peripheral neuritis. He stated that besides the central defects there was at times a slight peripheral depression of the field of vision. This is substantiated in the literature and in the observations made by my associates and me. Little⁷ reported a case in which there was only peripheral contraction of the field and no central scotoma, which was considered unusual. The conductivity of many of the nerve elements apparently is frequently impaired, as in many of the patients complete red-green blindness was noted.

Chromatopsia and photophobia have been noted by some patients before the marked reduction in vision. The objects appeared greenish blue or red. Just what significance this may have is hard to say, Terrien claimed that it was due to the toxic effect of carbon disulfide on

3 Heath, F. C. Amblyopia from Carbon Bisulphide Poisoning, *Ann Ophth* **11** 4, 1902

4 Frost, W. A., Gunn, R. M., and Nettleship, E. Report of the Committee on Poisoning by Bisulphide of Carbon and Chloride of Sulphur, *Tr Ophth Soc U Kingdom* **5** 157, 1885

5 Terrien, F. Deux cas d'amblyopie par le sulfure de carbone, *Paris med* **10** 317, 1920

6 Uhthoff, W., in von Graefe, A., and Saemisch, E. T. *Handbuch der gesamten Augenheilkunde*, Leipzig, Wilhelm Engelmann, 1911, vol. 11, pt. 2 a, p. 45

7 Little, D., in Discussion of Toxic Amblyopia, *Lancet* **2** 18, 1887

the visual purple That might also explain the frequently reported symptom of night blindness

The other ocular manifestations are likewise part of a systemic intoxication which is most evident in the central and in the peripheral nervous system Galezowski⁸ was the first to report paralysis and weakness of accommodation in these cases This disorder has been observed several times since, though the vision was usually so markedly reduced that measurement of accommodation was impossible

Pupillary disturbances were not constantly observed in the reported cases The pupils were frequently dilated and reacted poorly to light Inequality of the pupils also has been mentioned by Koelsch⁹ and others These phenomena were not always associated with amblyopia and have been known to persist after improvement in the general condition of the patient

Only 1 case in which nystagmus occurred has been reported, that of Berbes¹⁰ Laudenheimer,¹¹ Koelsch and others mentioned that nystagmus may be present but reported no personal observations The extraocular muscles were rarely affected, and no cases of diplopia have been reported Bergeron and Lévy¹² were the first to draw attention to anesthesia of the cornea in these patients They confirmed their observations on animals and noted that the corneal anesthesia appeared before that of the skin and lasted longer They considered that it was due to the systemic effect of carbon disulfide and not necessarily to the local action of the gas on the cornea This, moreover, was not a frequent finding in the reported cases

Carbon disulfide may often be the cause of severe psychogenic disturbances, many cases have been reported Of chief interest to the ophthalmologist are the patients reported by Charcot¹³ and Marie¹⁴ Their patients had hysterical hemianesthesia, with tubular fields,

8 Galezowski Troubles visuels consecutifs à l'intoxication par le sulfure de carbone, *Rec d'opht* 4.121, 1877

9 Koelsch, F Handbuch der Berufskrankheiten, Jena, Gustav Fischer, 1935, vol 1, p 553

10 Berbes, P Observation de pseudo-tabes dû a l'intoxication par le sulfure de carbone, *France med* 1.3, 1885

11 Laudenheimer, R Die Schwefelkohlenstoffvergiftung der Gummi-Arbeiter unter besonderer Berücksichtigung der psychischen und nervösen Störungen und der Gewerbe-Hygiene, Leipzig, Veit & Co, 1899

12 Bergeron, G, and Levy, P Note sur l'anesthésie de la corneé dans l'empoisonnement par le sulfure de carbone, *Gaz méd, Paris* 19 584, 1864, *Compt rend Soc de biol* 1 49, 1864

13 Charcot, J M Leçons du mardi à la Salpêtrière, Paris, E Lecrosnier & Babe, 1889

14 Marie, P Sulfure de carbone et hystérie, *Bull et mém Soc med d hop de Paris* 5 445, 1888

micropsia, maciopsia and monocular diplopia on the same side as the anesthesia

The ophthalmoscopic examination seldom revealed any changes in the fundus. There was occasionally slight hyperemia of the disk or temporal pallor. Galezowski, Baader¹⁵ and Uthoff are the only ones to report atrophy of the optic nerve following severe intoxication. Nuel¹⁶ and Hirschberg¹⁷ have reported slight stippling and pigmentation of the macula lutea.

Another ocular condition has been frequently noted among the spinning room workers in the rayon industry, though it is not definitely known how far carbon disulfide is responsible for its occurrence. The symptoms are usually photophobia, halos about lights, a feeling of sand in the eyes and finally pain and profuse lacrimation. Strebel¹⁸ called the condition keratitis punctata superficialis traumatica. He attributed it to hydrogen sulfide, which is present in the spinning bath. Klein,¹⁹ who examined some 600 patients, described the corneal condition as follows. First there is slight graying of the cornea, small punctate elevations then appear subepithelially and finally break down, and the cornea stains lightly. The lesions heal rapidly and do not leave any permanent opacity. The workers as a rule do not notice these symptoms until they have worked for two or three eight-hour shifts. To explain this latent period, Duke-Elder²⁰ advanced the theory that gases from the spinning solution sensitize the corneal epithelium to photochemical changes which would not affect the normal cornea. Most writers have expressed the opinion that the lesion is caused chiefly by hydrogen sulfide and that carbon disulfide probably plays a minor role.

MATERIAL AND METHODS

My personal observations concern the examination of 120 workers employed in a rayon industry in which the viscose process of manufacture was used. Most of them were working at the time. The investigation was part of a general medical examination, the complete results of the survey will be published in monograph form at a later date. The ocular tests included determination of

15 Baader, E. W. An Hirntumor erinnernde Vergiftungserscheinungen durch Schwefelkohlenstoff, *Med. Klin.* **28** 1740, 1932.

16 Nuel, J. P. Alteration de la macula lutea, *Arch. d'opht.* **16** 473, 1896.

17 Hirschberg, J. Schwefelkohlenstoffvergiftung, *Centralbl. f. prakt. Augenheilk.* **13** 268, 1889.

18 Strebel, J. Durch SO₂ verursachte Augenschädigungen (spez. zentrale punktförmige Viskoseverätzung der Hornhaute). Schutz durch Maskenbrille mit Zinkkohlefilter, *Schweiz. med. Wchnschr.* **53** 560, 1923.

19 Klein, E. Les lésions oculaires dans les fabriques de soie artificielle, *Arch. d'opht.* **45** 686, 1928.

20 Duke-Elder, W. S. *Text-Book of Ophthalmology*, St. Louis, C. V. Mosby Company, 1937.

corrected vision with pinhole, peripheral fields on a perimeter and central fields on a campimeter, stereocampimeter and Hartz charts, external examination, measurement of the near points of accommodation and convergence, and an ophthalmoscopic examination. The corneal sensitivity and pupillary reactions were checked by another observer (Dr F H Lewy) with Fiey's hairs and a Zeiss pupilloscope.

RESULTS

Approximately 75 per cent of those examined had some ocular complaints. They varied from the usual complaints that might be attributed to refractive errors to the definite symptoms of rayon keratitis. Two workers gave a history which suggested that transient amblyopia had occurred several years previously, but they had no evidence of it during the present examination. More than half of those examined had been employed for ten years or longer.

Ocular Defect	Location of Workers							
	Churn Room		Spinning Room		Elsewhere		Total	
	24		84		12		120	
	No	%	No	%	No	%	No	%
Diminished corneal reflex	8	33.5	54	64.0	5	41.8	67	55.0
Pupillary disturbance	15	62.5	32	47.5	1	8.3	48	40.0
Enlargement of blindspot	9	37.5	15	18.0	3	25.0	27	22.5
Field defects	4	16.5	3	3.6	1	8.3	8	6.7
Changes in disk	6	25.0	10	12.0	3	25.0	19	15.9
Vascular changes	7	29.0	24	28.5	3	25.0	34	28.4
Nystagmus	15	62.5	42	50.0	4	30.0	61	50.8

The findings are tabulated for the sake of comparison, though I realize that errors may be introduced by attempting to analyze results which involve such small figures. The churn room was considered to have the highest concentration of carbon disulfide. Those listed as working elsewhere were working in parts of the plant other than the churn room or the spinning room.

Diminution of the corneal reflex was found in over half of the workers examined, it was most common in the spinning room workers, despite the fact that all other signs were found more commonly in churn room workers. This would imply that diminution of the corneal reflex may be due in part to the same factors that produce the keratitis. Diminution of the pupillary reaction to light was the second most common finding, it was more frequently observed among the churn room workers.

Enlargement of the blindspot was not noted in any of the previously reported cases but was found in 22.5 per cent of the workers I examined. The enlargement varied from two to three times the normal size. In

a small number of cases there was a definite contraction of the peripheral field for a 1 mm white test object

Rotatory nystagmus was present in about half of the workers, occasionally it was evident in the primary position, but it was usually elicited only on horizontal or upward gaze

Vascular changes, which varied from early arteriolar sclerosis to marked arteriosclerosis, were found in slightly less than one third of the cases. The changes in the disk were not marked, they varied from slight blurring to a questionable pallor of the papillomacular bundle. None of those examined showed definite neuritis or atrophy.

No workers with symptoms of rayon keratitis were seen during the course of the examination, though many of them gave a typical history of keratitis and several had been transferred to other parts of the plant because of their susceptibility to soreness of the eyes. Several of them were examined with a slit lamp, but no corneal lesions were seen, nor was there any scarring from previous attacks of keratitis.

COMMENT

Carbon disulfide is known to be an exogenous toxin producing changes in the central and peripheral nervous systems. Ranelletti²¹ reported that in 80 per cent of the cases there is some evidence of neurologic disturbances. Koester²² has shown by experimental work that the toxin produces changes in the ganglion cells of the brain and in the myelin sheaths of the peripheral nerves. Birch-Hirschfeld²³ was unable to demonstrate any changes in the ganglion cells of the retina, while Offret²⁴ found degenerative changes in the optic nerve.

The ocular signs of carbon disulfide poisoning are essentially those of a systemic neurologic disturbance. Though amblyopia was the symptom most frequently noted in the previously reported cases, it was not found in any of the workers in the series reported here. One patient had poor central color vision, but no scotoma was demonstrable. In all the previously reported cases the poisoning occurred in an industry in which the exposure was much greater than in the rayon industry, and the symptoms frequently developed after a short exposure. Enlargement of the blindspot has not been previously noted in the literature, this I believe to be of definite significance. It was present in 10 of the

21 Ranelletti, A. Die berufliche Schwefelkohlenstoffvergiftung in Italien, *Arch f Gewerbepath u Gewerbehyg* **2** 664, 1931

22 Koester, G. Ein klinischer Beitrag zur Lehre von der chronischen Schwefelkohlenstoffvergiftung, *Deutsche Ztschr f Nervenhe* **26** 1, 1904

23 Birch-Hirschfeld, A. Beitrag zur Kenntnis der Netzhautganglienzellen, *Arch f Ophth* **50** 230, 1900

24 Offret, A. Essai sur l'amblyopie par le sulfure de carbone, Thesis, Paris, no 455, 1906

patients who were considered to have slight blurring of the disk margins. Depression of the peripheral field, as mentioned previously, is not an unusual finding when toxic amblyopia is associated with peripheral neuritis, as is the case with carbon disulfide poisoning.

The diminution of the light reflex, while only occasionally mentioned in all previous reports, occurred in nearly half of my cases. Two of the workers showed definite recession of the near point that was not due to presbyopia or to a refractive error.

Diminution of the corneal reflex was the most constant finding, as previously noted, it is probably due in part to the direct action of the gases and the spray from the spinning solution. In the reports on keratitis no mention was made of the corneal sensitivity, but there is probably some relation between the keratitis and the diminution of the corneal reflex.

The nystagmus I believe to be vestibular in origin. It is surprising that this has been reported only once before, while I was able to demonstrate it in over 50 per cent of the cases.

SUMMARY

I have reviewed briefly the ocular signs and symptoms of carbon disulfide poisoning and have reported on the examination of 120 workers who had been exposed to carbon disulfide for a long time. Many of them showed systemic signs of chronic intoxication. Enlargement of the blindspot I believe to be the most significant early ocular sign of chronic carbon disulfide intoxication.

A complete bibliography and correlation of these data with those of the other examiners will appear in monograph form at a later date.

Clinical Notes

HOMONYMOUS HEMIANOPIC PARACENTRAL SCOTOMA

Report of a Case

T D ALLEN M D, AND H F CARMAN JR, M D, CHICAGO

The purpose of this report is twofold first, to contribute to the literature one more case of hemianopic paracentral scotoma and, second, to corroborate the reports of previous authors

In reviewing the literature we found that prior to 1930 only a few cases of this type of defect of the visual field had been reported In 1907 Wilbrand¹ reported 8 cases and described them in detail In 1908 Posey² reported 1 case Stieren³ in 1924 found a hemianopic paracentral scotoma in a patient with an acute facial abscess in whom meningitis later developed, the scotoma appearing after development of the meningitis Newton⁴ described 1 case in 1936 O and H Barkan⁵ reported 2 cases in 1930, and in 1935 O Barkan and Boyle⁶ presented another case In the last account the authors summarized the clinical findings in cases in which this type of visual field defect is present Invariably the following symptom complex is found (1) difficulty in reading and fixation of objects located close to the central field on the side of the defect, (2) absence of reduced visual acuity, (3) characteristic changes in the visual field, namely, a homonymous scotoma that does not include the macula, with maintenance of the peripheral field so that an untouched portion of the field lies between the periphery and the scotoma, and (4) normal ophthalmoscopic findings

The rarity of this condition is probably due to the fact that it has never been emphasized and consequently it is frequently overlooked In the textbooks on perimetry little discussion has been devoted to this

1 Wilbrand, H Ueber die makular-hemianopische Lesestörung und die von Monakowsche Projektion der Makula auf die Schkphäre, Klin Monatsbl f Augenh 45 1, 1907

2 Posey, W Report of a Case of Right Homonymous Hemianopia in the Macular Regions, Ophth Rec 17 236, 1908

3 Stieren, E Central Homonymous Hemianopic Scotoma, Am J Ophth 7 764 (Oct) 1924

4 Newton, F H Paracentral Homonymous Hemianopic Scotoma, Am J Ophth 19 600 (July) 1936

5 Barkan, O, and Barkan, H Central and Paracentral Homonymous Hemianopic Scotoma, Am J Ophth 13 853 (Oct) 1930

6 Barkan, O, and Boyle, S F Paracentral Homonymous Hemianopic Scotoma, Arch Ophth 14 957 (Dec) 1935

type of defect in the visual field Traquair⁷ merely defined the defect. He stated:

A hemianopic scotoma is a central hemianopia. The central field in this respect resembles the whole field in miniature, the apices only of the quadrants affected being defective. Sparing of the fixation area is uncommon in hemianopic scotoma.

Traquair's term "fixation area" is not clear. If by this he means the macular area, then all cases reported in the literature in which there were sparing of the macula and preservation of good visual acuity are in contradiction to his statement. Fuchs⁸ stated:

In the case of partial hemianopic defects the lesion has the same situation but instead of affecting all, affects only a part of the fibres running from the chiasm to the cortex on one side or a very limited part of the visual cortex itself. Such partial defects may be limited to a central or paracentral scotoma symmetrically placed in the visual field of each eye, and such defects may be central—even cortical in origin.

The present lack of knowledge concerning the anatomy of some of the visual pathways and the fact that postmortem examination has not been performed in any of these cases to date make a discussion of the location of the lesion more or less futile. We can safely say, however, that the lesion may be at the level of the lateral geniculate bodies or anywhere above that point. The etiologic factor is undetermined but probably is a vascular accident in most cases. Toxic conditions, emboli and local infective processes must be considered.

In reviewing the cases thus far reported, including the case reported here, certain definite conclusions can be drawn: 1 Homonymous hemianopic scotomas are not as rare as formerly believed. 2 This type of defect in the visual field is usually associated with a constant syndrome, namely, (a) sudden onset of a defect in the visual field in persons often otherwise healthy, (b) characteristic complaints of difficulty with reading and fixation, (c) normal ophthalmoscopic findings, (d) absence of reduced visual acuity and (e) good prognosis for visual acuity. 3 The cause and the anatomic changes of this condition are still matters of conjecture. 4 Homonymous hemianopic scotoma is a condition that may be easily overlooked during routine examinations unless careful attention is given to the history and a perimetric examination made of the central field. 5 The case reported here agrees in most details with the findings in those previously reported.

REPORT OF CASE

Mrs. C. B. S. aged 48 was first seen June 28, 1937. She complained of a severe generalized headache of three days' duration and difficulty in focusing objects and in reading. A few hours previous to the onset of the headache she noticed that her "right hand vision was fuzzy." She stated that she saw only

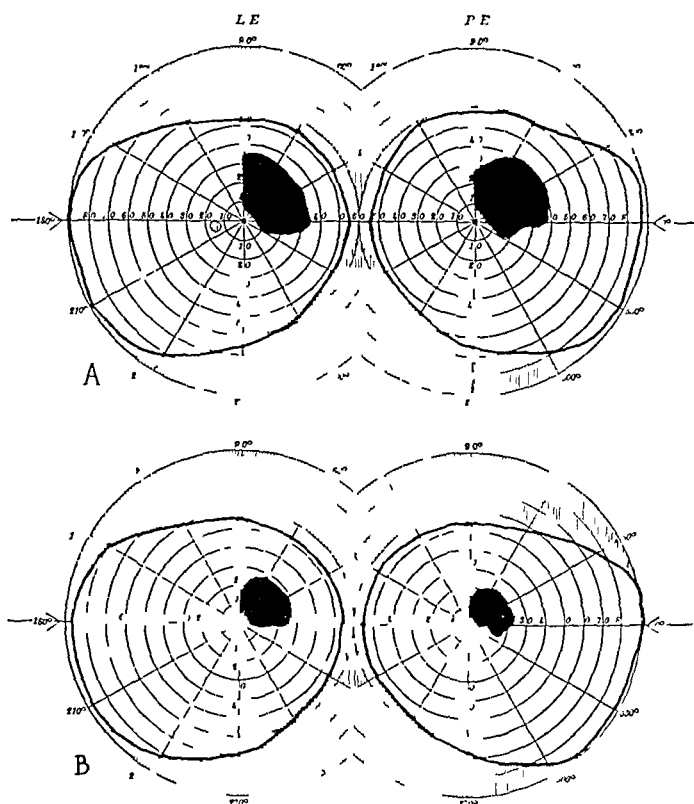
⁷ Traquair, H. M. *An Introduction to Clinical Perimetry*, London, H. Kimpton, 1927.

⁸ Fuchs, E. *Text Book of Ophthalmology*, ed. 8, translated by A. Duane. Philadelphia, J. B. Lippincott Company, 1924.

the left half of things. This visual disturbance persisted for a few hours and then began to clear, up so that at the time of our first examination her only visual complaints were "fuzzy" vision and difficulty in reading.

Further inquiry revealed that the patient was being treated by Dr. Harold O. Jones for uterine fibroid tumors and mild secondary anemia. Subsequently hysterectomy was performed by Dr. Jones, the cause of the secondary anemia thereby being removed. A general examination by Dr. Fred E. Ball did not reveal any other organic disturbances.

Vision in the right eye with a $+0.62$ sph, $+0.38$ cyl, ax. 180 was 20/20 +. Vision in the left eye with a $+0.25$ sph, $+0.38$ cyl, ax. 170 equaled 20/20 +. The near point with a $+1.25$ sph addition was 32 cm. for Jaeger test type I.



A, visual fields of patient taken on June 29, 1937, with a 3 mm white test object at 330 cm, and an illumination of 7 foot candles. *B*, visual fields taken on April 21, 1938, with a 3 mm white test object at 330 cm and an illumination of 7 foot candles.

The intraocular tension in each eye was 20 mm (Schiotz). There was no disturbance of function of either the extraocular or the intraocular muscles. The fundi were entirely normal, there were no pathologic changes in the retinal vessels, and both optic disks were of normal color and structure. The only abnormal ocular finding, except an error of refraction and presbyopia, was a right homonymous hemianopic paracentral scotoma. This defect was found on repeated examinations. Ten months after our first examination the scotoma had decreased somewhat in size. A complete disappearance is not expected and it will in all probability become stationary and permanent.

After consultation with the patient's general physician, 5 minims (0.31 cc) of sodium iodide three times a day was ordered empirically. This was used continuously from Oct 6, 1937, until April 28, 1938. No other form of treatment was instituted.

The prognosis in this case as in others like it is good, no loss of visual acuity is expected, and the patient should experience no visual difficulties once she accommodates herself to the presence of the blind-spot in her field of vision.

Abstracts from Current Literature

EDITED BY DR WILLIAM ZENTMAYER

Bacteriology and Serology

ANTIGENIC PROPERTIES OF CORNEA AND VITREOUS E WOLLMAN,
P GONZALES and P DUCREST, *Compt rend Soc de biol* 127
1188, 1938

The transparent tissues of the eye, cornea and vitreous do not share the immunologic properties of the lens but react in the same way as other vertebrate tissues. Rabbit-ox antiserum precipitates suspensions of cornea and of vitreous but does not affect that of ox lens. The lens of the cephalopod, however, reacts like the other tissues of its organism.

J E LEBENSOHN

Conjunctiva

ANTEPOSITIO CONJUNCTIVAE FORNICIS OPERATION IN SEVERE CASES
OF SPRING CATARRH N I SHIMKIN, *Brit J Ophth* 22 287
(May) 1938

The article of Shimkin is concluded with a summary, part of which is quoted here.

"The author points out that pathological investigations have shown that in spring catarrh the tarsus of the lid is quite healthy, and the affection consists in the disease of the conjunctiva and subconjunctival tissue only, in consequence of the abnormal growth of this tissue and its hyaline degeneration in the region of the tarsal conjunctiva.

"In view of the fact that the tarsus of the lid in spring catarrh is healthy, and the tarsal conjunctiva only is diseased, the author recommends careful excision of all the conjunctiva and subconjunctival tissue from the whole surface of the tarsus, beginning at the extreme lid border, and replacing it with healthy conjunctiva of the fornix. The author calls this operation 'antepositio conjunctivae fornicis,' as the conjunctiva of the fornix, separated deeply down towards the eyeball, is drawn forward on the tarsus, freed from the morbid conjunctiva and subconjunctival tissue."

W ZENTMAYER

LIMBIC FORM OF SPRING CATARRH FROGE, POURSINES and CHINIARA,
Ann d'ocul 175 236 (March) 1938

Spring catarrh, although not rare in France, is much less frequently seen than in other regions, such as the Orient. In France this disease is not only frequent but is also a continual source of worry to ophthalmologists because of the resistance it presents to all forms of treatment. In some cases cure is spontaneous, and in others the condition seems indifferent to all therapeutic measures.

The palpebral form often coexists with trachoma and does not attract the attention of the patient as does the limbic form. On the other hand, the limbic form may exist alone without any lesion of the palpebral conjunctiva, and it is this form that the authors here describe.

Details as to the clinical and microscopic aspects are given. The authors believe it necessary to differentiate between the lesions of the adult and those of the child and the adolescent. Adults, for instance, do not show, except in exceptional cases, the considerable edema of the limbus seen in the infant and the adolescent, the bulbar lesion of the adult consists chiefly of a simple hypervascularization. Often in the adult, as pointed out by Lagrange, one finds an anaphylactic phenomenon. This is not the case in the infant. The limbic form of spring catarrh presents in the infant an ocular lesion peculiar to its age and similar to phlyctenular keratoconjunctivitis.

S H McKEE

FUNGI IN NORMAL AND DISEASED EYES A FAZAKAS, *Arch f Ophth* 138: 416 (Feb) 1938

Fazakas has made a systematic study of the fungi which can be grown from normal and from diseased eyes. The incidence of positive growth (37 per cent of all diseased eyes and 26 per cent of all clinically normal eyes) and the variety of the fungi are amazing.

P C KRONFELD

Congenital Anomalies

A CASE OF PRIMARY BILATERAL ANOPHTHALMIA (CLINICAL AND HISTOLOGIC REPORT) E RECORDON and G M GRIFFITHS, *Brit J Ophth* 22: 353 (June) 1938

The developmental anomaly reported in this article was seen in a male child. Pregnancy and labor had been normal. There was no history of a developmental anomaly in the family of either parent, and there was no consanguinity. The child died of bronchopneumonia at the age of less than 3 months. A detailed anatomic description of the brain is given. Serial sections were made of the orbits. On the left side the sections were vertical and on the right side horizontal. In both orbits the six extrinsic muscles and their nerves were found embedded in fat. On the left side, four rectus muscles were attached by their anterior end to a minute nodule of fibrous tissue containing pigment granules, apparently lying free and not containing any recognizable cell structure. The pigment was brownish and did not give a reaction to the test for iron. The nodules did not contain any trace of retinal neuroepithelium or any type of cell other than the fibrous tissue. On the right side the muscles were not attached to the fibrous mass.

The author maintains that the case is one of bilateral primary anophthalmia in which there has been an absolute failure in the development of the optic vesicles and that it is distinct from those cases of

"degenerative" or "consecutive" anophthalmia in which the vesicles appeared to have formed and subsequently to have atrophied

The article is illustrated

W ZENTMAYER

Cornea and Sclera

VITAMIN D COMPLEX IN KERATOCONUS ETIOLOGY, PATHOLOGY AND TREATMENT OF CONICAL CORNEA, PRELIMINARY REPORT A A KNAPP, J A M A 110 1993 (June 11) 1938

After referring to a recent paper by Blackberg and himself in which the consistent production of keratoconus in dogs fed a vitamin D deficient, low calcium diet was reported, the author states that more recent experiments on rats have produced similar results. The results of treatment of 11 patients with vitamin D, in the form of viosterol, and calcium, in the form of mineral mixture tablets, are presented

The author gives the following conclusions

"A consideration of this material suggests that the calcium-phosphorus metabolism is an important factor in the development of the cornea. It would appear that the lack or deficiency of these metabolic factors would favor weakness of the membrane. This weakness then may manifest itself as a preliminary, noninflammatory ectasia of the cornea. The administration of Vitamin D and calcium in this condition has given gratifying results. In fact, the results apparently are so encouraging that it may be we have come upon a factor or the factor in the etiology of this perplexing pathologic entity. It is true that but eleven patients (eighteen eyes) with a rare condition have been treated. Still, the regular decisive improvement of all suggests that the vitamin D complex has a definite place in the therapy of keratoconus. It is hoped that further observations along this same line will serve to give a more conclusive conception of its place in our understanding and treatment of keratoconus."

W ZENTMAYER

Experimental Pathology

EXPERIMENTAL PRODUCTION OF LOCAL EOSINOPHILIA, PARTICULARLY IN RELATION TO SPRING CATARRH A KITSUKAWA, Acta soc ophth jap 39:17 (Feb) 1938

In this investigation Witte peptone was given intravenously. A degree of shock was produced which resembled an anaphylactic manifestation. It is therefore believed that this peptone, as well as histamine, is important in the study of anaphylaxis.

Tests with Witte peptone were carried out in the same manner as those with egg albumin. The results were as follows. If the peptone was instilled into the eye of the guinea pig which had not been previously prepared, no changes resulted, if the animal, however, had been sensitized, the instillation of the peptone caused a local anaphylaxis in the conjunctiva.

Microscopic changes were the same as were found in experiments with egg albumin, that is, there was a fleeting edema of the con-

junctiva, and on microscopic examination local eosinophilia of the conjunctiva was found after ten and after twenty-eight hours

ARNOLD KNAPP

General Diseases

DISEASES OF THE EYE IN LEPROSY P S RAMEYEV, *Vestnik oftal* 11: 787, 1937

Rameyev observed among leprous patients in a leprosarium in Uzbek (Turkestan) 38 persons, or 66.6 per cent, with ocular lesions. Many cases of trachoma were found in the region, but none of the leprous patients was affected with the disease. Consequently, Rameyev believes that possibly leprous patients are immune to trachomatous infection.

The common ocular lesions associated with leprosy are discussed.

1 Madarosis, or falling of the eyelashes, is one of the first and constant signs of leprosy and is due evidently to trophic disturbance in the hair follicle. Lagophthalmos is observed in the neural form and leads to blindness, if tarsorrhaphy is not done early, before opacification of cornea sets in.

2 The sclera is affected next in frequency. The color of the lesion is violet or pale yellow. The nodules or lepromas in the sclera have a tendency to spread on the cornea and conjunctiva, the shape of the lesion is frequently triangular, with its base at the limbus.

3 The cornea is frequently involved (in about 80 per cent). The process is sluggish, without reactive manifestations, so that the patient often notices it only when the vision is impaired because of central opacities of the cornea. Ulceration as a rule hardly ever occurs.

4 Leprous iritis and iridocyclitis are frequently of a low grade, chronic, serous plastic type, with exudates which lead to early occlusion and seclusion of the pupil, if treatment is not administered at once. At times miosis and irregular pupils are noted at the early stage of leprosy. Leproma of the iris is often located at the lower part of the anterior chamber. Chaulmoogra oil acts favorably in cases of keratitis and scleritis. Surgical excision of the leproma usually gives good results.

Rameyev advocates that an oculist be present at a leprosarium or that physicians in charge should have sufficient knowledge of clinical ophthalmology in order to prevent many cases of blindness.

O SITCHEVSKA

Glaucoma

THE OSMOTIC PRESSURE OF THE AQUEOUS HUMOUR IN EPIDEMIC DROPSY GLAUCOMA E O'G KIRWAN and S N MUKERJEE, *Brit J Ophth* 22: 329 (June) 1938

In 1934 Kirwan showed that epidemic dropsy is the only general disease at present known in which glaucoma forms an integral part, and hence is the most important clinical lead ever presented on the pathogenesis of glaucoma. The authors have studied the osmotic pres-

tures of the aqueous humor in normal eyes and in glaucomatous eyes in cases of epidemic dropsy and give the following summary and conclusions

"1 In epidemic dropsy glaucoma the difference between the osmotic pressure of blood serum and the aqueous humour is considerably reduced as the result of the appearance of proteins in the latter

"2 In consequence a reshuffling of ions takes place exactly in the same direction as predicted by the hypothesis of membrane equilibrium. The concentration of chlorine ions diminishes in the aqueous humour while an increase can be observed in the serum

"3 The evidence tends to support the view that in epidemic dropsy glaucoma the filtration of fluid into the anterior chamber takes place by the process of dialysation as usual, but that the equilibria concerned are at a level different from that of normal subjects, due probably to the altered permeability of the dialysing membrane"

W ZENTMAYER

Injuries

INJURIES ATTRIBUTED TO GLASSES OF AVIATORS R DE GAULEJAC,
Ann d'ocul 175 315 (April) 1938

The glasses of aviators are a constant cause of injury in aviation accidents. The injury may not only involve the orbit but at times the periorbital region, the nose and the neighboring sinuses. Though often superficial, the injury may affect adjacent bone, fracturing the nose and breaking into the frontal sinuses and bruising the inferior part of the orbit.

The injuries do not seem to vary with the kind of spectacles used, all seem to cause ocular injuries, orbital and periorbital and superficial and deep. In 4 of 8 recent cases of injury glass cut the tissues more or less severely, while in 3 the injuries were so severe that a detailed account of each is given.

S H MCKEE

Lens

THE OCCURRENCE OF SO-CALLED DINITROPHENOL CATARACTS WITHOUT INGESTION OF DINITROPHENOL H BARKAN and J W BETTMAN, Am J Ophth 21. 165 (Feb) 1938

The following summary is given

"An account has been given of a woman, 23 years old, who took a reducing agent, 'Slendrets,' for a week and lost only two pounds in weight. This preparation contained no dinitrophenol, yet the woman developed cataracts similar in every way to those we have associated with the ingestion of this drug.

"Thorough examination and history were negative except for the following data. A fractured nose, fracture of the right maxilla and the use of ergot over two years before entry, a fever of unknown etiology

one year before entry, use of quinine three months before entry, the extraction of five teeth because of apical abscesses two months before entry, and chronic pelvic inflammatory disease

"We do not believe that the above-mentioned positive findings were pathogenic factors in this case. The cause of the patient's cataracts remains unknown. They are certainly not coincident with the taking of dinitrophenol."

W S REESE

INTRA-CAPSULAR EXPRESSION OF CATARACT C V KRISHNASWAMI,
Brit J Ophth 22:274 (May) 1938

The method of expression employed by the author is a modification of the Smith procedure. Anesthesia is produced by blocking the facial and ciliary nerves. The corneal incision is a little less than half the circumference of the cornea. A conjunctival flap is formed. A radial incision is made in the iris. With the strabismus hook placed on the lower part of the cornea, gentle pressure is exerted toward the lower pole of the lens, and at the same time the bend of the hook is stroked to and fro along the lower limbus. Under this maneuver the zonule gives way at its weakest spot, which differs in different eyes. The stroking is now stopped, and the pressure alone is continued. The lens slowly expands the pupil and comes out through it, and then out of the section. The lens takes on a characteristic molding, depending on its consistency, in much of the manner of the fetal head coming out of the pelvis.

Illustrations accompany the article

W ZENTMAYER

THE p_H OF LENS IN NAPHTHALENE CATARACT OF RABBIT P REISS
and J NORDMANN, Compt rend Soc de biol 128:111, 1938

In the rabbit the p_H of the normal lens ranges from 7.1 to 7.5. In lenses with naphthalene cataract the shift was toward alkalinity rather than toward acidity as expected, the p_H of mature naphthalene cataracts averaging 7.8. The anticipated tendency toward greater acidity occurred, however, when the lenses were removed in the initial stage of the disturbance, the p_H of these lenses varying from 7.0 to 7.2.

J E LEBENSOHN

Lids

TRANSPLANTATIO CONCHAE AURICULAE TO CORRECT SPASTIC ENTROPION FOLLOWING TOTAL TARSECTOMY N I SHIMKIN, Rev internat du trachome 15:15 (Jan) 1938

The author has observed 5 cases of spastic entropion following total tarsectomy of the upper lid (Heisrath-Kuhnt excision). He restores the integrity of the lid by grafting auricular cartilage. A layer about 2 cm long, 1.5 cm wide and 0.75 mm thick, excised with its perichondrium conserved, is implanted in the lid through an incision 5 mm from

the border of the lid and retained by three Snellen sutures, which pierce the middle of the cartilage and the remaining strip of tarsus and are brought out between the lashes

J E LEBENSOHN

Ocular Muscles

MUSCLE WEAKENING BY CENTRAL TENOTOMY W G WATROUS and J M D OLMSTED, *Am J Ophth* 21:182 (Feb) 1938

On the basis of experiments on decerebrate cats the authors present the following conclusion

"Evidence has been presented which shows that if anything is accomplished by central tenotomy the beneficial results are due not to weakening of the hypertonic muscle through its tendon, but simply to a decrease in the mechanical advantage of the muscle acting on the eyeball through the formation of a new insertion, the result of adhesions"

W S REESE

Operations

GONIOSCOPY OF THE SURGICAL COLOBOMAS OF THE IRIS M PUIG SOLANES, *Am J Ophth* 20:731 (July) 1937

Puig Solanes discusses the theories explaining the beneficial effect of iridectomy. He concludes from a gonioscopic study in 5 cases that "(a) The reopening of the iridociliary angle is not necessary to the success of iridectomy in glaucoma (b) The excision of the base of the iris—unless future observations show the contrary to be true—is not accomplished in the majority of cases of iridectomy (c) The existence of an open angle and the perfect excision of the root of the iris do not guarantee the therapeutic success of the operation" He believes that the study of the action of surgical traumatism on the nervous system of the eye will help one to understand better the mechanism responsible for the good results in operations for glaucoma

W S REESE

A CORNEO-SCLERAL SUTURE IN CATARACT EXTRACTION ITS TECHNIQUE AND ADVANTAGES H B STALLARD, *Brit J Ophth* 22:269 (May) 1938

Stallard considers among other advantages of a corneoscleral suture its value in the prevention of postoperative hyphema, whereas previous to its use this complication occurred in about 30 to 35 per cent of cataract extractions. In the present series in which a suture was used there has been no such complication. The author describes an eyeless corneoscleral needle which he has devised. The point of the needle is dipped into the anterior half of the substantia propria of the cornea at a point 1 mm inside the limbus at 12 o'clock. The needle is made to traverse from 2 to 2.5 mm of the cornea in a transverse direction before emerging. With the point of the needle facing in the opposite direction, the needle is then passed through the conjunctival and subconjunctival tissues and engages in the superficial layers of the sclera 1.5 mm above

the limbus opposite the site at which the needle emerged. It passes transversely through the superficial half of the sclera for 2.5 mm and is brought out through the conjunctiva opposite its entry wound in the cornea.

The article is illustrated

W. ZENTMAYER

Orbit, Eyeball and Accessory Sinuses

AN ANALYSIS OF 71 CONSECUTIVE CASES OF UNILATERAL EXOPHTHALMOS. M. E. RANDOLPH, *Am J Ophth* 21:169 (Feb) 1938

This analysis confirms the view that sarcoma is the most common primary orbital tumor and indicates that the most frequent cause of unilateral exophthalmos in children is sarcoma, a rapid exophthalmos in the absence of inflammatory symptoms being strong presumptive evidence for this diagnosis. The mortality is high. Randolph cautions against the drainage of an orbital abscess secondary to infection of the adjacent sinuses until it has become localized. He mentions the trans-frontal intracranial approach for deep orbital tumors, suggesting that persons with such tumors be referred to the neurosurgeon.

W. S. REESE

LUXATION OF THE EYEBALL WITH COMPLETE RESTORATION OF ITS FUNCTION. REPORT OF A CASE. P. L. AKKERMAN, *Vestnik oftal* 11:581, 1937

A student came to the clinic with the history of having injured the left eye, "which snapped out of the orbit" while he tried to pass a half-closed gate in the dark. The left eyeball was found lying in front of the lids, which were spastically closed. The cornea was lusterless, and the vision was limited to perception of hand movements. An attempt at reposition of the eyeball by opening the lids was unsuccessful. Akkerman did a canthotomy, and the eyeball was placed into the socket with no difficulty. Slight exophthalmos was present, the conjunctiva was torn away from the upper half of the eye, and the site of the insertion of the external rectus muscle was swollen and bleeding. On the next day the vision was 0.4, chemosis of the conjunctiva was present, but the cornea and fundus were normal. The vision was restored to normal within a week, and the motion of the eyeball was normal in all directions.

The mechanism of the luxation is discussed, and a review of the literature is given. The importance of reposition of the eyeball before the optic nerve is stretched too much is emphasized. Simple canthotomy was a useful measure in this case.

O. SITCHEVSKA

Physiology

A PROCEDURE FOR MEASURING THE THRESHOLD SENSIBILITY OF THE RETINA. A. CHEVALLIER and H. ROUX, *Compt rend Soc de biol* 128:231, 1938

The dark-adapted subject places his head within an opaque globe lined with opaline. This acts as a diffusing screen for the feeble blue

light projected through an optical system similar to that in the photometer of Chaipentier. The illumination is then steadily reduced till no sensation of light is recognized. The entire visual field is thus equally stimulated, and the findings are independent of central visual acuity. The method gives constant values and is more precise and delicate than the test with Tscheining's glasses.

J. E. LEBENSOHN

Refraction and Accommodation

BENZEDRINE IN CYCLOPLEGIA S. J. BEACH and W. R. McADAMS,
Am J Ophth 21 121 (Feb) 1938

Beach and McAdams find that if benzedrine is used with homatropine or atropine little of the last two drugs need be used to obtain practically as good cycloplegia as when these cycloplegics are used repeatedly, and that the effect wears off much more quickly, permitting patients to use their eyes within one day after the use of homatropine and benzedrine and within from two to four days after the use of atropine and benzedrine. They favor using a 5 per cent solution of homatropine or a 1 per cent solution of atropine followed in two or three minutes by a 1 per cent solution of benzedrine and then a second instillation of the cycloplegic.

W. S. REESE

ON A CASE OF TRANSIENT INFLAMMATORY MYOPIA P. GORSE and
R. BERGES, *Ann d'ocul* 174 844 (Dec) 1937

The term inflammatory myopia is borrowed from an article by Jean Sedan (*Ann d'ocul* 163 358, 1926) in which he presented two personal observations. Tscherning not only calls the condition inflammatory myopia but says that this high myopia, or dangerous myopia of the illiterate, is often congenital and accompanied by insidious choroiditis.

Publications by different writers are reviewed, and the following case is described. A man aged 53, of robust health, had fifteen years previously been given glasses (a —10 D sph for the right eye and a —0.50 D sph for the left eye). He was again seen by the authors in June 1936 for refraction. The correction consisted of a —0.75 D sph for the right eye and an —0.50 D sph for the left eye. The following September a spasmodic type of rhinitis developed, and the eyes were so red that the condition was considered an allergic conjunctivitis. At the beginning of October he returned for examination, as the rhinitis still persisted. The right eye was normal, but the left eye was still red. The conjunctiva was injected and had a granular appearance, a large area of episcleritis was also seen. A curious phenomenon was that the patient complained of colored vision, yellow to the left and blue to the right. Treatment was instituted, consisting of the use of zinc sulfite, epinephrine ointment and, after the chemosis had subsided, yellow mercuric oxide ointment.

The condition in this case resembled spring conjunctivitis with prompt localization of the inflammatory symptoms in the left eye. Because of the persistent nature of the condition, the possibility of syphilis was considered and mercurial treatment was prescribed, which was undeniably beneficial. Since then the presence of an old syphilis

has been confirmed, but this is of secondary importance, the authors' first intention being to report a study of transient myopia from phlegmasia of the ocular membranes

S H McKEE

Retina and Optic Nerve

VARIATIONS IN THE PRESSURE OF THE RETINAL ARTERIES AND IN THE CAPILLARY PRESSURE IN RELATION TO COMPRESSION OF THE GLOBE C GANDOLFI, *Ann di ottal e clin ocul* 65:841 (Nov) 1937

Since pressure on the eye produced various reflex vascular changes, such as the oculocardiac reflex, the author studied the effect of such pressure on the vessels of the opposite eye. The pulse and general blood pressure as well as the pressure in a branch of the central retinal artery and the pressure in the macular capillaries were recorded before compression and again after compression sufficient to elicit the oculocardiac reflex. The retinal arterial pressure was recorded by Bailliant's dynamometer, and the capillary pressure was recorded by an apparatus similar to that used by Horniker. With this the patient, while observing a lighted blue-glass screen, notes as an entoptic phenomenon the pulse wave in the capillaries of the macular region, which cease when pressure sufficient to obliterate these vessels is employed. Data obtained by the latter method are open to certain objections and cannot be obtained for all patients. Twenty normal persons were employed, most of them being from 20 to 45 years of age.

After compression of the globe, the pulse was retarded and the general diastolic and systolic blood pressure was increased from 5 to 10 mm in most patients. The diastolic pressure in the retinal arteries was increased in 18 cases and remained the same in 2. The increase amounted to from 2 to 10 mm. The diastolic capillary pressure could be estimated in only 17 cases and was increased in 12. The increase in pressure seems to be mediated, like the oculocardiac reflex, by the trigeminal and the vagus nerves.

S R GIFFORD

DEHYDROGENATING PROCESSES IN THE RETINA AFTER INTERRUPTION OF THE CIRCULATION A FERRARA, *Ann di ottal e clin ocul* 65:851 (Nov) 1937

The author interrupted the central retinal vessels of rabbits for periods of from fifteen to ninety minutes by a ligature about the optic nerve at its exit from the globe. The eyes were removed from seven to eleven days later, and the retinal tissue was tested by Thunberg's method for its power to reduce methylene blue, the retina of the other eye being used as a control. The change in dehydrogenative power was too slight to be considered of any significance.

S R GIFFORD

ALKALINE RESERVE OF THE BLOOD IN IDIOPATHIC RETINAL DETACHMENT A BUCALOSSO, *Ann di ottal e clin ocul* 65:919 (Dec) 1937

The literature on the relation of acidity and alkalinity to swelling of the vitreous is reviewed. The vitreous has been found to be abnor-

mally alkaline in cases of glaucoma by some observers and has been shown experimentally to swell when its alkalinity is increased. Since a certain equilibrium must be assumed between the blood and the vitreous, the author investigated the alkaline reserve of the blood of 24 persons with idiopathic retinal detachment as compared with that of 10 normal persons. The average for the controls was 61.20 per cent, while that for the persons with detachment was 56.95 per cent. Figures of from 42 to 52 per cent were found for 8 persons with detachment, which brings the condition in these cases into the class of mild acidosis. In the cases of bilateral detachment the average value was especially low (52.40 per cent). It seems likely to the author that a low alkaline reserve is one of the factors favoring retinal detachment in certain cases.

S. R. GIFFORD

DETACHMENT OF THE RETINA AND ACCIDENTS. W. LOHLEIN, *Klin Monatsbl f Augenh* 99: 376 (Sept.) 1937

Lohlein undertook to formulate a clear understanding of the connection between detachment of the retina and accidents, giving especial consideration to expert testimony. Deploring the contradictory views of leading ophthalmologists, he mentions the two predominating reasons thereof: first, the diverging conceptions regarding the etiologic basis of traumatic retinal detachment and the impossibility to reenact the causative processes convincingly by experiments, and, second, the disagreement as to whether a certain trauma represents an accident in the sense of the law. Lohlein agrees with Velhagen, in whose opinion the final decision on the latter question must be left to the lawyer, the oculist may decide merely whether and to what extent the trauma may have been contributory in causing the detachment.

Taking it for granted that some injuries may lead to retinal detachment, the author describes and discusses the following types of causative injuries:

- 1 The first type consists of penetrating lesions of the anterior segment of the eyeball, followed by loss of vitreous and secondary traction through cyclitic hulls, and lesions of the posterior segment of the globe, causing tears of the retina and the consecutive formation of cords in the vitreous.

- 2 The second type consists of direct contusion of the eyeball, such as by transmission belts, footballs and tennis balls, the specific lesions caused by each being described.

- 3 The third type consists of indirect contusion of the eyeball by a bruise of the skull. Refuting Zur Nedden's opinion that injuries of this kind influence the interior of the eyeball only in a limited manner, Lohlein refers to gunshot wounds of the skull and face. Extensive tears of the choroid and retina resulted from indirect contusion without a lesion of the orbit. Healthy young men were knocked down by an exploding shell, receiving no direct injury but suffering a hole in the macula. A number of cases in point are cited, some of which were observed by the author. Extraordinary was the case of a healthy boy aged 16, with emmetropic eyes and no hereditary impediment. The

boy suffered a tear and detachment of the retina in one eye due to a fall on his face while walking on the street. The second eye remained intact during eight years' observation after the accident.

4 The fourth type consists of indirect contusion of the eyeball by severe concussion of the body. The mechanism of this type of injury, compared with that causing concussion of the brain, is illustrated by the reports of several cases and is affiliated in some instances with recurring detachment of the retina.

5 The fifth type consists of detachment of the retina after especially strenuous exertion of the body. This type requires great care of decision. Profuse hyperemia of the head and eyeball exists during bodily overexertion. Therefore, the difference between the pressure in the retina and the choroid and the traction of one or the other of the extrinsic eye muscles on the sclera may help in causing detachment of the retina.

In expert testimonies the following factors should be considered: the hereditary disposition to retinal detachment, the numeric distribution of detachment as to the age of the patient and the disposition of the senescent and myopic retina. The author adds his own cases to those of Vogt, Gonin and Lindner, in which irrelevant causes, such as sudden change of the gaze and forceful rubbing of the eyes, caused detachment of the retina in predisposed eyes. Lohlein adduces convincing material in disproving Zui Nedden's contention that trauma is irrelevant in the genesis of retinal detachment. Highly myopic eyes and belated detachments are given ample consideration in this connection.

The conclusion is this: that outside influences, including indirect injuries, are actually an important factor in the mechanism of retinal detachment. It is the oculist's duty to evaluate the anamnestic data and the clinical symptoms in an endeavor to arrive at a just and fair decision in each individual case.

K L SROLL

HARADA'S DISEASE K OKAMURA, *Acta soc ophth jap* 39:14, (Feb) 1938

Okamura has observed 16 cases of Harada's disease during the past two years. He included in this series cases of the type described by Harada and by Vogt. The patients were subjected to a careful general examination, which included a roentgenogram of the chest, the red blood cell sedimentation test, a study of the blood and the Mantoux test. These tests showed that all 16 patients were tuberculous. In fact, in 13 cases tuberculous changes in the chest were apparent roentgenographically, and in the remaining 3 the Mantoux test was positive. The Mantoux test was positive in 13 cases, and in 3 cases in which it was negative definite tuberculous changes were found in the chest on roentgen examination.

The eye presented changes which were suggestive of tuberculosis, such as nodules in the iris, disseminated choroiditis, retinal perivasculitis and hemorrhages of the vitreous.

The author concludes that Harada's disease is a form of tuberculous uveitis.

Similar general changes were found in 3 cases of sympathetic ophthalmia, and the author suggests that sympathetic ophthalmia is a form of Harada's disease, with a detrimental effect on the uvea

ARNOLD KNAPP

Trachoma

THE MICROSTRUCTURE OF EPITHELIAL CELLS AND ITS IMPORTANCE FOR THE AETIOLOGY OF TRACHOMA W GRUTER, Brit J Ophth 22: 300 (May) 1938

Gruter made the following observations in a study of trachoma and blennorrhagic conjunctivitis on examining the epithelium by vital and staining methods

"1 The secretory granulations swell and divide in the trachomatous epithelium. This process is diffuse (both in Golgi's apparatus and in the cytoplasmic zone) or focal (hood-shaped) (osmium impregnation)

"2 The ballonet structures referred to above swell up and divide both in the zone of Golgi and in the cytoplasmic zone

"3 The 'interior reticulated body' swells and turns cloudy (as can be seen clearly in the dark field and against the light). Typical signs of divisions then appear (fission of the polyhedral parts). An albuminous sediment forms in the honeycomb-like spaces in the polyhedral parts (visible in dark field and by eosin staining). Lastly appear fine nodules and still finer granules (like strings of pearls) on the interior reticulated body

"The *Hood-shaped structure* (Prowaczek's bodies) are not homogeneous but consist of various partial structures —

"(a) Focal proliferation of Golgi's sheath with more pronounced proliferation of the secretory granules (osmium images). These have also been described by V Reiss (*Arch f Ophthal*, 1913)

"(b) Interior reticulated body or partial structures protruding from under the nuclear calotte. Extremely fine nodules and granules appear (elementary bodies according to virus nomenclature)

"(c) Combination of (a) and (b), the reticulated body detaching itself and the inflamed Golgi sheath becoming stained with it "

In his conclusions, Gruter states "Inflamed trachomatous epithelial cells cannot be shown to contain any granular structure of a nature different from their own, whether in the Golgi zone, the interior reticulated body as described or in the cytoplasmic zone "

The author states that he cannot claim that the various granular structures described in literature and tested by him by so many different methods are foreign substances, i e, initial structures of a hitherto invisible disease germ in the cell. He considers the "trachoma rickettsias" described by Busacca to be inflammatory proliferations and divisions of granules which normally occur in the epithelial cells

W ZENTMAYER

STUDIES ON THE PATHOGENY OF TRACHOMA L. A. JULIANELLE,
Brit J Ophth 22: 336 (June) 1938

After a general survey of the literature on the pathogenesis of trachoma Julianelle details the work on which this paper is based. In his discussion he states

"The accumulated studies on the aetiology of trachoma by numerous workers as well as ourselves no longer permit a reasonable rejection of the evidence that the disease is an infection in its own right without the benefit of associated factors such as constitution, nutrition, co-related infections, etc

" by way of summary and conclusion, it may be said that the infectious agent of trachoma is characterised by low infectivity, occasional filterability, marked tissue specialisation, ineffectual immunogenic properties, slight propagative capacity, sensitivity to physical and chemical agents, and the accompaniment of inclusion bodies. All these properties suggest the probability that the infectious agent is a virus. Whether the virus may be defined in turn as the inclusion body or its component elements remains to be proved, although the evidence available at the present time suggests this may be actually the case, whether again the inclusion body may be defined further as a rickettsia must also wait upon further investigation. In the meantime, the three concepts of virus, inclusion body, and rickettsia are not so divergent as they appear at first glance, what differences each appears to have from the others are of the order less of fact and more of definition and classification."

W. ZENTMAYER

SUBCONJUNCTIVAL AUTOHEMOTHERAPY IN COMPLICATIONS OF TRACHOMA E. SELFA MARTINEZ, Rev internat du trachome 15: 87 (April) 1938

With the idea of stimulating the local defensive mechanisms in pannus ulcer and trachomatous keratitis, the author injects 0.5 cc of the patient's own blood in the superior cul-de-sac. The reaction is minimal, and marked improvement is generally observed within forty-eight hours. Nine cases are reported. This method has been extended to the treatment of pneumococcus hypopyon keratitis with equally encouraging results.

J. E. LEBENSOHN

Tumors

THE ANAPLASTIC OR DIFFERENTIATED GLIOBLASTOSPONGIOBLASTOMA OF THE RETINA G. FAVALORO, Ann di ottal e clin ocul 65: 881 (Dec) 1937

The classification of retinal tumors is discussed on the basis of modern knowledge as to the histogenesis of the various cells in the central nervous system and the types of neoplasm derived from them. Reasons are given for considering previous classifications unsatisfactory. The author reports his observations in 3 cases in which the clinical picture of so-called glioma retinae was present. Special stains, especially glial stains, showed that all 3 neoplasms were composed of

glia, but with the predominant cells in different stages of differentiation, producing three quite different histologic pictures

In 1 case the predominant cell was the small round cell, with scanty protoplasm and without prolongations. This is the type of cell which was formerly confused with that of the small round cell sarcoma. By its staining properties, however, and the detection of a few cellular spines, it may definitely be classed as an anaplastic or undifferentiated glia cell.

In a second case the cells were pleomorphic, differing greatly both in size and in shape, being bipolar, unipolar, pyramidal, elongated and irregular, with and without processes. These must be considered as typical or atypical spongioblasts. The cells of the rosettes seen in this and in similar tumors are spongioblasts of the ependymal series, which show a tendency to unite in circles, as do the spongioblasts about the neural canal. They have nothing to do with the neuroepithelial layer, and hence the term neuroepithelioma is a misleading one.

In a third case the cells were of various sizes, more or less irregularly rounded, with cellular processes which were short and spinous or long, anastomosing with each other to form a true neuroglial reticulum. These are spongioblasts of the astrocytic series, cells more highly differentiated than the two types just described. A fourth type of glia cell, the astrocyte, was not found in these tumors, but has been described in retinal tumors by Greef, Asuncion and others.

The author criticizes the common use of the terms retinocytoma and neuroblastoma because it has not been demonstrated that embryonal retinal or neural cells are present in the usual types of retinal tumor. He would separate the gliomas, including nearly all of the retinal neoplasms described, sharply from the neuroblastomas, it being doubtful if true examples of the latter type have been seen in the retina.

He proposes a classification of the gliomas on the basis of the types of cell present. Many tumors show different zones, in each of which the predominant cell is different. He distinguishes six types of cell: (1) the anaplastic glioblast (common), (2) the spongioblast of the astrocytic series (less common), (3) the spongioblast of the ependymal series (very common), (4) the glioblast, with many processes forming a netlike or asteroid syncytium (common), (5) the highly fibrillar glioblast, highly differentiated, with fibers united in bundles (rare), and (6) the astrocyte (not common).

On the basis of this classification, the tumor in the first case would be described as a spongioependymofibrilloglioblastoma, that in the second case as an anaplastic, differentiated ependymoglioblastoma, and that in the third case as an ependymospongioglioblastoma. The commonly malignant course of the retinal neoplasms is due to the common preponderance of the anaplastic, undifferentiated cell type.

A bibliography and 21 illustrations accompany the article.

S R GIFFORD

TUMOR OF THE LID IN A CASE OF BOURNEVILLE-PRINGLE'S DISEASE
E PURTSCHER and J WENDLBERGER, *Arch f Ophth* 138:388
(Feb) 1938

The term Pringle's disease is used to designate the cutaneous manifestations of tuberous sclerosis of the brain (Bourneville-Hartdegen's

disease) The authors report a case of tuberous sclerosis in a girl of 9 years In addition to other typical cutaneous manifestations of this disease, a fibroma-like tumor was removed from one upper lid Histologically, the tumor consisted of multiple small cysts, each of which contained one or several hair shafts The cysts had probably originated from hair follicles The deeper layers of the tumor consisted of imperfectly developed hair follicles, and in that portion of the tumor in an early stage of development there were arrested hair follicles The authors therefore recommend the name "follicle-nevus" for this growth

P C KRONFELD

Uvea

CHRONIC CELLULAR INFILTRATION OF THE UVEA IN SEPTIC ENDOPHTHALMITIS OF ECTOGENOUS ORIGIN H D LAMB, *Am J. Ophthalm* 21:137 (Feb) 1938

Lamb discusses the histologic changes following penetrating injury, operation or corneal ulcer After an examination of 32 eyes he found the most characteristic change of septic endophthalmitis to be the large number of polymorphonuclear leukocytes along the inner surface of the ciliary body and in the anterior part of the vitreous In each of these eyes there were macrophages or exudative cells of chronic inflammation in the anterior and posterior vitreous and in the anterior chamber There was in every case a chronic factor which tended to intensify as the original virulence of the etiologic toxin weakened

W S REESE

HEREDITARY CHORIORETINAL DISEASE M SCHUTZBACH, *Arch f Ophthalm* 138:315 (Feb) 1938

In a German family of 28 members belonging to 5 different generations, Schutzbach found 18 cases of obviously hereditary chorioretinal disease which occurred in two forms, viz, as progressive atrophy of the choroid leading to typical choroideremia or as a "pepper and salt" fundus without pronounced atrophy of the choroid The former type occurred only in men, and the latter, chiefly in women The relation between these two types was not clear The assumption is plausible that they represented different stages and degrees of the same disease The optic nerves and the retinal vessels remained normal in each type The mode of inheritance was probably of the dominant type The question of sterilization of the male members in whom the disease was definitely progressive was considered

P C KRONFELD

Therapeutics

RELIEF THERAPY ("ENTLASTUNGSTHERAPIE") FOR TABETIC OPTIC ATROPHY A MIKLÓS, *Arch f Ophthalm* 138:219 (Dec) 1937

The literature on the pathogenesis and the treatment of tabetic optic atrophy is reviewed During the last ten years the disease seems to have become milder and less rapidly progressive Lauber and Sobański believe that the course of tabetic optic atrophy is principally dependent on the

retinal circulation, that is, on the relation between the diastolic pressure in the retinal arteries and the intraocular pressure. With the dynamometric method, Lauber and Sobański have found the diastolic pressure in the retinal arteries to be so low in many cases of tabetic optic atrophy that chronic decompensation of the retinal circulation must have been present. The two authors have, therefore, recommended that tabetic optic atrophy be treated by relieving the circulatory decompensation (relief therapy, or "*Entlastungstherapie*"), that is, that therapeutic measures be employed which tend either to raise the blood pressure (tonics, caffeine and food rich in proteins) or to lower the intraocular pressure (pilocarpine or cyclodialysis).

Miklós has tried this form of treatment on a large number of patients and reports his results. He comes to the conclusion that there is a definite relation between the diastolic blood pressure and the course of tabetic optic atrophy. If the blood pressure remains abnormally low for a certain length of time, central and peripheral vision decrease rapidly. If the blood pressure rises or remains stationary at the normal or at a higher level, the vision remains stationary or decreases slowly. Tonics and caffeine, though generally ineffective as pressure-raising agents, are harmless and because of their possible usefulness are indicated in tabetic optic atrophy. Pilocarpine is, as a rule, equally ineffective as a pressure-lowering agent. In only a few cases did it lower the intraocular pressure by from 7 to 10 mm of mercury. Cyclodialysis is the most effective pressure-reducing procedure. "The ideas of Lauber and Sobański have proved correct. The method of relief therapy combined with antisyphilitic treatment is worth trying."

P C KRONFELD

SUCCESSFUL HYPERTHERMIC TREATMENT OF OPHTHALMOBLENNORRHEA BY THE APPLICATION OF A STEAM SPRAY. REPORT OF CASES I BRECHER, *Klin Monatsbl f Augenh* 99 301 (Sept) 1937

Brecher reviews the modern literature on regional hyperthermy as a therapy for ophthalmoblennoirhea. He refers to Wessely, who devised the original apparatus for the application of the "steam treatment" to the eye. Brecher obtains a constant temperature of 42 C with his method, used at a distance of from 10 to 12 cm from the eye. The virulence of the germs was destroyed without any adverse effect on the ocular tissues. Good results were obtained in 5 adults and 13 infants. The method is of great help in those cases in which the protein therapy is contraindicated. The treatment also relieved the pain and was of eutrophic value in cases in which there was complicating corneal involvement.

K L STOLL

Society Transactions

EDITED BY W L BENEDICT

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

PARKER HEATH, M D , Detroit, *Chairman*

Eighty-Ninth Annual Session, San Francisco, June 15-17, 1938

DERRICK VAIL, M D , Cincinnati, *Secretary*

NATURE OF THE FILTRABLE AGENT OF TRACHOMA DR PHILLIPS THYGESON, New York, and DR POLK RICHARDS, Albuquerque, N M

This article appeared in full, with discussion, in the October issue of the ARCHIVES, page 569

SCLEROMALACIA PERFORANS REPORT OF A CASE IN WHICH THE EYE WAS EXAMINED MICROSCOPICALLY DR FREDERICK H VERHOEFF and DR MERRILL J KING, Boston

This article will be published in full, with discussion, in a later issue of the ARCHIVES

PATHOLOGIC CHANGES IN AMBLYOPIA FOLLOWING TRYPARSAMIDE THERAPY DR P J LEINFELDER, Iowa City

Many instances of acute loss of vision have been observed after treatment with tryparsamide, but microscopic examinations of the eyes or optic pathways of persons so affected have not been reported. Reports of pathologic studies of the eyes and optic nerves of 2 persons in whom amblyopia developed after the administration of sodium arsanilate are available in the literature. A tabetic man aged 55 received an injection of 1 Gm of tryparsamide. Acute loss of vision was noted in forty-eight hours, which rapidly progressed to blindness. Death from uremia occurred nine days after the injection. A complete postmortem examination was made, and the eyes, optic nerves, tracts, lateral geniculate bodies and occipital cortex were removed for microscopic study. Acute degenerative changes were observed only in the ganglion cells and inner nuclear layer of the retina.

DISCUSSION

DR FREDERICK C CORDES, San Francisco. Of particular interest to me was the fact that examination of the occipital cortex, lateral geniculate bodies, optic tracts and nerves failed to reveal any evidence of acute inflammation or degeneration. The changes in the optic nerve were those of an old process, easily accounted for by the preexisting atrophy of the optic nerve. The changes noted in the retina have been observed in cases of acute poisoning from methyl alcohol, ethyl alcohol, quinine, nicotine and sodium arsanilate. According to Igersheimer's work, the last-mentioned drug also attacks the neuroepithelial layer.

Lillie and others have contended that the occasional reported cases of tryparsamide amblyopia have been primarily the result of an idiosyncrasy to the drug. The fact that 1 Gm produced such marked changes would tend to confirm this opinion. I should like to emphasize again the necessity of caution in the initial doses so as to detect any sensitivity to the drug.

Clinical observation has shown that aside from the subjective symptoms the earliest changes are those of a contracted field and microscopic changes in the optic disk. The fact that the greatest changes were found in the periphery of the retina and that no acute changes were found in the optic nerve emphasizes the importance of careful studies of the visual fields early in the course of therapy. Such studies will help to detect the occasional sensitive patient.

DR MAX FINE, San Francisco. It is unfortunate that the picture in Dr Leinfelder's case was somewhat confused by the presence of early atrophy of the optic nerve. There is general agreement, however, that tabetic atrophy of the optic nerve begins in the intracranial portions of the nerve. The absence of extensive atrophy in the presence of marked degenerative changes in the ganglion cell and inner nuclear layers suggests that the process involved is one other than tabetic atrophy of the optic nerve. A striking feature of the sections Dr Leinfelder has exhibited is the relative integrity of the central portion of the retina and the progressively severe changes as the periphery is approached. These findings correspond in a rough manner to the peripheral constriction of the visual field in cases of tryparsamide amblyopia. I should like to ask Dr Leinfelder whether in the study of his specimens he was able to find a difference in the extent of degeneration in the nasal and temporal halves of the retina. Such a finding would substantiate the clinical observation of relative sparing of the temporal field, which is an almost constant feature of tryparsamide amblyopia.

The delayed appearance of atrophy of the optic nerve in tryparsamide amblyopia, even when the changes in the visual field are severe, fits in with Dr Leinfelder's suggestion that the degeneration occurs first in the inner nuclear layer.

The question of the specific cause of tryparsamide amblyopia, the chemical group responsible and the reason why only 1 of 10 persons who receive the drug have ocular complications would appear to hinge on a factor either of idiosyncrasy or of a defective excretory mechanism. The occurrence of over 90 per cent of reactions with the first five injections speaks against a cumulative effect in the ordinary sense. Young and Meuhlenberger have shown through studies of urinary arsenic that whereas a great majority of persons excrete 95 per cent of the arsenic within twenty-four hours, a small number excrete the drug much more slowly, and in 1 instance arsenic was recovered from the urine after three weeks. It is possible that in such a person a sufficiently high concentration of the toxic agent is maintained in the susceptible tissues for a sufficiently long period to cause damage, while in the average person the same concentration may be reached but does not act over as long an interval. Studies in this direction should yield valuable information.

DR JOHN WEEKS, Portland, Ore. The only experiences that I have had with amblyopia due to the use of arsenical preparations in the

treatment of syphilis were with 2 cases of syphilis of long standing I regard the use of tryparsamide as something that should be undertaken only after careful tests have been made of the patient's sensitivity to the drug. There is rather a widespread opinion that whenever there is any atrophy of the optic nerve or any disturbance of the fundus from syphilis, tryparsamide should not be employed. I cannot speak positively in regard to that.

It is curious that the periphery of the retina should be so much more profoundly affected than the region of the macula. Amblyopia due to tryparsamide corresponds to some extent to that due to quinine, but tryparsamide has caused total blindness to persist in a few cases. No case of complete permanent blindness from quinine is on record so far as I have been able to discover.

DR FREDERICK H VERHOEFF, Boston. At present it seems to me to be of no practical importance whether tryparsamide or other arsenicals in producing blindness affect primarily the optic nerve or the ganglion cells, but it is of great scientific interest and may become of practical importance.

I have not the slightest doubt that Dr Leinfelder has shown in this case that the ganglion cells were markedly affected. I do not feel sure that the ganglion cells were primarily affected. It seems to me, in spite of the fact that histologically there is no evidence that the nerve fibers were primarily affected, that it is possible that they may have been affected to such an extent that as a result the ganglion cells would show changes, because any injury to the nerve fibers may also injure the ganglion cells.

As I say, this is not important at present, but it may become very important. I think that it is a question that arises constantly in regard to judging the effect of toxic agents on the optic nerve and retina. Frequently one cannot determine whether it is the ganglion cells or the optic nerves that are attacked primarily.

DR P J LEINFELDER, Iowa City. Dr Verhoeff's question concerning whether the ganglion cells or the nerve fibers were primarily affected can, I think, be definitely answered. Although neurohistologic technic is by no means infallible, research that I have done with the optic nerve has shown me conclusively that if the optic nerve is sectioned or injured, pathologic changes are observable on staining with osmic acid or by the Marchi method as early as three days after the injury. Furthermore, the neurofibril will be disintegrated, not in three days, but within ten days.

It is striking that in this case there was absolutely no evidence of acute degeneration in the nerve fiber of the optic nerve even close to the eyeball.

Dr Sharpe brings up the question of pupillary reactions. It has been conspicuous in the literature that pupillary reactions are at first retained in spite of the subjective blindness.

CORRECTION OF PTOSIS BY ATTACHMENT OF STRIPS OF ORBICULARIS MUSCLE TO THE SUPERIOR RECTUS MUSCLE. DR JOHN M WHEELER, New York.

This article will be published in full, with discussion, in a later issue of the ARCHIVES.

THEORY AND USE OF CROSS CYLINDERS DR EDWARD JACKSON, Denver

The cross cylinder planned for optical theory was not practically useful. Made to fit physiologic, psychologic and clinical conditions, it furnishes the best method of detecting and measuring astigmatism. Reversal in two positions shows astigmatism present or absent. The direction of its principal meridians is to be fixed first, and after that, the amount of astigmatism. The technic consists of holding the cross cylinder in the right position and reversing its meridians so that the patient can tell which way is best. A cylinder placed in the trial frame is to be turned or its strength changed until the best vision is obtained and any further change makes it worse. After the approximate correcting cylinder is reached, any further change makes vision worse. When the right cylinder is thus found, the strength of the spherical lens is to be varied to find the strongest convex lens and the weakest concave lens that will give the best vision. Optical theory, or experiments with cylinders, will show the reason for each step in such testing.

DISCUSSION

DR CHARLES K. MILLS, Woodland, Calif. Concerning my experience with the cross cylinder, I find that the test for the axis is practically foolproof, and when it is properly performed the patient will invariably select the correct axis, but great care must be exercised in making the test for strength or the patient's interpretation may introduce errors and lead one astray.

DR WILLIAM H. CRISP, Denver. The cross cylinder may be thought of as asking one of two questions. How much cylinder, and in what direction? The test for strength adds or deducts a cylinder of the same denomination as that before the patient's eye and gets the patient to say whether more letters are legible one way or the other. This test does not indicate just how much is to be added or deducted but merely shows that addition or deduction is to be made.

The test for axis offers two new axes and determines whether the patient wishes his lens turned in the direction of one or of the other. This test again does not immediately furnish an exact indication of how far the axis is to be moved but only that it is to be moved.

In each case the information is to be completed by making an arbitrary change of strength or axis of the kind indicated, and then the test is to be repeated until the point is reached at which no further change is demanded.

As to the problem whether to rely on the astigmatic measurements obtained under cycloplegia or those obtained after the pupil has returned to its normal size, I doubt whether one can safely dogmatize. I believe that in the majority of cases it is at least as safe if not safer to accept the astigmatic findings obtained under cycloplegia, for my own experience appears to indicate that in the majority of cases the brain has learned to select a certain limited area of the pupil and to disregard the peripheral zone when the pupil is dilated.

DR JOHN GREEN, St. Louis. The axis of the measurable astigmatism is more accurately determined by the cross cylinder than by astigmatic charts.

I am being rather dogmatic about this because these are the rules that I have laid down for my own guidance and my associates' guidance.

In determining the axis with the cross cylinder, keep the patient in a slight fog. This should be done whether or not the patient is under cycloplegia.

When the actual test is being made, admit to the patient that for the moment you have intentionally blurred his vision. Acknowledge that in neither position of the cross cylinder will the letters be crystal clear. "There are degrees of blurring. Neither side of this glass is good. Choose the lesser of two evils."

In determining the axis for astigmatism between 0.5 and 1 D, the most useful cross cylinder is the 0.37. In determining the axis for astigmatism under 0.5 D, the most useful cylinder is the 0.25.

In using the cross cylinder for strength, the sphere should be reduced to eliminate all fog. The most useful cross cylinders are (1) for astigmatism between 0.5 and 1 D, a 0.25 cross cylinder, (2) for astigmatism between 0.25 and 0.5 D, a 0.12 cross cylinder, (3) for astigmatism under 0.25 D, a 0.06 cross cylinder.

The 0.06 cross cylinder suggested by me ten years ago is in daily use and has been found indispensable for low degrees of astigmatism. With astigmatism under 0.5 D, this very weak cross cylinder will usually, at least for the observant patient, cause a reversal in distinctness of the Lancaster single cross lines or the triple barred cross of the Thomas chart.

VISUAL ACUITY ITS RELATION TO FORM SENSE AND THE APPLICATION OF THIS RELATIONSHIP TO MEDICOLEGAL PROBLEMS DR ALBERT C. SNELL, Rochester, N. Y.

A misunderstanding of the nature of the Snellen formula expressing visual acuity is responsible for much confusion in medicolegal cases. Visual acuity means sharpness of vision, Snellen's *Schärfe*. Sharpness is not a function of vision, it is not the form sense but a quality of the form sense. The form sense is properly measured by visual acuity tests which determine the size of the visual angle. From the principle of the "minimum separable" there is evolved the resolving power of the detail of the form sense, a visual function. The resolving power of an eye for all degrees of acuity can be determined mathematically. The principles as set forth in the report of the Committee on Compensation for Eye Injuries are scientific. Their general adoption by all states would remove much present confusion and establish a uniform method for evaluating visual disabilities.

DISCUSSION

DR D. F. HARBRIDGE, Phoenix, Ariz. If it is impossible to follow all the gyrations of this subject which Dr. Snell has gone through, one should remember the constant which he and his associates have evolved, that is, 0.83625. That is really the essence of the whole subject.

The report of the American Medical Association, adopted in 1925, as presented in the paper under consideration, represents much of the personal work and research of the essayist. The soundness of the method has been proved since the adoption of the report and is subject to no further challenge. The more the method is studied and applied, the

more one is convinced of its soundness. Experiment and experience covering many years were necessary to evolve the system delineated in the paper presented. All ophthalmologists do not come into contact with cases of industrial compensation, but those who do readily admit of the practicability of the method propounded in determining the visual efficiency of the patient whose ocular function has been disturbed by injury.

In 1926 Arizona created by law its present Industrial Commission and attending system of compensation. I was again afforded the opportunity of advising in the situation, whereupon I recommended the report of the American Medical Association to the Industrial Commission as a proper method for determining awards in cases of ocular injury. The adoption of the report by the commission maintains today.

For states not yet having adopted this report as a basis for awards in cases of ocular injury, local medical associations could likely bring about such an adoption by the appointment of a committee of ophthalmologists. In Arizona the state medical association annually appoints an Industrial Relations Committee of five physicians or surgeons to serve the Industrial Commission in an advisory capacity, especially as relates to debatable injuries and compensation. The commission has been most receptive to the suggestions and recommendations of this medical committee since its inception. The Arizona State Supreme Court has commended and upheld the position of this medical committee in its relation with cases of compensation through the Industrial Commission of the state. Arizona holds a unique position in nationwide industrial circles as regards this medical committee. States still not having adopted the report might consider similar contacts through their respective medical associations in bringing about a recognition of the method described.

DR RALPH O RYCHENER, Memphis, Tenn. One cannot avoid speculation when after the adoption of such an excellent report as that prepared by the Committee on Compensation for Eye Injuries one finds that there remain 23 states which have no legal schedule or method whereby visual losses may be computed. The question naturally arises as to why there should be such delay in protective legislation.

Organized labor, as a rule, affords the impetus for new laws relating to the protection of workmen, but this particular subject had never before been brought to the attention of the labor unions in Memphis. The local president proved to be intensely interested in my inquiry and has written me that he believes this schedule should be incorporated in the Workmen's Compensation Act and that he is preparing for legislation to that effect. The fate of such legislation will likely depend on the interest which ophthalmologists exhibit in explaining the benefits of such legislation to workmen and legislators who do not readily understand the technical phrases of the committee's report.

On the other hand, casualty insurance companies are not particularly interested in the adoption of such schedules but probably will not vigorously oppose them. The earnings of such companies are greater in the states under the old common law than in the states in which workmen's compensation acts are in vogue, because although higher losses are suffered in individual cases in the first groups, the rate of insurance

is increased sufficiently to make a handsome profit possible. I am assured, however, that the companies welcome a definite and fixed schedule for visual loss which will enable them to rely on definite appraisals of disability rather than on the opinions of several medical men, no two of which may be in accord as has been the case in previous litigation.

DR WILLIAM H CRISP, Denver. A number of states have tables which do not correspond to those of the American Medical Association as stated by Dr Snell's committee. About twenty years ago I was asked by the state of Colorado to prepare a table of this type. The best thing I could find at that time was the Chapman table of Wisconsin. I studied this table as I thought out carefully what the compensation was going to be in cases of extensive injury in which there were the average chances of diminished functional efficiency. It seemed to me the Chapman table was more generous than that of the committee of the American Medical Association and was not quite as good as it should be, and I slightly stressed those figures. The modified Chapman table has been in use by the state of Colorado ever since.

EXPERIMENTAL HYPERTENSION VASCULAR CHANGES IN THE EYES DR JOHN E L KEYES and DR HARRY GOLDBLATT, Cleveland

This article appears in full, with discussion, in this issue, page 812

FUSIONAL MOVEMENTS ROLE OF PERIPHERAL RETINAL STIMULI DR. HERMANN BURIAN, Hanover, N H

Studies concerning fusional movements of the eyes have been carried on by stimulating the central or central and peripheral parts of the retinas. No special investigations of the importance of the peripheral parts of the retinas alone in the production of fusional movements of the eyes have been made. Such a study is presented based on experiments with the aid of an arrangement which allows stimulation of any desired area of the two retinas with identical images, the size and form as well as the relative position of which can be controlled. The apparatus consists of projection lanterns and polarizing material. Fusional innervations to the extrinsic muscles result whenever peripheral stimuli fall on noncorresponding areas and the resultant relative vertical, lateral and cyclic movements of the eyes are in the direction of the displacement of the stimulated areas and are of almost the same amount, depending on the size of the stimuli and their distance from the fovea.

DISCUSSION

DR AVERY M HICKS, San Francisco. Under ordinary conditions of seeing, nearly all movements of the eyes are the result of images falling on a peripheral area of the retina. A subconscious stimulus is initiated in the sensory apparatus which mediates an innervation to the motor apparatus to produce the ocular movements which bring the two focal areas in alignment with the point of fixation. The character and degree of these movements depend on the location and the disparity of the areas stimulated. These movements of fixation are of such com-

mon occurrence that one hardly stops to consider the complex physiologic and psychologic acts involved

Hofmann and Bielschowsky investigated the nature of the involuntary movements of fusion. They found (1) that these movements could not be voluntarily started, accelerated or retarded but were produced only by unusual external conditions, (2) that the movements developed gradually and that the maximum deviation could be obtained only if the external condition continued to exist for a period of time, (3) that the absolute maximum limit obtained in a series of tests could not be increased to any great extent by long, continued practice but that this limit could be reached in a shorter time after practice, and (4) that after removal of the cause of the unusual innervation which produced the movements of fusion, the fusion innervation so incited did not disappear immediately, and this temporarily prevented the eyes from returning to their usual position of rest.

Dr. Burian has shown experimentally that the fusion movements resulting from the stimulation of peripheral area of slight disparity initiate movements of the eye which in general follow the foregoing fundamental laws.

DR. WALTER H. FINK, Minneapolis. Dr. Burian stated that his work adds to ophthalmic knowledge a new function of the peripheral portion of the retina. I do not believe that this is correct, because the fusion value of this portion of the retina has been recognized for some time. Lloyd recognized this fusion power and utilized it in his stereocampimeter when he designed charts to stabilize fixation by stimulating both foveal and peripheral fusion. The value of peripheral fusion has also been demonstrated in cases of bilateral macular defects, and its power has been employed in maintaining parallelism. It has been utilized also in stimulating binocular single vision. Through the stimulation of the peripheral fusion, binocular unity results and assists in breaking down macular suppression. In testing duccion power, better stability and uniformity of readings are obtained when the target used stimulates both the macular and the peripheral portion of the retina.

One must, in considering the relative power of foveal versus peripheral fusion, keep in mind certain aspects of the physiology of the retina. Certain factors are known to weaken macular fusion and give greater fusion power to the periphery. For example, a moving object from the side will exert an attraction on the central visual area which is imperative and sets in motion a reflex act. It is generally conceded that the macula does not function well when it is moving or when the object observed is moving. One's impressions depend on a constant movement of fixation. Also, foveal fixation is momentary, and if prolonged attention is forced, it fatigues easily, with a lowering of the fusion power. Macular fusion is weakened in dim light and a central scotoma is present, whereas the perimacular portion of the retina is most sensitive. The summation of peripheral stimuli has a weakening effect on foveal fusion. The rate of movement is an important factor in peripheral fusion, and a large object, if introduced slowly into peripheral vision, may remain unnoticed. It is therefore evident that the physiology of the retina is complex, and under certain conditions the impulse of peripheral fusion is greater than that of foveal fusion. It is, however, difficult to conceive that under every day conditions peripheral stimuli which are

introduced, eliminating motion as much as possible, will exert enough influence to disrupt macular stereoscopic vision

DR HERMANN BURIAN, Hanover, N H In answer to Dr Fink, I should like to make a few general remarks In making the quantitative experiments with the squares and the test lines, the central fusion was entirely eliminated I did not use anything other than test lines I have shown the mean deviations with which these experiments work, and the settings I have made myself in their accuracy, and I think that this accuracy is sufficient proof that the elimination of the central fixation has not done any harm to the experiments

POSTOPERATIVE COMPLICATION OF CATARACT OPERATIONS DR CONRAD BERENS and DR DONALD W BOGART, New York

On the basis of a study of the postoperative complications occurring in 1,004 cataract extractions performed at the New York Eye and Ear Infirmary during the last two years and a review of the incidence, cause, prevention and treatment of these complications, we believe that the percentage which result in blindness or impaired vision can be reduced Complete preoperative studies, combined with careful operative technic and postoperative treatment, will often prevent serious complications after the extraction of cataract

DISCUSSION

DR WATSON W GAILEY JR, Bloomington, Ill A close analysis of the postoperative complications described by the authors is interesting in that it shows that the percentage of loss of vision is practically the same with both intracapsular and extracapsular extraction

A poor incision in which the iris is involved or one which is made too deep, including the sclera, is certainly conducive to hyphemia It is not out of line to suspect that too much pressure in making fixation or too much pressure in dislocating the lens could be responsible for choroidal or retinal detachment or even an expulsive hemorrhage Closure of the wound with sutures, preferably corneoscleral, has been a great step forward in the prevention of postoperative prolapse

My personal experience has been that prolapse of the iris occurs more frequently in cases in which intracapsular extraction is done than in those in which capsulotomy is performed The majority of my prolapses have occurred on the fourth or fifth day The large ones are repaired by excision and are covered with a Kuhnt flap The small ones respond excellently to the application of trichloroacetic acid after the method of the late Harold Gifford It is wise to wait, if possible, until the eye is white when dealing with small prolapses

I have also found that hyphemia is encountered more frequently in cases of diabetes and in cases in which the iris is very rigid and in which it is difficult to dilate the pupil It has been my observation that hyphemia appears more frequently when a full iridectomy has been performed than when either peripheral iridectomy or iridotomy has been done Any efforts I have made to control the occurrence of hyphemia have been fruitless I am inclined to believe that retrobulbar injections of procaine hydrochloride combined with epinephrine hydrochloride may be responsible for the incidence of intraocular bleeding

DR JOSEPH L McCOOL, San Francisco Many of the postoperative complications which follow cataract extraction are the result of inadequate preparation of the patient from the psychic standpoint and of accidents which may occur during the operation

If the surgeon will take the time and trouble to explain something of the procedure to the patient and assure him that nothing he may do at the time of operation will mitigate against a successful result, he will be agreeably surprised to find the operative risk much better than he had anticipated To be sure, any one of the barbiturates given in adequate doses and long enough before the operation to obtain their maximum effect will add much to the success of the operation

For more than fifteen years I have used a deep orbital injection of a solution of procaine hydrochloride and epinephrine hydrochloride in every operation in which the eyeball is to be opened I use 1 cc of a 4 per cent solution of procaine hydrochloride and 1 drop of epinephrine hydrochloride in a dilution of 1 1,000 I have never had any reason to regret doing so, and on innumerable occasions have been thankful that I used it

I am willing to admit that this method does cause hypotony, but never marked nor enough to mitigate against the success of the extraction It will and frequently does cause a slight weakness of the inferior rectus muscle but as the eye is controlled by the suture of the superior rectus muscle, this weakness is of no importance I have never been able to convince myself that it was responsible for undue bleeding after operation On the other hand, its advantages are many and valuable The iris is so completely anesthetized that an accidental nicking of this membrane in making the incision causes no sensation or reaction on the part of the patient It follows, therefore, that the subsequent manipulations of this membrane are painless, and whether one does a peripheral or a full iridectomy, it can be accomplished painlessly and with deliberation If there is a moderately rigid sphincter and peripheral iridectomy is done, unless the iris is completely anesthetized there is some discomfort as the emerging lens slowly stretches the sphincter The subsequent toilet of the wound is facilitated by the adequate anesthesia and can be performed carefully and thoroughly, some of the complications incident on this part of the operation thereby being avoided

DR E C ELLETT, Memphis, Tenn Opinions seem to differ in regard to the bad effects of focal infections on the prognosis of an intraocular operation, but personally I am convinced of their importance, especially as regards the teeth Other important matters are complete anesthesia and akinesia For the past year I have been using retrobulbar injections with increasing satisfaction They overcome spasm of the rectus muscles better than any other method, but on account of interference with downward rotation a stitch under the superior rectus muscle is frequently necessary

The advantages of a sufficiently large corneal incision and a free capsulotomy have been emphasized, and I heartily agree with what was said about the advantages of a conjunctival flap and some form of suture The latter permits a safe and leisurely toilet of the wound, removal of lens matter, clots and bits of capsule and reposition of the iris and smoothing out of the conjunctival flaps Sutures will not prevent prolapse of the iris, but they will limit its extent and make the

excision of a prolapsed iris infinitely safer and easier than it would otherwise be. I think that the best time to deal with this complication is as soon as it is discovered. Delay only means more trouble. Small prolapses and ectasia of the wound are well met by the application of trichloroacetic acid, as Barkan has described. If akinesia makes closure of the lids difficult, they should be closed by a stitch or by fixing the lashes of the upper lid to the cheek with collodion, as suggested by Gill. The intracapsular operation is a most valuable contribution to cataract extraction, but it is not always advisable or possible.

VASCULAR OBLITERATION FOR VARIOUS TYPES OF KERATITIS ITS
SIGNIFICANCE REGARDING NUTRITION OF CORNEAL EPITHELIUM
DR TRYGVE GUNDERSEN, Boston

This article will be published in full, with discussion, in a later issue of the ARCHIVES

MIXED TUMORS OF THE LACRIMAL GLAND DR THEODORE E SANDERS, St Louis

This article will be published in full, with discussion, in a later issue of the ARCHIVES

VERNAL CONJUNCTIVITIS DR HAROLD F WHALMAN, LOS ANGELES

Vernal conjunctivitis is a chronic hyperplastic condition of the conjunctiva manifesting itself in many forms. The simplest is the smooth form in which the conjunctiva is slightly thickened and pale and the entire palpebral surface is involved. In some of these cases the bulbar conjunctiva adjacent to the fornix appears pale and thickened. The follicular form possesses all the characteristics of the simple form and, in addition, there are numerous small, round, discrete nodules. In the vegetative form the conjunctiva has the appearance of a greatly thickened, warty mass.

Bulbar involvement in my experience frequently has accompanied involvement of the lid. It has been observed that the bulbar conjunctiva may have the appearance of a smooth, milky thickening, follicles may appear on it, or actual vegetations may be found near the limbus.

From an etiologic standpoint there appears to be good evidence for a constitutional factor as well as for an external one, the former preparing fertile soil for the reception of the latter. Fifty per cent of all patients in my experience gave a personal history of allergy. Another 20 per cent gave an immediate family history of allergy.

The treatment of vernal conjunctivitis has been unsatisfactory as far as accomplishing a complete rehabilitation of the tissues to a normal state. Relief from symptoms can be obtained with a variety of solutions, and in my experience these tend to be on the acid side. Acetic acid, 0.25 per cent, gives relief in some cases. More effective than this is a phosphate buffer of p_H 6.6 instilled several times a day, as suggested by Hosford. Trichloroacetic acid painted on the everted and cocaineized lids reduces the size of the cobblestones and aids in the relief of symptoms. Solid carbon dioxide has a similar effect. Radium is effective, but one should hesitate to use radium in cases of involvement of the limbus.

in which the eyeball cannot be protected. Simple excision of the giant forms is rapid and effective. A useful procedure in the vegetative type is the utilization of an elliptic mucous membrane graft from the lip. Tests for specific allergins and subsequent desensitization have proved of little value.

DISCUSSION

DR M. N. BEIGELMAN, Los Angeles. As Dr. Whalman pointed out, vernal conjunctivitis is quite prevalent in southern California. I have at present on file records of 120 cases of this disease observed in a total of about 15,000 clinical and private patients, which constitutes an average of 8 to 1,000. This rather high incidence may be partly due to the fact that for a number of reasons I was interested in the milder forms and in the earlier stages of vernal catarrh. In this respect a great deal must be added to the classic conception of the disease. Textbooks, old and new, describe vernal catarrh in the familiar terms of cobblestone granulations on the tarsal conjunctiva and of gelatinous formations on the lids. These changes, as a matter of fact, are characteristic of the advanced, final stage of vernal conjunctivitis.

Dr. Whalman spoke about the allergic aspects of vernal catarrh, and particularly about the results of desensitization, in a rather reserved tone. I fully share his attitude. True, there are some well known facts, the eosinophilia, a positive allergic history, concomitant diseases of the upper part of the respiratory tract and cutaneous lesions, which link vernal catarrh with so-called allergic diseases. Among these allergic conditions, vernal catarrh, however, occupies seemingly a special place, first, because of its histologic picture and, secondly, because of its response, or rather lack of response, to desensitization.

I have compared sections of conjunctiva from more than 30 patients with vernal catarrh with sections of mucosa taken from the nose and the accessory nasal sinuses of allergic persons. The latter specimens were obtained through the courtesy of colleagues in the ear, nose and throat department of the Los Angeles County General Hospital.

In going over the slides, one cannot help being impressed with the fact that one is dealing with two fundamentally different types of tissue reaction. Whereas the mucosa of the upper part of the respiratory tract of allergic persons is almost always edematous, often to a considerable extent, one fails to find evidence of edema not only in specimens obtained from patients with advanced fibrosed vernal catarrh but in those obtained from patients with the disease in the earliest stages. I have sent, over a period of years, 11 patients with vernal catarrh to competent allergists for treatment. I am sorry to report 11 failures.

Certain analogies between vernal conjunctivitis and established allergic disease, as was stated before, cannot be denied. They should be considered, however, as a starting point for future investigation rather than as a closing answer to the complicated problem of vernal catarrh.

DR ALBERT N. LEMOINE, Kansas City, Mo. Early in my work on allergy I immunized about 15 patients with the pollens to which they were found to be hypersensitive. This immunization was done in the latter part of the winter before the time for the onset of the attacks, and all of the patients were much improved or completely relieved for the first year, but the second year there were varying degrees of recur-

ences, and for the third year most of the patients were found to be practically not benefited at all. This is quite in line with other artificial immunization, that is it is only temporary in a majority of the cases.

I was impressed with Dr. Whalman's advice to use a buffer solution slightly on the acid side. This has a scientific basis and should be beneficial. For the same reason I have used an ointment containing 10 per cent citric acid once or twice daily. When it is to be instilled once daily, it is preferable to use it at bedtime. When it is used twice daily, the second instillation should be given in the morning. I have found that it greatly benefits most of the patients, in fact, I have found that it relieves the itching and sticky, rubbery discharge and reduces the size of the vegetation more than any other form of treatment I have used. In my opinion, alkaline buffer solutions as suggested by some authors are contraindicated. In the past I have used an alkaline buffer solution with no noticeable results.

TREATMENT OF CANCER OF THE EYELIDS DR. GEORGE S. SHARP Pasadena, Calif.

The treatment of cancer of the eyelids is primarily one of irradiation. The lethal or cancerocidal dosage is calculated according to the histologic type and dimensions of the growth. The irradiation is delivered by an external source, such as a radium plaque, or by interstitial means, such as radium needles or radon seeds. Frequently the combination of external and interstitial irradiation may be adapted for the best permanent result. Forty-eight patients with cancer of the eyelids were treated from 1932 to 1937 inclusive and biopsy gave positive evidence of cancer in all instances. The follow-up study has included 92 per cent of the patients. Many have not yet lived five years after treatment, but all have lived more than one year, and there has been no evidence of recurrence or complicating deformity in the group.

DISCUSSION

DR. EVERETT L. GOAR, Houston, Texas. For many years it has been my custom to refer all patients in whom I suspected a malignant lesion of the eyelids to a dermatologist for treatment. I do not believe that treating these lesions surgically serves the best interest of the patient. It is true that these growths can be removed safely and surely by wide excision, by cautery or by fulguration, but the loss of tissue and subsequent scarring is undesirable. Many a patient has been subjected to a plastic operation on the eyelid that would not have needed it if the neoplasm had been treated by radium.

Having referred these patients for treatment, it was necessary for me to turn to the consultant, Dr. C. M. Griswold, for information, and he has furnished me with statistics of patients treated by him during a ten year period. His records show 208 patients with cancer of the eyelids, 136 of whom were males and 72 of whom were females. As to location, 100 of the growths were at the inner canthus, 24 were at the outer canthus, 68 were in or near the central area of the lower lid and 16 were in or near the central area of the upper lid. Five of the patients were under 30 years of age, but the vast majority were 50 or over. While biopsies were not done for every patient, Dr. Griswold

estimates that about 5 per cent of the growths were of the squamous cell type, while 95 per cent were of the basal cell or basal squamous cell type

All the patients were treated by topical applications of radium, with an average per patient of 550 milligram hours. There were 8 failures in the series. Most of the remainder of the patients have been followed for a varying period of from one to ten years, and their lesions are satisfactorily healed. Cicatricial ectropion has not occurred as a complication from the treatment, but in 5 patients in whom the lesion had perforated the lid and the conjunctiva was involved, symblepharon developed. No irradiation cataract has been noted in the series. All patients had a permanent loss of lashes on the affected lid.

Dr. Griswold has used the same technic throughout the series. A 2 mm. lead mold, which is wrapped in pure gum rubber to prevent secondary irradiation, is placed between the radium and the cornea. The radium plaque, encased in a 0.5 mm. platinum shield, is applied directly to the lesion. Three or four applications are made within a week. In the majority of cases in the early stages this has caused the lesion to disappear within six or seven weeks, leaving a thin, pliable scar that is scarcely visible.

DR. DOHRMANN K. PISCHEL, San Francisco. Many ophthalmologists have felt that when possible surgical excision of small epitheliomas was the simplest and quickest method for dealing with such tumors. If the growth is small, this method appears to me to be correct. If the surgical procedure is properly planned and executed, no deformity will result, and it is true that convalescence is short and comfortable. However, the small lesions that are amenable to surgical treatment are also most amenable to irradiation, and I personally feel that irradiation is the method of choice for such radiosensitive tumors, as Dr. Sharp has described.

Unfortunately, these cancers are most often seen when in a late stage, and here surgical treatment is impossible. It is therefore interesting to see the excellent results which can be achieved by radiation therapy. It must, of course, be emphasized that such excellent results are not to be obtained by careless work.

AN OPERATIVE PROCEDURE FOR GLAUCOMA OF SHALLOW CHAMBER TYPE. MULTIPLE EXCISIONS OF THE ROOT OF THE IRIS AND DEEPENING OF THE ANTERIOR CHAMBER. DR. OTTO BARKAN, San Francisco.

This article will appear in full in a later issue of the ARCHIVES.

SULFANILAMIDE TREATMENT OF TRACHOMA. PRELIMINARY REPORT. DR. FRED LOE, Rosebud, S. D.

In August 1937, 2 patients at the Rosebud Indian Hospital who had had trachoma, one for eighteen months and the other for two years, were selected for treatment with sulfanilamide. Both patients had been treated with grattage followed by the administration of silver nitrate intermittently during the period of their infection, with no noticeable improvement. On the basis of their body weight they were given $\frac{1}{3}$

gram (0.02 Gm) of sulfanilamide, with an equal amount of sodium bicarbonate, per pound of body weight daily for ten days. Then the dose was decreased to $\frac{1}{4}$ grain (0.016 Gm) of sulfanilamide a pound daily for fourteen days. No other medication was allowed during this period. Within five days after treatment was begun changes in the conjunctiva were observed, the redness gradually disappeared, the granules and papules decreased in size and the blood vessels became increasingly visible. With a maintenance dose of $\frac{1}{4}$ grain of sulfanilamide a pound daily, the two patients were apparently cured of trachoma within one month.

After the aforementioned dose of sulfanilamide was used in the treatment of 140 patients with trachoma, the following conclusions were made as to improvement:

Improvement of subjective symptoms consisted of cessation of lacrimation within twenty-four hours, loss of photophobia within twenty-four hours and improvement of vision within seventy-two hours in cases of pannus. Improvement of objective symptoms included paling of the conjunctiva and paling of the trachomatous patches and flattening of the granules and follicles. In the cases in which there had been no scarring from instrumentation, the conjunctiva apparently resumed its normal velvety texture at the end of two months. The blood vessels of the conjunctiva became more visible on the fifth or sixth day of treatment, and daily thereafter they became more normal. In 30 cases of pannus in which treatment was given in the hospital it was noted that the opacity began clearing between the eighth and the fifteenth day, depending on the density of the pannus, with great improvement of vision. The granules on the lower lids were found to be the last objective symptom to disappear.

DISCUSSION

DR HARRY S. GRADLE, Chicago. Stimulated by the original report of Dr. Loe to the Department of the Interior on the effects of sulfanilamide on trachoma among the Indians, I started treating a series of patients with the drug. Twenty-five persons were selected from the patients in the Trachoma Clinics of Southern Illinois, where all are outpatients, and 16 from those of the Illinois Eye and Ear Infirmary, all of whom remained in the hospital. The types of trachoma were distributed as follows:

Stage	Trachoma Clinics	Infirmary	Total
I	1	0	1
IIa	8	1	9
IIb	6	2	8
IIIa	6	7	13
IIIb	4	1	5
IV	0	5	5
	<hr/> 25	<hr/> 16	<hr/> 41

Of this group of patients, 5 were forced to discontinue the drug within a few days because of systemic symptoms that ranged from mere headache and nausea to actual cyanosis, necessitating digitalis. The patients at the Illinois Eye and Ear Infirmary were followed by laboratory study daily, with the following findings: (a) Bacteriologically, organisms were found in only a few cases. (b) The hemoglobin

showed a fairly uniform decrease of from 10 to 15 per cent (c) The red cell count showed no change except for a moderate decrease in isolated cases (d) The white cell count showed a fairly uniform decrease of from 1,000 to 3,000 cells

Summarizing the impressions gained from three weeks' observation of these patients, who did not receive any local treatment except a cleansing wash with physiologic solution of sodium chloride, the following seemed to be the salient features

1 Those with trachoma of stages II and III responded fairly uniformly in a most surprising fashion in that the velvety patches and hypertrophy disappeared rapidly The thickened and hyperemic conjunctiva became thinner and pale, the individual vessels became visible, and the secretion disappeared in the majority of instances The results of three weeks of treatment with sulfanilamide approximated those with from three to six months of local treatment, although there was no scar formation

2 Regardless of the stage of trachoma, photophobia and lacrimation disappeared rapidly This subjective improvement was most striking and, to the patients, most gratifying

3 Persons with trachoma of stages IIb, IIIa and IIIb showed marked improvement in vision when the vision was decreased due to pannus In cases in which the trachoma was of longer duration and of the malignant type or in which there were corneal scars due to old ulcers the amount of visual improvement was negligible In some instances the visual results were almost miraculous

4 Persons with trachoma of stage IV and of stage IIIb in the malignant form showed practically no improvement

So, taken by and large, it would appear that in the use of sulfanilamide one has a new means of combating trachoma in the acuter stages The drug is not without danger and should be taken only when medical observation is possible every twenty-four to forty-eight hours Apparently children and persons of the older, plethoric type do not tolerate it as well as others How efficacious it is in stage I, I have no means of knowing, nor is it known yet how lasting are the improvements noted In a year from now Dr Hirschfelder, Dr Ackerman and I will report on these same patients, who will have no local treatment in the meantime, unless an exacerbation should occur

At this point it seems most appropriate to give all credit and priority to Dr Loe, who conceived the idea, and to the Indian Medical Service, which made it possible for him to try it out on a large scale

News and Notes

SOCIETY NEWS

Treacher Collins Prize—Under the title Treacher Collins Prize the council of the Ophthalmological Society of the United Kingdom has instituted a prize of £100 to be awarded triennially for the best essay submitted on a subject selected by the council

The prize is open to qualified medical practitioners of any nationality, but the essay must be written in the English language. The subject for the first award of the prize is "Cerebrospinal Disease and Its Relation to the Optic Nerve"

The closing date for sending in essays for the first award is Dec 31, 1938. They should be submitted to the honorary secretary, Ophthalmological Society of the United Kingdom, 5, Racquet Court Fleet Street, E C 4, from whom also any further particulars can be obtained. No name should be on any essay, but a distinguishing pseudonym or quotation, which should also be on a sealed envelop containing the candidate's name and address. This envelop should accompany the essay.

Société d'ophtalmologie de Paris—The annual meeting of the Société d'ophtalmologie de Paris will be held on Nov 20, 1938. The following addresses have been announced:

Dr Lhermitte will speak on the pathology and pathogenesis of softening of the brain.

Dr Magitot will speak on the society, from its foundation to the present day.

The principal report will be delivered by Dr Dubois-Poulsen, on so-called hypertensive retinitis.

Formation of the American Orthoptic Council—On Oct 11, 1938, a group of ophthalmologists met in Washington, D C, and organized the American Orthoptic Council.

Three members had been appointed for this purpose by each of the three national ophthalmologic societies as follows: for the American Ophthalmological Society, Dr John Dunnington, Dr Alfred Cowan and Dr Edwin Dunphy, for the Section on Ophthalmology of the American Medical Association, Dr Derrick Vail, Dr Everett Goan and Dr Harold Gifford, for the American Academy of Ophthalmology and Otolaryngology Dr LeGrand Hardy, Dr Grady Clay and Dr Avery Piangen.

The following officers were elected: president, Dr LeGrand Hardy, vice president Dr Derrick Vail, and secretary-treasurer Dr Edwin Dunphy.

It was voted that this council be known as the American Orthoptic Council and that its function shall be the regulation of the principles and practice of orthoptic training. The following resolutions were adopted: 1. Orthoptics has a definite place in ophthalmology. 2.

Ophthalmologists should know more about this subject 3 Ophthalmologists in general are unable to carry out this work themselves, and therefore technicians are needed 4 The national societies through the American Orthoptic Council should take over the training of technicians, and a central station should be established for training of such technicians

By-laws were adopted which provide that the council shall be composed of nine executive members, three from each national society, and three associate members, who shall be orthoptic technicians or instructors, to be selected by the nine executive members It was also provided that examination shall be held at least once a year and that a certificate of proficiency shall be awarded to those candidates who have satisfied the board of examiners A set of rules governing the training and practice of orthoptic technicians and a code of ethics governing deportment of such technicians were also adopted

A syllabus for student orthoptic technicians will be issued shortly

Obituaries

JOHN MARTIN WHEELER, M D
1879-1938

John Martin Wheeler, son of Henry Orson Wheeler and Elizabeth Lavina (Martin) Wheeler, was born on Nov 10, 1879, at Burlington, Vt. He was a descendant of Thomas Wheeler, of Cranefield, Bedfordshire, England, who came to America with his wife, Anne Halsey, and settled at Concord, Mass. His father, a lawyer, was superintendent of schools at Burlington for thirty-three years. His early education was obtained at the public schools in Burlington, after which he entered the University of Vermont, receiving the degrees of Bachelor of Arts in 1902, Doctor of Medicine in 1905 and Master of Science in 1906. He served his alma mater as instructor in anatomy in 1906 and 1907, during which time his first scientific contributions were published, on "The Viability of Typhosus Bacillus Under Various Conditions."

Becoming interested in ophthalmology, he secured an internship at the New York Eye and Ear Infirmary, from which he graduated in 1908. He began the practice of his specialty in New York in 1909, in association with Dr. D. W. Hunter, a connection he maintained until Dr. Hunter's retirement in 1917. He was actively identified with the New York Eye and Ear Infirmary for many years. He served that institution with true devotion in all capacities. He was first an assistant surgeon and then senior assistant surgeon in the clinic of Dr. John E. Weeks until 1919, when he was appointed an attending surgeon. He was a member of its board of directors and a consulting surgeon at the time of his death. In addition to this active connection, he was associated with the Bellevue Hospital and the Fifth Avenue Hospital for many years. He was visiting surgeon in charge of the ophthalmic service at the Bellevue Hospital from 1925 until 1928, when he resigned to accept the directorship of the ophthalmic service of the Presbyterian Hospital. At the time of his death he was director of the Institute of Ophthalmology, Presbyterian Hospital, and consultant ophthalmologist to the following hospitals: The Bellevue Hospital, the New York Eye and Ear Infirmary, the Fifth Avenue Hospital, the New York Post-Graduate Medical School and Hospital, the Neurological Institute of New York, the Sloane Hospital for Women, the Babies Hospital, the Psychiatric Institute, St. Luke's Hospital, of Newburgh, N. Y., and the Hackensack (N. J.) Hospital.

The knowledge he gained from this wide clinical experience enabled him to speak authoritatively on all phases of ophthalmology. His inquisitive mind spurred him on to learn everything possible from every case. He was a keen diagnostician, whose sound judgment and practical suggestions made him a most helpful consultant.

As an undergraduate teacher he was an instructor in ophthalmology at Cornell University Medical College from 1911 to 1915 and professor



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of ophthalmology at New York University from 1921 to 1928, at which time he resigned to accept a similar professorship at Columbia University. While he was greatly interested in seeing that his medical students acquired a sound practical knowledge of ophthalmology, his real interest lay in postgraduate instruction. After many years of postgraduate teaching at the New York Eye and Ear Infirmary, where his courses on surgery attracted students from far afield, the ideal of his life was realized in the opening of the Institute of Ophthalmology

in 1932, with him as its director. Although the acceptance of this new position necessitated the giving up of a large private practice at great personal sacrifice, he gladly did it because of his love for postgraduate teaching. He then began to devote the greater part of his time to the training of his residents and staff. His tireless energy, his wise and sympathetic counsel and his genuine sincerity made him an ideal leader. His quiet unassuming manner endeared him to all. His rounds and seminars were occasions never to be forgotten by his associates.

He entered the medical corps of the army during the World War as a captain, with an assignment to the air medical service. After a comparatively short time he was transferred to Fort McHenry, Md., where he was placed in charge of the ophthalmic service. This was an active hospital post filled with disabled veterans who required all sorts of reconstructive surgical treatment. Here was his long-looked-for opportunity to learn all he could about plastic surgery, so with great enthusiasm he began this new work. His thorough knowledge of anatomy and sound surgical judgment combined with his ingenuity and diligent work soon won for him a national reputation as a plastic surgeon. He loved the intricacies of a difficult plastic problem and derived great satisfaction from its successful completion. He reported this army experience before many medical meetings and with great modesty received the acclaim of the ophthalmologic world.

He was not a prolific writer, contributing only about 50 articles to the ophthalmic literature, but whatever he wrote was well done. He had no use for mere words, so his articles were always brief and to the point, at the same time being clear and explicit. His writings were largely on surgical procedures. In 1915 he wrote the chapter on "Eye Surgery" in Johnson's "Operative Therapeutics." His thesis for the American Ophthalmological Society, in 1916, was entitled "A Study of Hemorrhage into the Anterior Chamber Subsequent to Operations for Hard Cataract." He has written a number of well known articles on plastic surgery.¹

In addition to these plastic procedures, he reported on an opening for secondary cataract by a single straight incision and iridotomy by

¹ Wheeler, J. M. Free Dermic Grafts for the Correction of Cicatricial Ectropion, *Am J Ophth* **3** 251-255, 1920, War Injuries of the Eyelids. Plastic Operations For a Few Types, *Arch Ophth* **49** 35-42, 1920, Restoration of the Margin and Neighboring Portion of the Eyelid by a Free Graft from the Lower part of the Eyebrow and the Skin Directly Below It. Report of an Illustrative Case, *J A M A* **75** 1055-1057 (Oct 16) 1920, Restoration of the Obliterated Eye Socket, *Am J Ophth* **4** 481-488, 1921, Correction of Cicatricial Ectropion by Use of True Skin of the Upper Lid, *J A M A* **77** 1628-1631 (Nov 19) 1921, The Use of the Epidermic Graft in Plastic Eye Surgery, *Internat Clin* **32** 359-370, 1922.

the same method, at which time he described a useful dissection knife which bears his name. He then became interested in exophthalmos and published several excellent articles on this subject. As his presidential address before the American Academy of Ophthalmology and Otolaryngology in 1934, he outlined the duties of the ophthalmologist toward the blind in a sane article entitled "Blind People." In the same year he reported before the American Ophthalmological Society an ingenious method of advancement of the superior oblique and inferior oblique ocular muscles. His later writings were on new operative procedures for the correction of spastic entropion and ptosis, and his last article was entitled "The Use of the Orbicularis Palpebrarum Muscle in Surgery of the Eyelids."

He was actively connected with many medical societies, being a member of the American Medical Association, the American College of Surgeons, the American Ophthalmological Society, the American Academy of Ophthalmology and Otolaryngology (past president), the Society of Plastic and Reconstructive Surgery (past president), the Medical Society of the County of New York, the Medical Society of the State of New York, the New York Academy of Medicine and the New York Ophthalmological Society. Although a regular attendant at the meetings of most of these organizations, he often had little to say, for with characteristic shyness he used to remark "I came to learn, not to talk." For many years he was a member of the editorial staff of the *American Journal of Ophthalmology*. He was also one of the founders of the newly created American Board of Plastic Surgery.

He was twice the recipient of an honorary degree of Doctor of Science, the first degree being conferred on him by the University of Vermont in 1928, while Middlebury College paid him a similar tribute in 1933. In 1931 he was decorated Commander of the Order of the Crown of Siam in recognition of the services he had rendered its ruler. In 1936 he received the Leslie Dana Medal for "his outstanding achievements in the prevention of blindness and the conservation of vision." He served seven years as a member of the directorate of the National Society for the Prevention of Blindness.

In 1912 he married Julia Warren Smith, of Burlington, Vt., who survives him along with their four children, Martha, Charles, Edward and Ann. Dr. Wheeler was a devoted husband and father, spending most of his spare time with his family. He shunned society and loved the peace and quiet of home life. His family and his work were his great interests, and he found little time for golf, art and music—his hobbies.

As a man, he was quiet and unassuming, carrying his great honors with extreme modesty. In his work, he was painstaking and thorough.

His perseverance and determination were never better shown than after the loss of his left eye from a sarcoma of the choroid in 1935, he continued to operate with his usual skill and dexterity. To him this catastrophe was a challenge, which he bravely accepted and conquered. This incident best bespeaks the character of John Martin Wheeler, who died at his summer home in Underhill Center, Vt., on Aug. 22, 1938. In his death ophthalmology has lost one of its truly great men, for he was not only a highly successful practitioner and dextrous surgeon but a true friend, an inspiring leader, a great teacher and a sympathetic and wise counselor.

JOHN H. DUNNINGTON, M.D.

In the spring of 1907, Dr. John Wheeler, then a teacher in the department of anatomy of the Medical School of the University of Vermont, came to the New York Eye and Ear Infirmary, with an introduction from the executive surgeon, the late Dr. Richard Derby, and it was my duty as resident in ophthalmology to entertain him. From that time an intimate friendship was formed. Dr. Wheeler became a member of the house staff in 1908, as an intern he was conspicuous for his diligence, and even then his dexterity as an operator attracted attention.

As most men are largely guided by their preceptors I have always felt that the meticulous care in plastic operations shown by the late Dr. Dwight Hunter and the exquisite operative skill of Dr. John E. Weeks, both of whom were then surgeons at the Eye and Ear Infirmary, must have had a profound influence on Dr. Wheeler, for it was in ophthalmic surgery that he particularly excelled.

In addition to the professional attainments of Dr. Wheeler, which are so well known to the world of ophthalmology, the personal side of this man was impressive. He endeared himself, both to doctor and to patient, by his calm, quiet and unobtrusive manner. He shunned publicity and disliked the notoriety of the press which so often comes to men of prominence. His interest in the young doctor was never failing, and some day these men will proudly say that their success was due to his material assistance and guidance.

As head of the Institute of Ophthalmology of Columbia University, it was not only his demand that his associates should be capable and that the institution should function as an efficient place of learning but also his great ambition that there should ever be a spirit of sympathy, kindness and friendliness. And such was the life and character of its chief. He was a practical Christian, charitable and constantly doing good.

After his tragic operation three years ago, his determination and will to adjust himself quickly were a marvelous display of courage, and to his intimates there seemed, as one expressed it, "the rebirth of a man"

The passing of John Martin Wheeler ended the beautiful life of "A Great Physician"

While it is a gigantic undertaking, it is hoped that the staff of the Institute of Ophthalmology will complete the textbook on ophthalmic surgery which he had begun, as a contribution to his memory

F PHINIZY CALHOUN M D

Dr. Wheeler's birthplace, Burlington, Vt., is one of the most beautiful of small cities and the site of the state university. His father was much like him, a modest, unassuming man who came home from the Civil War to raise and educate a family on the limited income of a country lawyer. This he supplemented for over thirty years by the nominal salaries of superintendent of public schools and treasurer of the university.

Dr. Wheeler was educated in these schools and afterward received both his academic and his medical degrees at the university. By all present standards it was a very inadequate education, though there was nothing better to be had at the time. The university was a small one, with little endowment and less material equipment, but it had on its faculty a few men who were really great personalities and inspiring teachers. It offered the additional advantage of being almost unbelievably inexpensive.

His boyhood environment was one of the great factors in Dr. Wheeler's life. Here he acquired his salty humor and his eagerly collected fund of Vermont stories. Here he returned to marry his boyhood playmate, who became not only his devoted wife and the mother of his children but the confidant who shared all his hopes and plans. Here he returned year after year for rest and relaxation. Here he built the beautiful log camp in the mountains in which during many summers he kept open house for his friends, and here he returned, unknowingly, to die.

Dr. Wheeler had the temperament of the great surgeon, the willingness to assume responsibility, the ability to rise to emergencies and, when he had done his best, the ability to banish worry.

As the older men retired, his reputation grew, not because he was pushful or aggressive but because of his universally conceded ability.

He loved teaching and the training of assistants and students, and his operative clinics were always crowded. He was a firm disciplinarian,

but for the men who did things just as he wanted them done he spared neither time nor trouble in the training, while his interest and assistance followed them long after they had become established in other fields.

He had the unique ability of inspiring subordinates and building up an esprit de corps among them, of assigning to each the tasks appropriate to his ability, of encouraging them to do original work, often work he had hoped some time to do himself. He naturally could select his subordinates from the whole country, for to have been trained by Dr. Wheeler was almost a guarantee of success in any community.

No man ever more enjoyed the affection of his associates and his subordinates. No man ever inspired less jealousy and ill will.

He was, of course, a great surgeon, for he not only did superlatively the operations that his predecessors had devised but originated many new operations or made improvements on the old. In the World War he was assigned the care of the mass of unfortunates who returned to this country crippled or disfigured by wounds and hasty operations. He developed out of this supposedly hopeless task a marvelous technique of plastic surgery, which seems likely to put him among the ophthalmologic immortals.

As he grew older and his reputation increased, more and more of the desperate cases were referred to him for a final judgment, when the chances of failure even at his hands were often far greater than the chances of success.

He was an ideal consultant, always helpful and understanding and willing to assume responsibility. He often went far beyond the bare requirements of medical ethics in his efforts to avoid criticism or to save the face of a colleague. His thoughtfulness and kindness for his patients whether rich or poor were proverbial.

He was for many years an active supporter of the National Society for the Prevention of Blindness, and in 1931 became one of the organization's directors and one of the most useful members of its board.

In 1936 he was awarded the Leslie Dana Medal, bestowed each year on some layman or physician for conspicuous service in the prevention of blindness, his medal being fittingly inscribed, "Skilled surgeon, great teacher, understanding and sympathetic physician and friend."

Notwithstanding the catastrophe which befell him a few years ago, with steadfast courage he convinced himself and his associates that he could still operate up to his own high standards and kept up his work, both executive and surgical, to the very end. His last years were in many respects his greatest ones.

ELLICE M. ALGER, M.D.

Book Reviews

An Introduction to the Mathematics of Ophthalmic Optics By Paul Boeder, Ph D Price, \$2 50 Pp 244, including index Rutland, Vt Tuttle Company, Inc , 1937

This little book is so easy to read that it must have been quite difficult to write

Its greatest asset is the extreme simplicity and lucidity with which the subject matter is presented Even the reader who has forgotten all of his high school mathematics may nevertheless rely on Boeder to carry him through to an elementary understanding of the theory of Gauss

The introduction consists of a brief discussion of measurement and numbers This is followed in part I by a brief review of the arithmetic of fractions and a discussion of negative numbers

Part II deals with elementary algebra The diopter is defined Expressions for the dioptric power of a surface and that of a thick lens are introduced The author's discussion of the information to be derived from manipulation of the formula

$$D_e = \frac{D_1}{1-dD_1} + D_2$$

is particularly effective in demonstrating the power of the mathematical attack

The derivation of the formulas for the conjugate distances and magnification of a two surface system is clear and explicit The formulas are developed with the vertices, focal points and principal points as reference points The optical system formed by the combination of a reduced eye and a correcting lens is discussed

Part III treats of geometry and trigonometry Among other topics discussed are the geometric method of image construction, the lens gage, the entrance and exit pupils of the eye, Snell's laws and reflection by plane and spherical mirrors

There is a compact index to formulas in addition to the usual index

The illustrations are numerous, simple and effective Almost all the examples and applications deal specifically with ophthalmic optics Answers to the examples are supplied

The book is admirably suited to the needs of the unaided reader An adverse criticism which might be made is that after the discussion of the algebraic method in part II, the geometry and trigonometry of part III, although adequately treated, seem somewhat of a regression When the author has gone so far, one wishes that he would go further A development of the matrix evaluation of the Gauss coefficients for a three surface system might be substituted for part III This would considerably enlarge the scope of the book, permitting, for example, the introduction of the schematic eye and a discussion of telescopic spectacles Boeder's unusual expository ability should make even these relatively difficult subjects clear and thus bring the book to a fitting climax

ELEK LUDVIGH

Neuro-Ophthalmology. By R Lindsay Rea, London Price, \$9
Pp 568, with illustrations St Louis C V Mosby Company
1938

This book is dedicated to the memory of the late Prof Johnson Symington, who occupied the chair of anatomy in Queen's University at Belfast, Ireland

It is intended for ophthalmologists working in neurologic hospitals and for students in neurology and to facilitate the cooperation of the ophthalmologist and the neurologist in the study of their connecting subjects

After a short introduction on the functional examination of the eye required in the study of diseases of the nervous system, the anatomy and disturbances of the pupil and the muscles and nerves of the eye are carefully described, then follow descriptions of lesions of the optic nerve, such as papilledema, atrophy of the optic nerve, lesions of visual tracts, with the localizing signs as brought out by the study of the visual fields, and lesions of the macula The localizing value of ocular symptoms in diseases of the brain forms an important chapter Congenital and degenerative abnormalities, subarachnoid hemorrhage, tumors of the optic nerve, the region of the optic chiasm and pituitary body, ocular manifestations in diseases of the nervous system, diseases of the vegetative nervous system, ocular manifestations of head injuries, poisons which affect vision, headache and amaurosis are the subjects treated in the remaining chapters

This shows that the author has assembled a vast amount of information which is clearly and logically presented and well illustrated There are a few points on which some difference of opinion is permissible, but none of them are of sufficient importance to need comment Dr Rea is to be commended for the admirable manner in which he has presented a difficult subject There has been a definite need for such a presentation, as there has been no recent book on the neurology of the eye in English The many advances which have taken place in neurology have been fully noted, and Rea's "Neuro-Ophthalmology" is up-to-date and will be a welcome addition to the library of every ophthalmologist

ARNOLD KNAPP

Eingriffe am Auge. By Prof L von Blaskovics and Prof A Kreikei
Price, 20 25 marks Pp 454, with 648 illustrations Stuttgart
Ferdinand Enke, 1938

It is fortunate that a German translation of Blaskovics' textbook is now available The original work was published in Hungarian about one year ago and was reviewed in the March 1937 issue of the ARCHIVES, page 567 The German edition has been written by Prof A Kreikei, a former assistant of Professor Blaskovics and now his successor as director of the University Eye Clinic in Debrecen, Hungary Some additions have been made, and the important subject of the operative treatment of detachment of the retina has been enlarged and rewritten Blaskovics' outstanding contribution to ophthalmology has been the surgical correction of lesions and deformities of the eyelids It is natural, therefore, that the chapter on the eyelids takes up about one half

of the volume and contains a description of many of the operations which have made the author famous. Blaskovics has been a pioneer in developing new methods in this field and from the first established himself as a master in plastic surgery.

The study of these operative procedures will be of the greatest interest and value to every ophthalmologist who does surgical work. An outstanding feature of the book is the excellent illustrations, and their value, particularly in describing the different steps in an operation, cannot be exaggerated. The book making, both for clarity of text and illustrations, deserves praise, and the price is most reasonable.

ARNOLD KNAPP

Fundus oculi Diagnostica oftalmoscopica By Prof. Q. Di MARZIO, Bologna. L. Salomone, Rome. 1937.

The guiding principle of this atlas, dedicated to Benito Mussolini, is to demonstrate the usefulness of the ophthalmoscope in general diagnosis. It contains 100 large plates with 212 drawings which have been reproduced from original oil paintings by the modern method of offset calcolithography. The pictures are reproduced in the upright image as seen by the ophthalmoscope of Gullstrand. The courses of many of the conditions are presented, so that a comprehensive picture is given. A brief description of the fundus lesion and a brief history, including the results of roentgen examination and examination of the visual fields, which explain the relation to general disease, accompany each drawing.

The subject matter is arranged in the following order: normal eyegrounds, congenital anomalies and diseases of the optic nerve, of the retina and of the choroid. Among the illustrations, those which are outstanding and unusual are those depicting tumor at the head of the optic nerve, serous meningitis, cystic opticochiasmic arachnoiditis, hemorrhagic retinitis in chronic myeloid leukemia, tuberculous retinal periphlebitis, preretinal cysticercus, many examples of detachments with holes before and after operation and traumatic peripapillary rupture of the hyaloid membrane.

The author is to be complimented on the publication of a most instructive atlas. The reproductions in colors are lifelike and excellent, reflecting great credit on the artist and on the publishers.

ARNOLD KNAPP

Les médications de choc en ophtalmologie By L. HAMBRESIN. Pp. 252, with 11 illustrations. Paris: Masson & Cie, 1937.

This volume is the tenth annual monograph of the French Ophthalmological Society.

By the expression *choc thérapeutique*, the French mean any induced upset in physiologic equilibrium which begins and ends abruptly, leaving behind it no lasting anatomic lesion. English-speaking ophthalmologists are prone to speak of the agent used—protein therapy, hemotherapy, etc.—ignoring the manner in which the upset is produced. When this slight difference in conception is understood, the scope and value of this monograph become apparent.

The symptoms of therapeutic shock depend on the agent employed, the dose and the site and method of employment. In most cases injection of the foreign substance is followed by a chill, arterial hypotension and slow pulse. Later the temperature rises sharply, the pulse becomes more rapid and the blood pressure increases. All degrees of these basic phenomena may be encountered.

In most patients therapeutic shock may be induced with safety, but the method should not be used in the presence of tuberculosis, diabetes or cardiac disease. Great care should be exercised in applying the treatment to those persons in whom the presence of asthma or urticaria indicates an allergic diathesis.

The agents injected to produce therapeutic shock are the peptones, milk, charcoal, the various therapeutic serums and vaccines (both specific and nonspecific) and the colloidal forms of gold, silver and sulfur. Turpentine is used to form a local abscess. Whole blood may be used (autohemotherapy) or serum only (auto-serotherapy). In all cases the effect is heightened by the appearance of fever, which may also be produced by the induction of malaria and by the Kettering hyperthermia.

Many diseases are listed as being benefited by therapeutic shock. To the well known inflammatory lesions commonly accepted as being helped by the procedure, the author adds ocular pemphigus, glaucoma, muscular paralysis and ophthalmic migraine.

If one shock-producing agent is ineffective, another may be tried. The method should be used as an adjuvant to the older forms of treatment and not as the sole therapeutic procedure. The author believes that whole boiled milk is the agent that will be preferred by most ophthalmologists.

G. M. BRUCE

Les méningiomes supra-sellaires. Diagnostic du syndrome chiasmatique. By Dr Louis Guillaumat, Paris. Price, 30 francs. Pp 205. Paris: Gaston Doin & Cie, 1938.

This monograph begins with a description of the normal anatomy of the suprasellar region, the clinical symptoms and roentgen findings in cases of suprasellar meningioma, based on 22 cases observed in the neurosurgical service of Clovis Vincent in the Hôpital de la Pitié, are then fully described. Certain clinical groups are identified, and their diagnosis and treatment are carefully considered. Suprasellar meningiomas arise from the tuberculum sellae and from the jugum sphenoidale. Those arising from the tuberculum sellae are small and must be carefully dissected between the clinoid processes and the optic nerves, those arising from the jugum sphenoidale are larger and may grow to occupy the interhemispheric space, raising and separating the frontal lobes. The former raise the chiasm and cause bitemporal hemianopia. The tumors which arise more anteriorly have time to reach a certain size before making any contact with the optic nerves, and the unilateral preponderance of their development is shown by nonsymmetric changes in the visual field, loss of vision on one side with a temporal defect on the other or a central scotoma caused by intracranial compression on an optic nerve. A transitional form resembles a tumor of the olfactory groove and a tumor arising from the lesser sphenoid wing. In the

growth of the tumor, functional disturbances are produced from compression of the third ventricle and of the frontal lobes. The most noticeable symptom of pressure on the neighboring organs is involvement of the optic nerves and secondly of the optic chiasm. The ocular symptoms consist in atrophy of the optic nerve with occasional slight edema which clouds the margin of the disk. This atrophy may be bilateral, as in 13 cases in the series of 22, or unilateral, as in 6 cases, in 3 of which the Foster Kennedy syndrome was present. Bilateral papilledema was present in 2 cases, and the fundus was normal in 1. As for the defects in the visual field, typical bitemporal defects are not the rule, and the lesions are irregular. Of 19 cases, the defect was bitemporal in 16, homonymous in 1, horizontal in 1 and concentric with scotoma in 1. The signs of intracranial hypertension, the participation of neighboring structures and the roentgen findings are considered in turn.

The diagnosis of suprasellar meningioma usually rests on the defect of the visual field, the roentgenographic picture and the course of the disease. An excellent chapter is devoted to differential diagnosis. The diagnosis of suprasellar meningioma is often difficult. Thus, in these 22 cases the clinical diagnosis was made only in 10. In 6 an adenoma was suspected, in 2, opticochiasmic arachnoiditis, in 3, a meningioma of the lesser wing of the sphenoid bone, and in 1, meningioma of the right frontal area.

As to the prognosis in the 22 cases, 8 of the patients died after the operation, 3 could not be followed, 1 recovered some sight and the vision of the rest was improved. The only treatment is operation. The case histories are fully reported.

In conclusion, the author states that the study of suprasellar meningiomas is only a chapter in the etiology of chiasmic compression, it suggests the important role that the vessels of the circle of Willis play in causing hemianopias and the necessity of considering the surgical treatment of certain types of atrophy of the optic nerve.

Dr. Guillaumat has written a most scholarly and instructive treatise, which can be recommended for careful study by the ophthalmologist and the neurologist.

ARNOLD KNAPP

CORRECTION

A typographic error occurred in the review of "A Child's Graded Reading Book for Eye Specialists" by Dr. Henry R. Nesburn and Mr. Daniel L. Risley, in the October issue (*ARCH. OPHTH.* 20:697, 1938). The price was published as \$15.00 instead of \$1.50.

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 Monday of each month from October to May

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 p m, third Monday of each month except June, July and August

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 September to May

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 8 p m, second Thursday of each month from September to June

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 from October to June

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 Place Omaha Club, 20th and Douglas Sts, Omaha Time 6 p m, dinner,
 7 p m, program, third Wednesday of each month from October to May

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ARCHIVES OF OPHTHALMOLOGY

VOLUME 20 (old series Volume 77) DECEMBER 1938

NUMBER 6

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ANISEIKONIA

WALTER B LANCASTER, MD

BOSTON

Aniseikonia is a term I coined for a difference in size of the optical images of the two eyes. By the optical image is meant not the retinal image but that which reaches consciousness as a perception. This definition leaves unanswered the question of where the cause of the difference is to be found, whether in the refraction mechanism or in histologic differences in the sensory elements of the retina or in the structures still higher up.

CONSEQUENCES

Aniseikonia throws a burden on what I am fond of calling "the neuromuscular mechanism for binocular vision." In some way the eyes must compensate for the aniseikonia, and in ordinary cases they do, so that binocular vision goes on satisfactorily. However, the amplitude of adjustment in compensation for aniseikonia is limited. Compare this amplitude of adjustment with other amplitudes of adjustment, viz., the amplitude of accommodation which compensates for certain common errors of refraction or the amplitude of fusion which compensates for heterophoria.

Whenever any one of these mechanisms of compensation shows signs of being overtaxed, symptoms occur. The symptoms which arise from decompensation in the mechanisms of ocular adjustment are singularly similar. It is not usually easy to tell from the symptoms, because they are the general symptoms of eyestrain, whether the accommodation or the fusion faculty or the unknown mechanism which cares for aniseikonia is being overtaxed. The fact that this mechanism is quite unknown at present should not be regarded as cause for reproach, since even after many years there is a serious lack of agreement as to the mechanism of accommodation. This does not prevent one from knowing much that is important and invaluable in practice about

Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 9, 1938.

The discussion on this paper, which was combined with that on a paper by Dr. Conrad Berens, appears in the transactions of this society in this issue of the ARCHIVES, page 1109.

accommodation and how to deal with it. Considerable is known about aniseikonia considering the short time it has been under investigation and the limits in the number and resources of the workers whose leader was not an ophthalmologist, not even an optometrist, but an artist before he became a physiologist of the first rank by concentrating on these visual problems.

CAUSES

The most obvious cause of aniseikonia is anisometropia and the wearing of glasses of different magnifying power for the two eyes. An everyday problem in the practice of the ophthalmologist is how to deal with anisometropia. Small differences are easily managed by most patients, but few can take care of large differences.

While the fathers of ophthalmology knew about the troubles caused by anisometropia, they were not agreed as to the explanation. Donders and many others thought the unequal images important. Hess and others thought that the prismatic effect of looking through the periphery of the lenses was sufficient to tax the fusion faculty.

The next most obvious cause of aniseikonia is asymmetric convergence. When an object is brought near to the eyes, its image increases in size. If it is in the midline so that the eyes converge symmetrically on it, the increase is equal in the two eyes, but if the object is far to the right or left, it is obviously not equally near each eye but much nearer one eye. The retinal image in that eye is therefore larger, as was pointed out over two hundred years ago by Desaguliers. As in the case of aniseikonia due to anisometropia, so in that due to asymmetric convergence the eyes are able to compensate in some way for the aniseikonia and to carry on without trouble.

In some cases no well known cause exists to account for the aniseikonia. In such cases also the eyes are able to compensate for small or moderate amounts.

What do the eyes do when faced with a large amount of aniseikonia? When the task of compensation for any of the defects of binocular vision is too great for the amplitude of adjustment, the exit is by suppression and so by avoidance of binocular vision, with escape from eyestrain at the price of strabismus.

Given a case of strabismus, the question should arise. To what feature of the neuromuscular mechanism for binocular vision is it due? Is it perhaps due to the fact that the amplitude of fusion is inadequate to overcome a tendency to deviation from some cause, or is the relative accommodation inadequate to overcome a hypermetropia which is easily overcome by the total accommodation (which of course requires convergence for its full activity)?

In an uncertain percentage of cases the cause will be found to be aniseikonia of too great a degree. How common this is is not known, because it is not easy to measure aniseikonia in cases of strabismus. The ophthalmoeikonometēr is designed only to test patients with binocular vision and fairly good vision in each eye. There is good reason to suspect that aniseikonia is a fairly common factor of importance in the causation of strabismus.

The consequences of aniseikonia may be considered from two points of view: (1) how it affects seeing and (2) what symptoms it causes.

The holopter will serve as an illustration of its effect on seeing. This is well demonstrated by the tilting plane. The significance of the experiment with the tilting plane is that it shows how aniseikonia can affect seeing, perspective and space perception, and therefore orientation.

There are many possible applications of the use of eikonic lenses, as for example, for aviators in landing. An ordinary person in viewing surfaces (landscapes) may have obscure difficulties in adjustments that would be helped by the wearing of eikonic lenses.

Such cases are not, in my opinion, very numerous.

Much harm has been done to the cause of aniseikonia by exaggerated claims for its cure. This is natural enough when a new discovery is made. The relief of patients for the first time by eikonic lenses is a thrill to the discoverer which must excite enthusiasm. Indeed, lack of enthusiasm under the circumstances would be deplorable.

I have already mentioned clinical consequences of aniseikonia—eyestrain in some of its protean manifestations.

There have been various objections or criticisms to the claim that aniseikonia is a factor in binocular vision of sufficient importance to play a part in the practical treatment of eyestrain. I will mention three.

1. The benefit alleged to follow the use of eikonic lenses is really due to suggestion—a form of psychotherapy.

It is, of course, true that psychotherapy accounts for much of the benefit obtained in the treatment of eyestrain by spherocylindric lenses and by prisms as prescribed for astigmatism and heterophoria. No one with much experience and an open mind can fail to have seen cases illustrating this. The treatment of aniseikonia is no exception. On the contrary, the novelty, the prolonged and impressive examination, the "whoopie," if one likes to call it that, tend to impress the patient, who, by the nature of the condition (asthenopia), is more neurotic than the average patient. Hence the psychic factor plays a large part. Doubtless, more than one examiner has failed to allow for this in cases he has reported. Not to recognize the importance of this factor in esti-

rating the value of the treatment is a mark of lack of breadth of view and lack of adequate experience. It has always been emphasized at the Department of Research in Physiological Optics at Dartmouth Medical School.

On the other hand, to conclude that psychotherapy is the whole explanation, to say that since this case and that case are obviously of a psychic nature, all cases are of a psychic nature or are to be explained in some other way than that claimed by the investigators, is even more narrow, prejudiced and scientifically stupid. The only worse scientific sin is scientific dishonesty. To condemn a thing without a fair impartial hearing, without investigating it, is close to condemning it dishonestly.

2 A second criticism was advanced from the Wilmer Ophthalmological Institute and was elaborated by Friedenwald. The criticism is justified by a superficial study of the problem. Hess and others have pointed out the effect of the prismatic action of different lenses before the two eyes when the wearer looks through the periphery of the lenses. Eikonic lenses may have a considerable prismatic effect. They could act in two ways. 1 They could cause a strain by inducing a phoria for which the eyes have to compensate by fusion. 2 If there happened to exist a phoria of the right kind and degree, the eikonic lenses would correct it and so give comfort. Friedenwald, with his well known ingenuity and skill, builds up a plausible picture of anisophoria. By this he means phoria which differs in different directions of gaze, i. e., it is not concomitant. It is like the phoria produced by a slight paresis of some ocular muscle or muscles.

When the patient is being tested for aniseikonia, he is made to fix the eye on the central black disk, then to fix it on one after another of the four peripheral dots and stars. If there is anisophoria, when he looks, say, to the right, the dot and star will not be superimposed but separated in a typical way, the distance increasing the farther the eye is rotated in that direction. A lens with the properties of the eikonic lens could correct this.

There are various objections to this theory, but the following are easy to grasp and are convincing. (a) The tilting table effect cannot be explained by anisophoria but is readily explained by aniseikonia. (b) Instead of shifting the gaze from the center to the four peripheral dots, a different setup may be used. The essence of anisophoria is the difference in phoria in different directions of gaze. With this new setup, fixation is constant in the primary position. The observer looks at a suitable test object at the center of the screen seen binocularly. At about 200 mm from this is a large object seen in the periphery of the retina. This is projected on the screen with polarized light as two images, one for each eye, one polarized vertically and the other horizon-

tally The eyes seeing through polarized glasses see one image with each eye If one image is displaced either by aniseikonia or in any other way, the distance from the center object which is being fixated to the peripheral object is not the same for each eye The eyes cannot continue to fuse both the central object and at the same time the peripheral object, one must give way One would expect that with the attention riveted on the object it would remain single and the peripheral object would become double Instead, the peripheral images are fused and the center object is seen double By measuring the separation by suitable means, the inequality (aniseikonia, if present) can be measured Since the fixation throughout is on the center object there is no possibility of anisophoria affecting the result

Incidentally, this importance of peripheral stimuli to fusion has been shown¹ to be so powerful as to make it impossible to maintain macular fusion under certain circumstances

When I first saw the ophthalmoeikonometer, I raised the question When the eyes shift from the central large black disk to the small dot and stay, how does one know that fusion of the disk now in the periphery is maintained and that the eyes do not try instead to fuse the dot and stay? I was told that trial had shown that in fact fusion is maintained by the stimulus in the periphery Now there is astonishing proof of it

3 The third objection was raised at the Howe Laboratory of Ophthalmology and elaborated by Ludvig It is based on the age-old observation that in asymmetric convergence the object is nearer one eye than the other, and so its retinal image is larger This difference in size is greater than many of the differences which are claimed clinically to cause symptoms

Aside from other answers to this criticism, the most convincing is the simple experiment of measuring the difference in size in asymmetric convergence when the object is the same distance from each eye This is readily done with the Ames haploscope

One eye looks at a circle and the other at a square the size of which equals the diameter of the circle When they are superimposed, the circle would be tangent to the four sides of the square If one is larger or smaller than the other, the difference would be easy to recognize and easy to measure by noting how much the smaller image needs to be moved nearer the eye to become equal to the size of the other image

The astonishing fact is found that in asymmetric convergence there is an automatic increase in the size of the optical image of the adduct-

1 Burian, H Fusional Movements Role of Peripheral Retinal Stimuli, *Tr Sect Ophth*, A M A, 1938, to be published

ing eye This equals, and therefore compensates perfectly for, the difference in the size of the retinal images due to the difference in distance as seen (without the haploscope) in ordinary vision

The way the progressive development of the investigation has solved many of the objections raised by both friendly and hostile critics is one of the most impressive reasons for confidence in the Dartmouth Eye Institute

Time will not permit an enumeration of the many mathematical, experimental, theoretic and practical clinical investigations made by Ames and his staff Considering the novelty of the problems and the necessity of gathering and training a group of workers capable of supplementing each other's deficiencies and of cooperating with the musketeers' spirit of "all for one and one for all," and also the necessity of conceiving new methods of study, of designing and constructing new apparatus, of designing new types of lenses and enlisting the cooperation of the manufacturing optician in making them, of applying clinically the discoveries brought to light in the laboratory, which requires training technicians in the use of the apparatus, of devising new ways of approach to the problem in the effort to clarify its obviously numerous obscure and puzzling features, some of which have been detected by critics but many more of which have been seen and faced by the workers themselves—considering all these and many other difficulties which have been or are being overcome, the achievement in the few years that Ames has been working, having started literally from the beginning, is amazing It commands the enthusiastic support of all who have the interests of the progress in ophthalmology at heart To throw obstacles in its way simply because, like all pioneer work, it is not flawless is most unbecoming, not to say unfair Constructive criticism is always in order and always welcome

PHYSIOLOGIC AND CLINICAL OPHTHALMOLOGIC PROBLEMS IN RELATION TO INDIVIDUAL VARIABILITY

A BRUCKNER
BASEL, SWITZERLAND

(Concluded from page 756)

LIGHT AND DARK ADAPTATION

As an example of the general adaptation of the eye mentioned at the beginning of this article, observation shows that the eye is able to adapt itself to very different degrees of intensity of light⁷⁵ The investigations of the twentieth century have increased knowledge concerning light and dark adaptation greatly, although many questions are not yet solved

The method of measuring the degree of general adaptation consists in the observation of an uncolored field of a definite size which can be illuminated by different intensities of light The range of these intensities is great Many forms of apparatus have been constructed, one of the best seems to me to be that of Nagel⁴⁸ The intensity measurable can be varied from unity to 8,000,000

The subject first studied is normal and pathologic dark adaptation, although light adaptation also shows individual differences and perhaps pathologic deficiencies But at present not enough is known about it, especially as the measurement of increasing light adaptation presents greater difficulties than that of dark adaptation only⁷⁶

Former investigations were confined to the determination of the relative thresholds, without measurement of the absolute intensity of

75 A non-neutral (not absolutely white) illumination, such as is given by an ordinary artificial source of light with its insufficiency of short wavelengths, causes a general alteration of the sensitivity of the eye Usually this is noticed only when the phenomenon is very strong I had an experience in connection with this problem many years ago Awakening in the morning in a strange room, I saw all things bluish green, even my hands were the color of a corpse The explanation is that while I slept the sun had shone through the closed lids into my eyes for a long time Therefore, the eye was considerably exhausted for yellowish red, the color perceived by closed eyes, and the negative phase produced a hypersensitivity to the bluish green color and an insensitivity to red and yellowish color After about two hours normal sensitiveness returned The phenomenon is similar to the so-called erythropsia after snow dazzle

76 Lohmann, W Ueber Helladaptation, Ztschr f Psychol u Physiol d Sinnesorg (Abt 2) 54 161, 1922

light in the observation field. From such study in many cases of night blindness sufficient information may be obtained for practical purposes. But to detect slight differences of dark adaptation it is necessary to measure the intensity exactly, in lux or foot candles. Since it was desired to be more exact, the results obtained for a normal observer were compared with those for a person with a disturbance of dark adaptation, on the assumption, which is not justified, that all normal persons have the same course and degree of dark adaptation.

There are some difficulties or sources of error which must be avoided.

At first it seems to be easy to determine whether one sees the field of the adaptometer or not. But in the diminution of the thresholds some troublesome factors arise. The problem is to determine the difference between the subjective sensation of gray from one's eye which arises when an absolutely dark field is observed and a sensation only noticeably brighter produced by an objective stimulus. This is often difficult, because after a stay of about ten minutes in the dark room, as an expression of the change in the visual substance entoptic phenomena arise, such as bright clouds and flickering. These sources of error may be partly eliminated by experience and attention.

The width of the pupil must be rendered constant by artificial mydriasis. Although in the darkness the pupil dilates automatically, differences occur according to age; older persons have narrower pupils than younger ones. The amount of the light entering the eye naturally depends on the size of the pupil. The formula πr^2 shows that when the diameters of two pupils have the ratio 2 : 8 their areas have a ratio of 3 : 48, hence the intensity of light on the retina with these pupils is of the ratio 1 : 16. The thresholds, therefore, must vary if the pupil does not dilate owing to illness, for instance, in *tabes dorsalis*. The suggestion made by some writers that the reduction of adaptation in this disease is evidence of the central regulation of dark adaptation is not convincing, since this source of error was not always excluded. For the same reason the suggestion of Lasareff⁷⁷ is not correct. He stated that with increasing age the adaptation to light was found to diminish more and more. Therefore, Lasareff found by extrapolation of the curve that at about 150 years the adaptation should be zero and suggested that 150 years should be the moment of physiologic death! Besides the doubts which must naturally arise about extrapolation in general, this suggestion is certainly false, since he neglected the size of the pupil, which as a result of the increasing rigidity of the tissue of the iris becomes narrower with increasing age. After

⁷⁷ Lasareff, P. Sur le changement de la sensibilité au cours de la vision périphérique avec l'âge de l'homme, *Riv di biol* **10** 617, 1928.

artificial dilation of the pupil, such as that used by Hans Karl Muller⁷⁸ and Geneviève Matthey⁷⁹ in their experiments, carried out in Basel, a diminution of dark adaptation dependent on age is not to be found.

A further factor influencing the result is the state of the visual organ at the beginning of the experiment, depending on the duration and the degree of the previous illumination. Recent observers have illuminated the eye by means of a brightly illuminated white sphere (3,000 lux vertical to barium sulfate) for ten minutes after a stay of thirty minutes in a dark room⁸⁰. One may then suppose that the conditions of general adaptation are equal in all cases, especially if during the process of light adaptation the eyes are moved in all directions.

It is known that a retinal zone of some excentricity—about 20 degrees—to the macula adapts best to darkness. One often takes this into consideration, using a small red point, situated above or beside the field of the adaptometer, for the fixation of the eye.

All these precautions were taken in researches carried out by Hans Karl Muller⁷⁸ and his pupil Geneviève Matthey⁷⁹ in the ophthalmologic clinic in Basel.

In the measurement of the threshold value of the illumination of the field, a stimulus a little above the threshold was first given. This was diminished and the moment of disappearance noticed. The values were taken every three minutes during a stay up to three fourths of an hour in the dark room. A curve was then constructed in a manner differing from the usual procedure.

Nagel⁴⁸ and Piper⁸¹ did not plot the values for thresholds themselves but used their reciprocal values and treated these as a measure of the sensitiveness of the eye. In the first few minutes after light adaptation the thresholds are very high, but they quickly decrease. Their reciprocal values are very small. For this reason a curve, as shown in figure 39, taken from Piper, must produce the impression that during the first six to eight minutes the dark adaptation increases very slowly. That is not true, as any one who enters a dark room will observe.

78 Muller, H. K. (a) Ueber den Einfluss verschieden langer Vorbelichtung auf die Dunkeladaptation und auf die Fehlergrösse der Schwellenreizbestimmung während der Dunkeladaptation, *Arch f Ophth* **125** 624, 1931, (b) Zur Darstellung des Dunkeladaptationsverlaufes in Kurvenform für klinische Untersuchungen, *ibid* **125** 614, 1931.

79 Matthey, G. Eine "Standardkurve" der Dunkeladaptation für klinische Untersuchungen, *Arch f Ophth* **129** 275, 1932.

80 Hertel. Untersuchungen des Lichtsinnes mit einem Kugeladaptometer, ein Weg zu seiner Standardisierung, in *Concilium Ophthalmologicum*, Leiden, Netherlands, Eduard Fjdo, 1929, vol. 4.

81 Piper, H. Ueber Dunkeladaptation, *Ztschr f Psychol u Physiol d Sinnesorg* (Abt. 2) **31** 161, 1903.

If the intensity of the thresholds themselves or their logarithms are plotted, the curve falls quickly in the first minutes, as is shown in figure 40

In passing, some experiments may be mentioned which were intended to exclude the influence of a longer duration of the stimulus in causing a local adaptation. Momentary stimuli were used which, according to the so-called method of mean variation, were presented by a camera shutter, as shown in figure 41. The value of the thresholds depends on the time of exposure, as in using colored stimuli for the light-adapted eye. But this method is inconvenient, since it requires a number of single experiments, which fatigue the observer. Nevertheless, the results demonstrate that with the increase in the time of exposure the thresholds diminish. Therefore, it may be concluded that after exposure to a stimulus of a not too great duration the thresholds will be lower.

It was obvious that it would be possible to obtain a curve demonstrating the normal course of dark adaptation only by ascertaining the

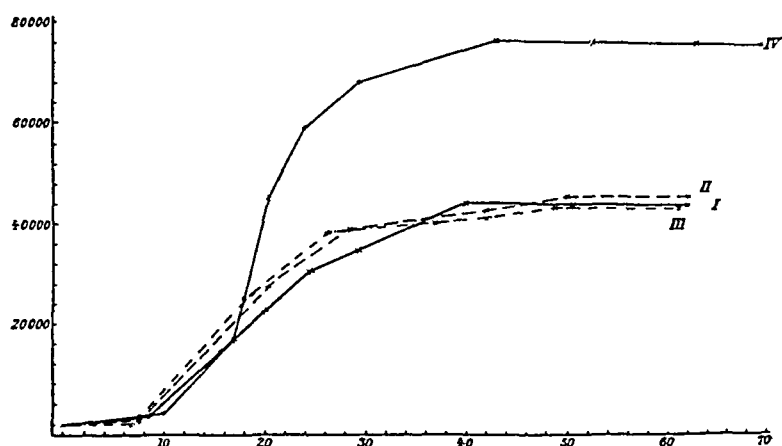


Fig 39—IV indicates binocular adaptation and observation, I, right eye observes, both eyes adapted, II, right eye dark, left eye light adapted, only right eye observes, III, right eye dark, left eye light adapted, both eyes observe (After Piper⁸¹)

averages from a great number of observers. Matthey examined 54 normal persons by monocular vision.

Figure 42 shows the standard curve for normal persons from the measurements made by Miss Matthey. There is no point in calculating the curve of variation to the other side, since the values would to a great extent lie below zero. I think that these curves, although obtained on only 54 persons, demonstrate the individual variability and also show whether dark adaptation is normal or not. When a curve lies outside, especially higher than the broken line, the dark adaptation is to be considered pathologic.

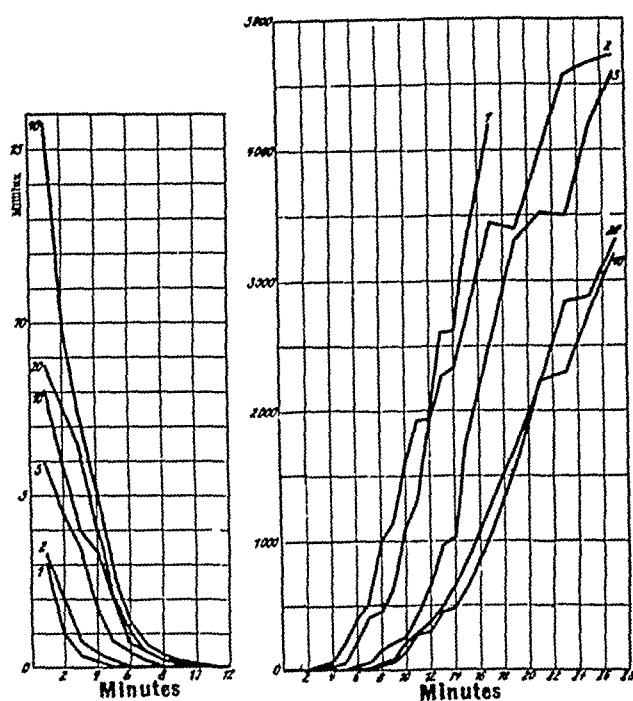


Fig 40—Adaptation curves (left chart) after preillumination of varying duration, represented by the irritation thresholds, which are given in millilux (average value of 10 single experiments) The numbers on the curves correspond to the duration of preillumination in minutes (After H K Muller ^{78a}) Adaptation curves (right chart) after preillumination of varying duration, represented by the reciprocal values of the irritation thresholds (average values from 10 single values, except with forty minutes) The numbers on the curves correspond to the duration of preillumination in minutes (After H K Muller ^{78a})

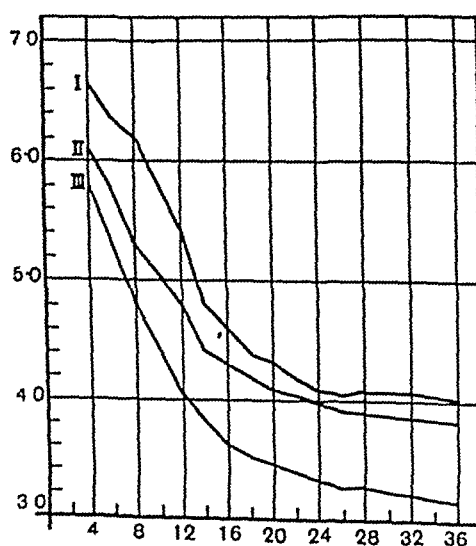


Fig 41—Curve of the threshold values for exposures of one-fiftieth second (I), one-tenth second (II) and with time illumination (vanishing threshold, III), with reference to a relative unit, represented by logarithms (After Bruckner ⁶⁴) Abscissas indicate duration of dark adaptation in minutes

Recently a standard curve has been published by Ferree, Rand and Stoll⁸² It is based on over 200 single curves. Unfortunately, the curve extends only to the twentieth minute after the beginning of adaptation, although important differences may occur after this time.

Wolfflin⁸³ suggested that persons with marked pigmentation of the fundus have better dark adaptation than those with little pigmentation. Certainly the latter are often more affected by strong illumination, they have headache, are dazzled, and so on, apparently because the damping of the light in the retina is too slight as a result of lack of pigmentation of the fundus oculi. But Miss Matthey did not find any differences in eyes with these two types of pigmentation (fig 43).

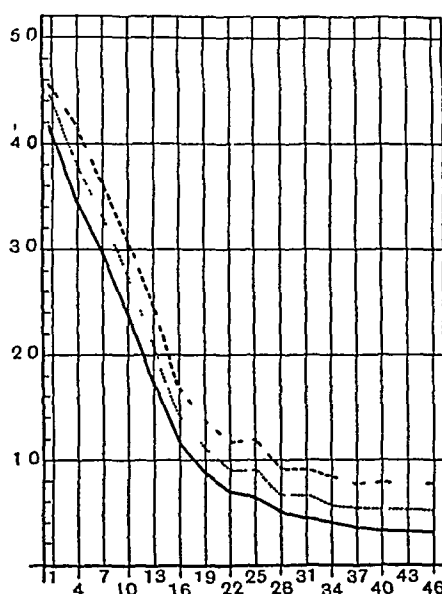


Fig 42—Standard curve of dark adaptation. The continuous line corresponds to the averages expressed as logarithms with a base of 10. The broken line shows the upper limit of scatter found by addition of the logarithms of the mean values and three times the amount of mean error of the single observation (3σ), while the dotted line represents the sum of the average and the single value of errors. (After Matthey⁷⁹)

The dependence of the dark adaptation on age, suggested by many authors and recently by Ferree, Rand and Stoll, has already been mentioned (fig 44).

There is also another result of some importance. If the observation is begun after there is good adaptation to light, in the first five

⁸² Ferree, C. E., Rand, G., and Stoll, M. R. Critical Values for the Light Minimum and for the Amount and Rapidity of Dark Adaptation, *Brit J Ophth* **18** 673, 1934.

⁸³ Wolfflin, E. Einfluss des Lebensalters auf den Lichtsinn bei dunkeladaptiertem Auge, *Arch f Ophth* **61** 524, 1905.

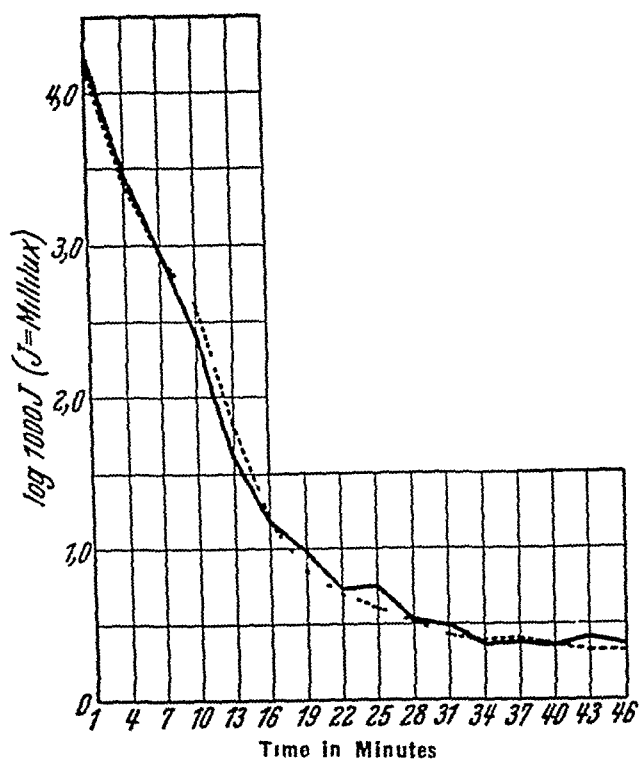


Fig 43—Dark adaptation The unbroken curve corresponds to the average values of the intensities of stimulation thresholds in persons with a relatively slight pigmentation of the ocular fundi, the dotted curve is for persons with a relatively marked pigmentation of the fundus oculi (After Matthey⁷⁹)

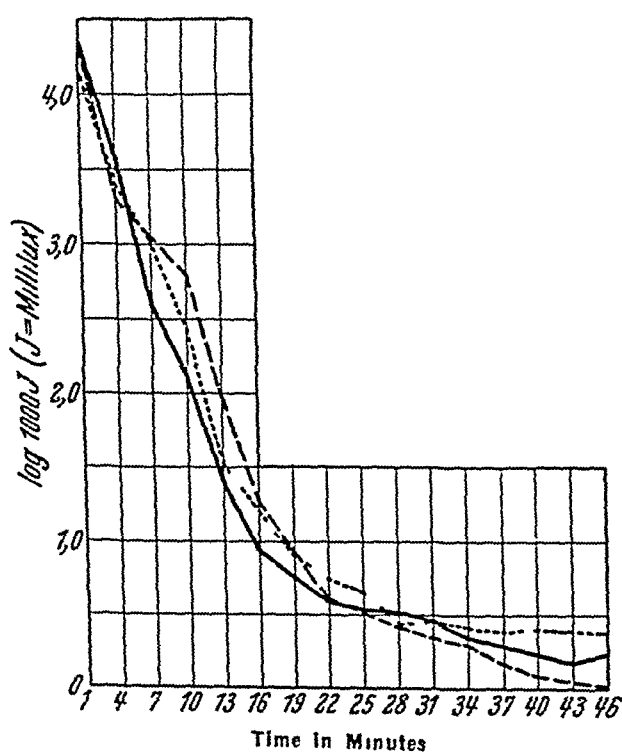


Fig 44—Dark adaptation The dotted curve corresponds to the average values of the intensities of stimulation thresholds in persons of 11 to 17 years, the broken one is for persons of 18 to 39 years and the unbroken one for persons of 45 to 59 years (After Matthey⁷⁹)

to seven minutes the daylight apparatus functions alone, then follows a period during which both the dark and the daylight apparatus will be stimulated by the light, and finally a period during which only the dark apparatus reacts to the low stimuli

This consideration leads to the theoretic suggestion of four possible types of adaptation

1 A type with fairly high thresholds both at the beginning and at the end, corresponding to an imperfect working of both the light and the dark apparatus

2 A type with relatively high thresholds at the beginning and fairly low thresholds at the end of the course This corresponds to a poorly

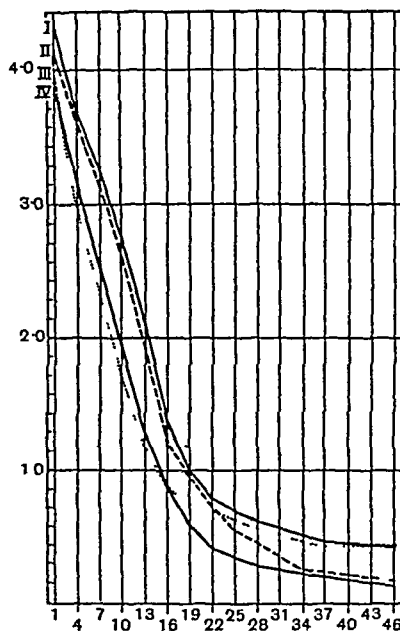


Fig 45—Course of adaptation curves in the four different types of adaptation which can be distinguished (After Matthey⁷⁹)

functioning daylight apparatus with a dark apparatus which functions well

3 Inversely, a type with relatively low thresholds at the beginning and relatively high thresholds at the end

4 The best type, with low thresholds throughout, corresponding to a good functioning of both the light and the dark apparatus

The curves in figure 45 show these four types of adaptation If the differences, especially at the beginning of the adaptation, do not seem to be large, it must be remembered that the curves represent the logarithmic values of the stimuli, and that the real differences are large

If the occurrence of these four types is confirmed by further observations, this will support the so-called duplicity theory. While Hering⁸⁴ expressed the opinion that the visual substance was uniform, the zoologist Max Schultze⁸⁵ in 1866 tried to prove by comparative anatomic studies that cones predominated in the retina of the daylight animals and rods in that of the night animals. This theory was amplified by Parinaud⁸⁶ in Paris and von Kries in Freiburg⁸⁷. According to the duplicity theory, the light apparatus lies in the cones and the dark apparatus in the rods of the retina (fig 46). The latter alone contain the visual purple in their external limbs. The absorption which takes place when the different wavelengths of light pass through a solution of visual purple accords satisfactorily with the curve of the brightness of the spectrum at low intensity observed by the dark-adapted eye, under these conditions the eye does not see any color, but everything seems whitish gray or black. The curve coincides with that of the bleaching

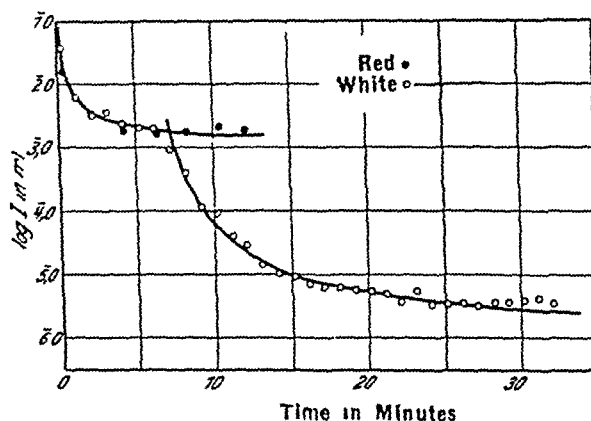


Fig 46—The white circles correspond to the logarithms of the stimulation thresholds in an adaptation experiment. There is a definite turning point between the fifth and the tenth minute. The black circles are the logarithms of the stimulation thresholds in another adaptation experiment, in which only the cones of the fovea have been excited. The curves correspond to the values which Hecht calculated on the basis of his adaptation formula. Hecht has, for the mathematical analysis, separated the adaptation curve into two independent curves. One comprises the course of adaptation up to the turning point and illustrates the dark adaptation of the light apparatus, the other corresponds to the adaptation of the scotopic apparatus. (After Hecht, *S. Ergebn d. Physiol.* **32**: 282, 1931.)

⁸⁴ Hering, S. 1920, vol 2.

⁸⁵ Schultze, M. Zur Anatomie und Physiologie der Retina, *Arch f. mikr. Anat.* **2**: 175, 1866.

⁸⁶ Parinaud, H. La vision, Paris, Octave Doin, 1898.

⁸⁷ von Kries. Zur Theorie des Tages- und Dämmerungssehens, in Bethe, A., and others. *Handbuch der normalen und pathologischen Physiologie*, Berlin, Julius Springer, 1929, vol 12, pt 1, p 678.

values of spectral lights for the visual purple (fig 47) This fact suggests that the latter is the substance the disintegration of which stimulates the rods in the retina

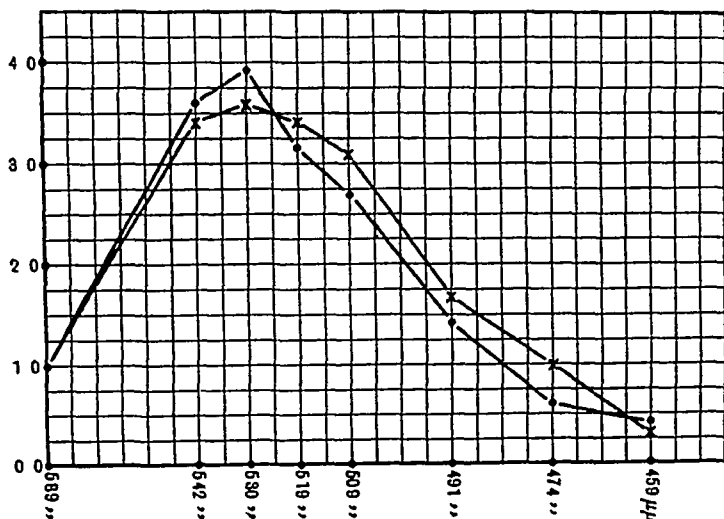


Fig 47—The X and dash line indicates the bleaching values of spectral lights for frog's visual purple, and the dash and dot line, the human scotopic luminosity curve. The abscissas indicate the wavelengths of the prismatic spectrum of the Nernst light, the ordinates, an arbitrary scale (After Trendelenburg *Ergebn d Psysiol* **11** 1, 1911)

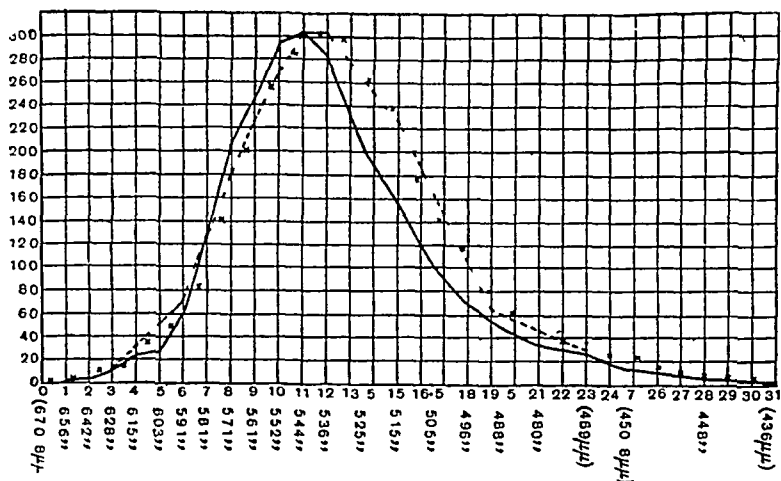


Fig 48—The continuous line indicates the achromatic scotopic luminosity curve of a deuteranope. The dotted line is the luminosity curve of a monochromat. The abscissas indicate wavelengths of the prismatic spectrum of gaslight, the ordinates, an arbitrary scale (After von Kries, J. *Ueber Farbensysteme*, *Ztschr f Psychol* **13** 241, 1897)

As mentioned before, the scotopic luminosity curve is the curve for persons with total congenital color blindness (figs 48 and 14)

The observations of general adaptation hitherto described were all concerned with the results obtained with one eye. When both eyes are used, the binocular thresholds change when the dark adaptation has progressed, namely, after a stay of ten or twelve minutes in the dark 100m. The binocular threshold has then only half the value of the monocular (fig. 39). The statements of writers differ on this point. Nevertheless, there is a remarkable difference between the light-adapted eye and the dark-adapted eye. The summation of the sensations is not to be found with the light-adapted eye, even when stronger stimuli are used. The brightness of an uncolored field is almost the same with monocular as with binocular vision.

Meanwhile, new experiments dealing with this problem have been carried out by G. Schumacher⁸⁸ in the ophthalmologic clinic in Basel. As a stimulus this author used red light, which can be perceived only by the daylight apparatus. He found that only the dark apparatus has the faculty of summation of the impressions of both eyes.

The difference between the dark-adapted and the light-adapted eye is of theoretic interest. The summation of the stimuli as the correlative of the psychic sensations must be localized in the central part of the visual pathway. In all probability it is situated in the corpus geniculatum laterale or the visual cortex of the posterior lobe or even higher. In view of the influence of the state of adaptation on the summation, one must suppose that these central parts are also influenced by the general adaptation.

Starting from the duplicity theory, which is based on the presence of different retinal elements, it is not easy to understand how the change of excitability in the retina can influence the nerve centers. Although it has not been sufficiently demonstrated, one must suppose that, just as I have shown for local adaptation, the light-dark adaptation is not confined to the peripheral organ.

Some other facts point in the same direction. The entoptic visibility of the blindspot is especially distinct when the eyes are slightly dark adapted, for instance, in looking at the sky after sunset. Extreme degrees of dark or light adaptation are both unfavorable for this observation. Because the visibility of the blindspot, as already mentioned, is localized in the central parts of the visual pathway, it seems that the general adaptation influences these processes dependent on the contrast.

⁸⁸ Schumacher, G. Ueber das Verhalten der monokularen und binokularen Reizschwelle während der Dunkeladaptation des Tages- und Dämmerungsapparates, *Acta ophth.* 15 5, 1937. This article gives references to other literature on this subject.

A little while ago Frey,⁸⁹ in Zurich, Switzerland, found centrifugal neural paths between central parts of the brain trunk and the retina. This fact also suggests, from the anatomic point of view, not only a central influence on the peripheral organ but the possibility of a reciprocal relation between the brain and the retina. In this way the importance of dark adaptation in the phenomena of contrast may be understood.

I shall now speak of the stimulation of the eye not by the adequate light stimulus but by the electric current. The results here differ if the thresholds of the light-adapted and of the dark-adapted eye are compared. Georg Elias Muller⁹⁰ found the same thresholds for both, but others⁹¹ found the threshold for the dark-adapted eye about half that of the light-adapted eye. Certainly the results were obtained before chronaxia, first studied by Lapicque, was known. Therefore, Achelis and Merkulow,⁹² who considered this point, were probably right when they stated that the irritability of the retina was equal in both cases. It seems to me, however, that this question is by no means fully elucidated. In all cases of electric stimulation the amount by which the threshold stimulus is diminished is of a totally different size from that observed when the specific light stimulus is used. The electric current must act on the visual substance at another part, perhaps, as some authors (for instance, Hecht) have suggested, in the optic nerve and not in the retina. In this respect it may be of interest to record that a summation of electric stimuli is apparently to be found only in the dark-adapted eye (table 4).

DISEASES ACCOMPANIED BY DISTURBANCES OF DARK ADAPTATION

I shall discuss briefly some diseases which are accompanied by disturbances of dark adaptation. This disturbance is called night blindness, or chicken blindness, as the Russians call it on the supposition that fowls go to sleep early in the evening because they cannot see at night.

89 Frey, E. Vergleichend-anatomische Untersuchungen über die basale optische Wurzel, die Commissura transversa Gudden und über eine Verbindung der Netzhaut mit dem vegetativen Gebiet im Hypothalamus durch eine "dorsale hypothalamische Wurzel" des Nervus opticus bei Amnioten, Schweiz Arch f Neurol u Psychiat **39** 255, 1937, **40** 69, 1937.

90 Muller, G. E. Ueber die galvanischen Gesichtsempfindungen, Ztschr f Psychol u Physiol d Sinnesorg **14** 329, 1897.

91 Bruckner, A., and Kirsch, R. Ueber den Einfluss des Adaptationszustandes auf die Empfindlichkeit des Auges für galvanische Reizung, Ztschr f Psychol u Physiol d Sinnesorg (Abt 2) **47** 46, 1912.

92 Achelis, J. D., and Merkulow, J. Die elektrische Erregbarkeit des menschlichen Auges während der Dunkeladaptation, Ztschr f Psychol u Physiol d Sinnesorg (Abt 2) **60** 95, 1929.

Those affected by night blindness cannot see sufficiently at low intensities and therefore are often disturbed when outdoors at night, especially in the country. During the World War, such persons, who in the cities did not perceive any incapacity, were unfit for military service, a great part of which was carried on after dark. I saw many

TABLE 4—*Summation of Binocular Excitations by Electric Stimuli in the Light-Adapted and in the Dark-Adapted Eye of Two Observers*
(After Bruckner and Kusch⁹¹)

					Summation in	Average Summation in		
Light	{	1	K	16 +	24 —	40%	}	40.5%
		2	K	16 +	33 —	33%		
		3	B	25 +	38 —	40%		
		4	B	39 +	40 —	49%		
Dark	{	5	K	55 +	31 —	64%	}	75.5%
		6	K	51 +	23 —	69%		
		7	B	42 +	8 —	84%		
		8	B	34 +	6 —	85%		

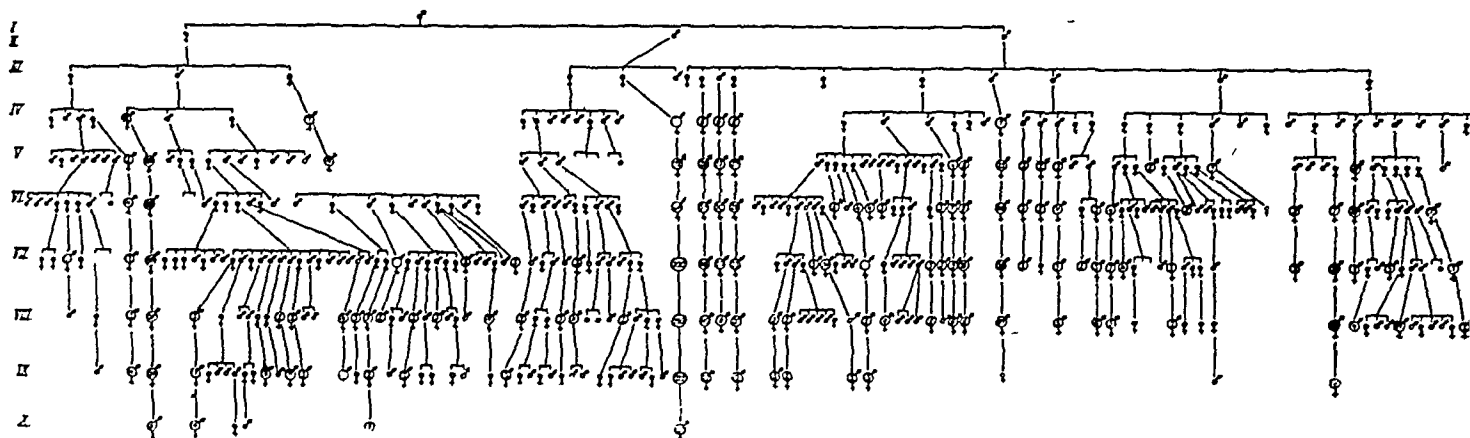


Fig. 49—Dominant heredity of night blindness (pedigree of Cumer-Nettleship) ♂ indicates affected male, ♀, affected female, ♂, healthy male, ♀, healthy female, the circles containing numbers, the number of healthy offspring (After Francheschetti, in Schieck, F. and Bruckner, A. *Kurzes Handbuch der Ophthalmologie*, Berlin, Julius Springer, 1930, vol. 1.)

of these men. They were sent back because their dark vision was insufficient. The degree of night blindness was often slight and the clinical state of the eye normal.

A good deal of night blindness is hereditary. The famous pedigree published by Cumer-Nettleship, which includes ten generations, is well known (fig. 49). The hereditary form is dominant. Curves of the adaptation made under the strict conditions mentioned in this article do not exist.

An acquired form of night blindness, with or without a condition of the conjunctiva known as xerosis, is well known to be caused by an insufficiency of vitamin A. In Denmark, during the war, night blindness was endemic because the population had to eat margarine,

which does not contain vitamin A. A similar condition is frequently encountered in Russia, as a result of fasting during Lent. Occasionally, it occurs also in Western Europe in the spring. The treatment is easy, because it is only necessary to give the patients vitamin A in the form of cod liver oil, fresh vegetables or carrots.

The disturbances of adaptation found in retinitis pigmentosa, a degeneration of the retina which is included in the so-called hereditary familial degeneration of the central nervous system, is known by all ophthalmologists. Types are found to vary from one generation to another. Unfortunately, so far there is no successful treatment for this disease.⁹³

As a result of the anatomic alterations in the retina there are deficiencies of dark adaptation in high myopia. The stretching of the inner layer in the posterior part of the eyeball apparently affects the nutrition of the retina.

I shall discuss with a little more detail a special form of myopia found in the eyes of children. An investigation into this condition was carried out in the ophthalmologic clinic of Basel in the eyes of 22 young children.⁹⁴ Probably such myopia is congenital. A definite type of heredity was not to be found. The fundus oculi is relatively lightly pigmented at the posterior pole, but the region of the macula, on the other hand, is markedly pigmented. The visual acuity is always more or less diminished, in the majority of cases it is not more than a third of normal. The dark adaptation, as seen in figure 50, which shows the average values in the 22 cases in comparison with the standard curve of the normal eye, is not much higher, and after three fourths of an hour the thresholds are nearly the same. In respect to the evaluated curves, the thresholds at the beginning of the adaptation are decidedly higher than the normal.

These facts suggest that in this special form of myopia the daylight apparatus is in some way deficient and the dark apparatus is not impaired. Still more, this opinion seems to be supported by a single curve, such as that shown for instance in figure 51, which shows the relation of the upper limit of the variations of normal adaptation to the adaptation curve of the myopic child. During the first ten minutes the curve lies outside the limit of normal, but it is thereafter nearly normal.

⁹³ Investigations just published by E. M. Josephson and M. Freiburger (Carotene Therapy of Retinitis Pigmentosa, *Nature*, London **139** 155, 1937) seem to prove that the intramuscular injection of carotene diminishes the night blindness in the initial stages of retinitis pigmentosa.

⁹⁴ Bruckner, A., and Franceschetti, A. Myopie im Kindesalter, *Arch. f. Augenh.* **105** 1, 1931.

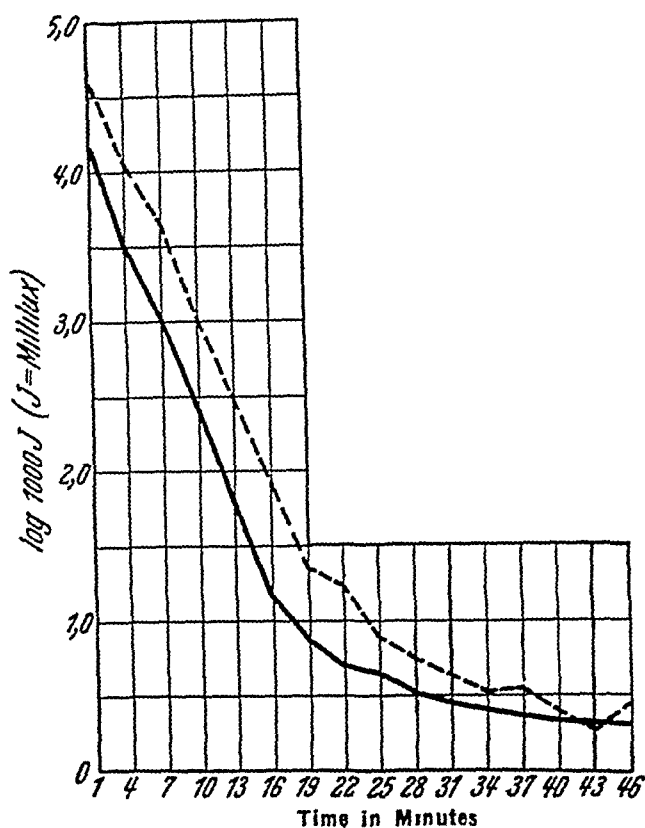


Fig 50—The continuous curve represents the “standard curve” of dark adaptation, the dash line corresponds to the average values of the intensities of the threshold stimuli in 22 children with myopia of the Bruckner and Franceschetti type (After Bruckner and Franceschetti⁹⁴)

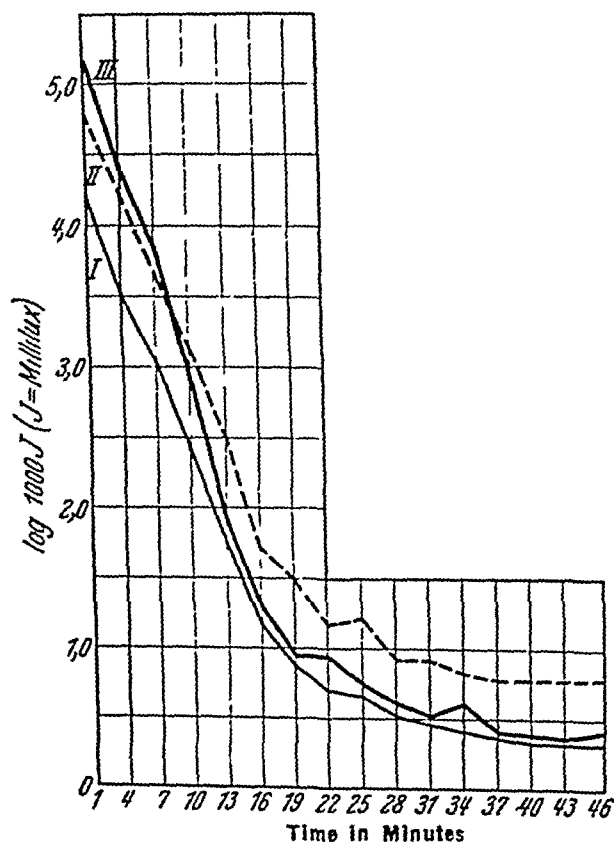


Fig 51—Curve I indicates “standard curve”, curve II, upper boundary of spread, curve III, adaptation curve of a child with myopia of the Bruckner-Franceschetti type (After Brückner and Franceschetti⁹⁴)

CLINICAL METHODS

FOCAL ILLUMINATION

Although in individual clinical cases the estimation of the visual acuity is often difficult, the results obtained by intelligent persons with normal eyes agree sufficiently. The variation in the illumination of the test cards is of most importance. If this is too low or too high the visual acuity is less than with a moderate degree of illumination. Probably the curve of visual acuity is not a linear function of the intensity of the illumination according to the Weber-Fechner law but is, as Janisch⁹⁵ suggested, a so-called chain function (*Kettenfunktion*). The difficulty arises from the fact that alteration in the intensity of illumination also produces changes in the adaptation of the eye, and it takes some time for these changes to occur.

But I shall not discuss again this complicated subject, which helps one to understand the physiology or psychology of the procedure known as focal illumination. This consists in the concentration of light by means of a convex lens on that part of the eye which it is desired to examine exactly, e. g., a cornea with opacity. This is seen much more clearly by focal illumination than by simple diffuse light. If the Weber-Fechner law were valid for the relative intensities of illumination involved, there would be no improvement of discrimination.

One reason for the increase in visibility of the opacity is the slight degree of dark adaptation which takes place when this method is used in the dark room, but there is another, more physical, factor. According to Lord Rayleigh, the intensity of the light diffracted by the dispersed particles of a medium increases in proportion to the sixth power of the size of the dispersed particles. But this is valid only in the case of the colloidal dispersions from 0.1 to about 1 millimicron (fig. 52). If the dispersed particles are larger, the diffraction again decreases and the visible opacity diminishes.

Thus it is understood why in the intensive light of the slit lamp the normal cornea appears not clear but relucet. If the cornea has opacities, these may also be seen by daylight, because the degree of dispersion in them is larger than in a clear cornea. I suggest that it is in this and not in the rough anatomic structure of the scars in the cornea that the reason for the opaque appearance of these parts must be sought.

TONOMETRY

Generally speaking, the ophthalmologist makes use of the visual sense when examining his patients. The physician or the surgeon more frequently employs the sense of hearing or touch. Therefore the

⁹⁵ Janisch, E. Das Exponentialgesetz als Grundlage einer vergleichenden Biologie, Berlin, Julius Springer, 1927.

ophthalmologist often feels lost if he cannot use his eyes, as in the case of a growth in the orbit or elsewhere

But in the investigation of the intraocular pressure, the sense of touch is often used in feeling the eyeball through the lids. This method, first employed by Bowman,⁹⁶ is still used by many physicians, although the majority now use a so-called tonometer. By means of this instrument the estimation of the intraocular pressure is transferred from the sense of touch to that of vision. It is necessary only to observe the position of an indicator on a scale.

Although these instruments have also their sources of error, the measurement is thereby made much more exact than the mere touch. The absolute value of the intraocular pressure is not measured, chiefly because the rigidity of the cornea varies according to the age of the patient. Nevertheless, by the use of new curves, calculated by Hans



Fig 52—Dependence of the intensity of the dulness on the size of the particles, it increases to a maximum and then decreases again (After Ostwald, C W W Kolloidwissenschaft, Elektrotechnik und heterogene Katalyse, Kolloidchem Beihefte, 1930, vol 32, nos 1-4)

Karl Müller,⁹⁷ this pressure may be estimated with sufficient accuracy (fig 53)

It is interesting to determine the amount of error present when the physician estimates the tension in millimeters of mercury by the fingers and immediately before or after this determines the tension instrumentally. This estimation is more complicated than the original method of Bowman. He distinguished nine degrees: normal tension, tension doubtfully, just or distinctly increased or diminished, and the two extremes, with the eyeball hard as a stone or soft as pap.

In figure 54 the pressure determined by the tonometer is shown by the abscissas and the pressure determined by the fingers by the ordinates. M , the curve of averages, shows that from 10 to 30 mm

⁹⁶ Bowman, W. On Glaucomatous Affections and Their Treatment by Iridectomy, Brit M J 2 377, 1862

⁹⁷ Müller, H K. (a) Augendruck und Lebensalter, Arch f Augenh 105: 504, 1932, (b) Ueber die Fehlergrösse der Augendruckmessung mit dem Tonometer von Schiotz, ibid 105 516, 1932

the coincidence is satisfactory. But in the higher pressures the estimate by the fingers is always too low. In the spread allowed by one (σ) and three times the standard deviation (3σ) the error may be

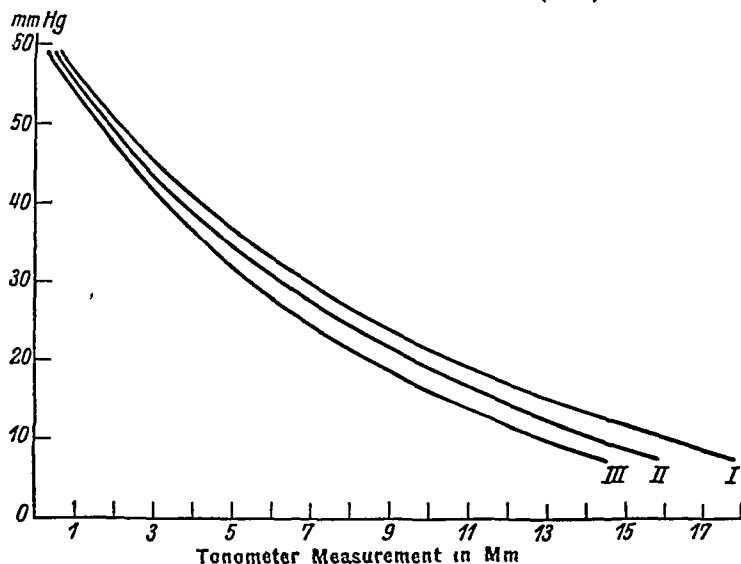


Fig 53—Tonometric gaging curves for the 75 weight (original Schiotz tonometer). Curve *I* is for children between 1 and 10 years of age, curve *II*, for persons between 20 and 44 years, curve *III*, for persons between 45 and 90 years. (After H K Muller^{97a})

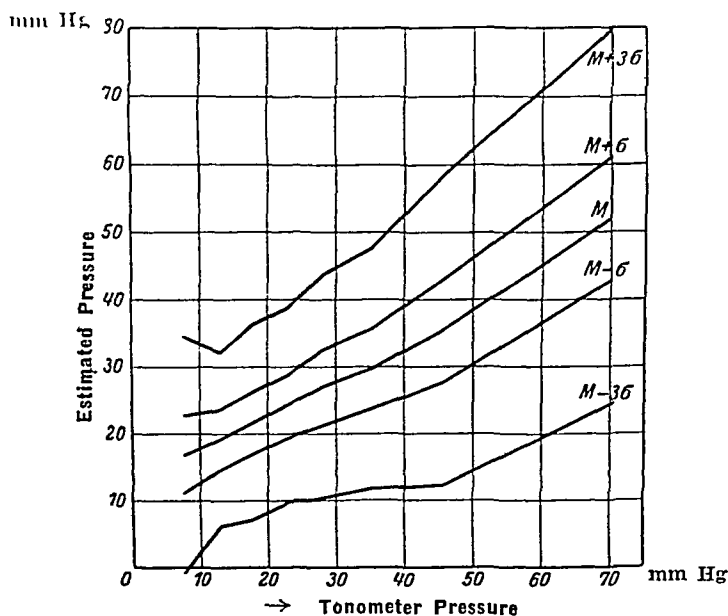


Fig 54—Spread of the digital estimation of pressure. (After H K Muller^{97b})

considered excessive. For instance, a tension of 60 mm may be estimated as 20 mm or one of 25 mm as 40 mm of mercury. If a pressure of 25 mm is considered the upper normal limit, the results show that it is almost certain that the pressure lies within the normal limits when the eye is estimated as very soft.

Even in determining only the difference between the two eyes, the errors are important. The experience of my colleagues and myself has shown that in 36 per cent of cases the difference was not correctly estimated.

These results show clearly that it is impossible to rely on the fingers as a method of estimating tension in glaucoma.

SENSITIVENESS OF THE CORNEA

In order to obtain knowledge of the state of the sense organs of the skin in disease it has been necessary to obtain exact values for the stimulus applied in testing that sensation. In the skin one distinguishes different qualities of sensation—those of touch, pain, cold and warmth. Knowledge of the function of these senses has been furthered by Max von Frey,⁹⁸ Goldscheider,⁹⁹ Blix,¹⁰⁰ and others. Von Frey used hairs (fig 55) of different stimulus value, which he determined from the relation between the area of the hair and the weight it was able to support. This pressure value, as von Frey called it, is determined by



Fig 55—Stimulus hair (After von Frey^{98a})

the formula $\frac{\text{grams supported}}{\text{area of hair in sq mm}}$, which gives a figure of greatest importance for measuring tactile thresholds in skin.

The same method has also been used in recent times by ophthalmologists, for instance, Marx,¹⁰¹ Studemann,¹⁰² and Pfimlin.¹⁰³ It was used especially to determine the thresholds of the cornea.

98 von Frey, M. (a) Beiträge zur Physiologie des Schmerzsinnens, Ber u d Verhandl d k sächs Gesellsch d Wissensch zu Leipzig, math-phys Kl **46** 187, 1894, (b) Untersuchungen über die Sinnesfunktionen der menschlichen Haut, Abhandl d math phys Cl d k sächs Gesellsch d Wissensch **23** 208, 211, 212 and 228, 1897.

99 Goldscheider. Thermorezeptoren, in Bethe, A., and others. Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1926, vol 11, pt 1, p 131.

100 Blix, M. Experimentelle Beiträge zur Lösung der Frage über die spezifische Energie der Hautnerven, Ztschr f Biol **20** 141, 1884.

101 Marx, E. Die Empfindlichkeit der menschlichen Hornhaut, Leipzig, S Hirzel, 1925.

102 Studemann, H. Quantitative Prüfung verschiedener Anaesthetica in ihrer Wirkung auf die Hornhaut, Arch f Ophth **115** 119, 1924.

103 Pfimlin, R. Zur klinischen Unterscheidung verschiedener Formen des Herpes corneae, Ber u d Versamml d deutsch ophth Gesellsch **48** 212, 1930.

There has been a lengthy discussion about the qualities of sensation which can be perceived by the cornea. Von Frey supposed that it is possible to perceive only pain and cold, because in this membrane there are anatomically only two kinds of sense organs—the free ends of nerve fibers in the epithelium and the little bodies of Meissner-Krause. On the other hand, Goldscheider emphasized the ability of the cornea to perceive also the simple sensation of touch, if the stimulus is feeble enough. I am unable to agree with von Frey in his statement that one cannot judge the quality of sensations by perceptions but can consider it only with regard to the anatomic apparatus. Finally, perception alone has to decide the qualities of the sensations. Therefore, Stein¹⁰⁴ declared that the thresholds for touch and for pain in the cornea were almost equal, a finding which seems to be confirmed by the observations made in Basel, to which I have referred.

However, the hair method introduced by von Frey is a very good one and permits one to recognize the degree of the sensitiveness of the cornea in normal and in pathologic conditions much more exactly than when using a few fibers of cotton wool with an unknown stimulus value. Admittedly, the von Frey hair test has sources of error, but I shall not deal with these.

Many investigations by von Frey's method have been carried out to measure the efficiency of anesthetics on the cornea. Figure 56 shows some curves prepared by Studemann,¹⁰² which demonstrates how differently some anesthetics affect the sensitiveness of the cornea. Studies were first made on rabbits. A slight winking of the lids was taken as an indication that the animal perceived the stimulus.

When the thresholds of the human cornea are tested, the patient states when he feels a sensation. By this method one can satisfactorily determine whether the sensitiveness is diminished or not, because the mean threshold in the center of the normal cornea corresponds to a hair of from 0.2 to 3 Gm per square millimeter. The sensitiveness changes from point to point. Figure 57 shows a diagram published by Strughold¹⁰⁵ showing the different levels of thresholds. It will be noted that the sensitiveness is lower in the periphery of the cornea than in the center.

It appears that about 10 per cent of human beings have a higher threshold in the cornea, equivalent to a value of from 10 to 15 Gm per square millimeter (Pfäflin¹⁰³). These, too, are individual differences, the clinical value of which has not yet been established. It is not known, for instance, whether these corneas are more liable to

104 Stein, H. Welche Empfindungsqualitäten vermittelt Hornhaut und Bindehaut des menschlichen Auges? *Klin Wchnschr* 4 819, 1925

105 Strughold, H. Die Sensibilität der Horn- und Bindehaut des normalen menschlichen Auges, *Zentralbl f d ges Ophth* 19 353, 1928

attack from herpes corneae, a disease which is always accompanied by a diminution of the corneal sensitivity

The cold sensation of the cornea, studied especially by Strughold¹⁰⁵ has not been studied clinically I shall merely demonstrate the distribution of the points where cold can be perceived from an illustration published by this author (fig 58)

I believe that a more detailed investigation of the different qualities of sensation in the anterior segment of the eye may give interesting results

DISEASES OF THE EYE DEPENDENT ON CONSTITUTION AND ON CLIMATE

I have repeatedly spoken about the individual differences in the sensory function of the eye This difference of reaction between one person and another may be inherited or acquired

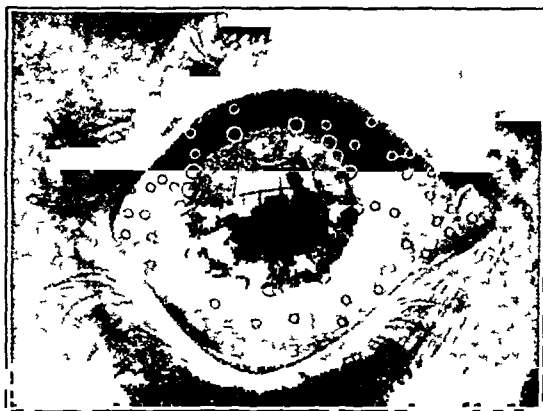


Fig 58—The topography of the cold points in the anterior part of the human eye (After Strughold, H, and Karbe, M *Ztschr f Biol* 83 2075, 1925)

I shall consider some examples of diseases of the eye which demonstrate the variable reactions of different persons to the same influences or factors causing diseases I shall touch on a question which is of great interest to modern medicine, namely, how the congenital constitution influences pathologic processes The same disease never takes exactly the same course in 2 cases The infectious diseases especially show extreme variations Formerly, when dependence on bacteriology was at its height, these differences were attributed to the variability of the micro-organisms Nowadays, differences in the constitution of the affected person are given a much greater weight than twenty-five years ago Undoubtedly, this brings up difficult problems, the extent of which is not fully realized and the solution of which is still far distant Of the manifold ophthalmologic subjects of this type, I shall discuss only two The first is tuberculous diseases of the eye, and the second, the dependence of some diseases on weather and climate

TUBERCULOUS DISEASES OF THE EYE AND GENERAL
CONSTITUTION

I shall consider the real tuberculous diseases of the eye, namely, those which are produced by a metastatic infection by the bacillus of tuberculosis itself, and of those conditions, only those of the uvea, namely, iritis, cyclitis and choroiditis. I shall also say a few words about scrofula of the eye as a tuberculotoxic condition.

It was Julius von Michel, of Wurzburg, Germany, who fifty years ago recognized that many inflammatory conditions of the uvea are tuberculous. Formerly all ophthalmologists, like those in France today, considered the majority of these disorders to be syphilitic. Certainly the cause depends on the locality, but now, with modern improvements in the methods of clinical investigation and by means of the Wassermann test, the cause of the condition can be ascertained in the majority of the cases.

I shall confine myself to a discussion of research which shows that the type of reaction to disease is dependent on general constitution and age.

It is well known that the primary infection in an animal takes an entirely different course from that of a secondary infection. Similar observations can be made in cases of tuberculous conditions of the eye, with their frequent relapses. While the first attack of tuberculous iridocyclitis is usually accompanied by severe symptoms of inflammation, these are often missing in relapses. Therefore, it sometimes happens that the patients themselves do not recognize the new attack. This is an example of the different reactions of the local tissue to the poison of the tubercle bacillus. Probably a local immunity is set up. However, in some cases the first attack also proceeds quietly, owing perhaps to the different mass of the bacteria carried into the eye by the blood stream or to a preexistent local immunity.

With regard to the frequency of tuberculous conditions of the eye, some important facts have been discovered. Ophthalmologists in the northern and southern parts of central Europe noticed that to the south of the Main River and in the region of the Rhine these conditions are much more frequent than in the northern parts. For instance, in Königsberg I observed only a single case of tuberculous choroiditis during four years, while I frequently saw cases in Wurzburg and in Basel. The same difference has been noted by Axenfeld between Rostock and Freiburg, Germany, and by Lohlein between Griefswald and Freiburg. These authors were completely at a loss for an explanation.

In my opinion, the influence of race is important. This suggested itself to me while I was reading a book by the psychiatrist Kiet-

schmei¹⁰⁶ He expressed the opinion that the genial temperament relatively frequent in the southern part of Wurtemberg is the result of the mixture of the different types of the race In the northern part of Germany tall, slender, asthenic or athletic persons are to be met with and are perhaps now better known through political considerations than from exact measurements by anthropologists There is a remarkable bodily difference between the types called asthenic and athletic and the smaller and broader type, with the so-called pyknic construction of body, so prevalent among the inhabitants of the Alps

The first two types, as Kletschmer¹⁰⁷ suggested, are correlated with the so-called schizothymic psychic behavior, the latter type, with the cyclothymic The latter are the persons who change in their reactions and alternate between optimism and pessimism, they are extroverts, while the others are introverts When these two physical and psychic types are mixed, as at the boundaries of the two parts of central Europe, internal psychic strained relations result, and these are often productive of genius

To suppose that differences of bodily constitution would influence the frequency of ocular tuberculosis was obvious The tall, slender persons of asthenic build are more frequently attacked by tuberculosis of the lungs than the small, broadly built type Still, the general impression suggested is that these pyknic types are especially affected by ocular tuberculosis but not by progressive tuberculosis of the lungs, as I shall show

Some measurements made in Basel by my colleague, Professor Gigon of patients with tuberculosis of the eye suggest that this idea may be right When compared with the measurements obtained by Neuer and Feldweg¹⁰⁸ in Germany for patients with tuberculosis of the lungs, certain differences are apparent The mean circumference of the chest is different, also the weight in relation to height (tables 5 and 6) If calculations are made with the indexes of Rohrer and Bornhardt¹⁰⁹ (tables 7 and 8) it will be seen that these patients represent the pyknic type and are in a better general bodily condition

In Rohrer's index the values between 1.11 and 1.31 are the limits for the asthenic type, from 1.36 to 1.49, for the athletic type, and from 1.47 to 1.58, for the pyknic type Therefore, the majority of these patients, especially the women, correspond to the last type

106 Kletschmer, E Geniale Menschen, Berlin, Julius Springer, 1929

107 Kletschmer, E Körperbau und Charakter, ed 2, Berlin, Julius Springer, 1922

108 Neuer, I, and Feldweg, P Körperbau und Lungenphthuse Beitrag zur Konstitutionswissenschaft, Ztschr f d ges Anat (Abt 2) **13** 88, 1927

109 Rohrer and Bornhardt, cited by von Rohden, F Methoden der konstitutionellen Körperbauforschung, in Abderhalden, E Handbuch der biologischen Arbeitsmethoden, Berlin, Urban & Schwarzenberg, 1925, vol 9, pt 3, chap 4

TABLE 5—Average Circumference of Chest of Patients with Tuberculosis of the Eye

Body Height, Cm	Number	Results in Basel Clinic, Cm	Results of Neuer and Feldweg, Cm
Men			
160 6 165 5	9	88 1	87
165 6 170 5	6	92 0	88
170 6 175 5	3	86 8	89
175 6 180 5	1	99 5	90
Women			
145 6 150 5	4	82 6	78
150 6 155 5	15	81 9	80
155 6-160 5	14	83 1	82
160 6-165 5	11	87 5	84
165 6 170 5	3	85 3	86
170 6-175 5	1	87 5	88

TABLE 6—Average Weight of Patients with Tuberculosis of the Eye

Body Height, Cm	Number	Results in Basel Clinic, kg	Results of Neuer and Feldweg, kg
Men			
160 6 165 5	9	66 12	57 5
165 6 170 5	6	72 6	60 5
170 6 175 5	3	66 9	63 5
175 6 180 5	1	81 8	66 5
Women			
145 6-150 5	4	58 9	44
150 6 155 5	15	64 1	48
155 6-160 5	14	63 4	52
160 6 165 5	11	68 9	56
165 6 170 5	2	66 5	60
170 6 175 5	1	85 5	64

TABLE 7—Measurements of Patients in Basel According to Bornhardt's Index *

Men		Women	
	Number		Number
<0	1	<0	2
+0 1 to +3 5	3	+0 1 to +3 5	6
+3 6 to +9 0	11	+3 6 to +9 0	16
>+9 0	5	>+9 0	25

$$* J = \text{weight} - \frac{\text{Circumference of Chest} \times \text{Body Height}}{240}$$

TABLE 8—Measurements of Patients in Basel According to Rohrer's Index †

Men		Women	
	Number		Number
<1 33	4	<1 33	4
1 33 to 1 40	2	1 33 to 1 40	7
1 41 to 1 50	7	1 41 to 1 50	5
>1 50	7	>1 50	33

$$* J = 100 \times \frac{\text{Body Weight}}{\text{Body Height}}$$

Concerning Bornhardt's index it may be remembered that + 9 corresponds to a very good state of nutrition, + 3 5 shows sufficient nutrition and a negative value indicates a bad condition

It must be remembered that the investigations made by my colleague and myself were chiefly confined to elderly persons, while Bornhardt's index is derived from younger ones. Further, it must not be forgotten that women after the climacteric become heavier, which, however, does not prove that the general state of nutrition is really better but indicates rather that the tissues may contain more water. Nevertheless, I think that these results perhaps give a hint for the elucidation of geographic differences in the frequency of tuberculous diseases of the eye.

A point which seems to be of importance is the preponderance of women over men. The first attack especially happens at a definite age. Concerning this point we found from a series of 100 cases of tuber-

TABLE 9—*Age of Onset of Tuberculosis of the Eye in Males and Females (After Achermann *)*

Age	0-9 Yr	10-19 Yr	20-29 Yr	30-39 Yr	40-49 Yr	50-59 Yr	60-69 Yr	70-79 Yr	Total
Female		10	9	15	18	14	3	1	70
Male	1	10	10	2	6	1			30
Sum	1	20	19	17	24	15	3	1	100

* Achermann, E. Klinisch statistischer Beitrag zur Kenntnis der Iridocyclitis tuberculosa, Schweiz med Wchnschr 56 1119, 1926

culosis of the eye that the beginning of the illness occurs in the majority of patients during the fifth or sixth decade (table 9). This holds good also when considered in relation to population. The second maximum of tuberculous disease, during the second and third decades, is apparent only if the figures are considered in relation to the whole population.

Consideration of sex incidence shows that the disease is more frequent in women than in men (table 9) in the fifth decade. Perhaps there is a second peak in the second decade, but the number of cases is not sufficient to justify a definite conclusion. In the male the peak lies in the second and third decades, the minimum in the fourth is not certain, owing to the small number of cases.

These differences in the onset of tuberculosis of the eye are certainly due in women to a diminution of the power of resistance during the climacteric and perhaps also to similar changes at the beginning of sexual maturity. The maximum for the males in the second and third decades may be caused by increased demands of the daily work which the youthful organism is not yet fully able to meet.

The view that iridocyclitis in elderly women is mostly of tuberculous origin, and not of a completely unknown causation, is nowadays, I believe, generally adopted in Central Europe, although the Austrian ophthalmologists for a long time would not accept it.

All tuberculous conditions of the inner parts of the eye are metastatic, arising from some other focus in the body. This focus in nearly all cases lies in the lungs or in the glands of the chest. Accordingly, the first intraocular attack often follows an attack of influenza or grip, by which an incompletely healed focus becomes mobilized and the bacteria reach the eye or some other organ. This stage of the illness corresponds to that of generalization and coincides with the second stage of Ranke¹¹⁰. It lasts many years.

Although it must be assumed that in all cases of intraocular tuberculosis there is an older focus in the chest which is still active or which has been reactivated, many authors have emphasized that even in cases of serious involvement of the eye disease cannot be found clinically in the lungs. There is especially no progressive focus in the lungs, and the patients seldom die in consequence of tuberculosis of the lungs. Some authors formerly suggested that when nothing is to be found in the lungs, the cause of iridocyclitis is probably tuberculosis¹. This opinion is certainly not justified, because when one examines the patients with roentgen rays some tuberculous lesions of the chest are usually found. It is certainly often impossible to say whether or not there is an active process in the lungs.

But Werdenberg,¹¹¹ of Davos, Switzerland, emphasized that if a thorough general examination is made evidence of tuberculosis is likely to be found in the chest. He found that in 60 per cent of more than 1,000 cases the condition in the chest was insignificant, but in 30 per cent it was serious. However, it must be remembered that the patients sent to Werdenberg in his sanatorium in Davos usually have a serious or extreme condition.

A correlation between the form of the disease in the chest and that in the eye could not be established. The majority of relapses are certainly caused by incompletely cured conditions in the chest.

Intraocular tuberculosis is, as I have shown, to be compared to the other metastatic forms in the body. Rollier¹¹² at Leysin, demonstrated that general surgical tuberculosis also is not associated with severe lesions in the lungs. Perhaps it is of interest that my assistant

110 Ranke, K. E. Primäraffekt, sekundäre, und tertiäre Stadien der Lungentuberkulose, *Deutsches Arch. f. klin. Med.* **119** 201, 1916.

111 Werdenberg, E. Zur Beurteilung und Behandlung der Augentuberkulose, *Klin. Monatsbl. f. Augenh.* (supp.) **94** 1, 1935.

112 Rollier, A. Die Heliotherapie der Tuberkulose mit besonderer Berücksichtigung ihrer chirurgischen Formen, ed. 2, Berlin, Julius Springer, 1924.

Dr Achermann during his investigation of 100 cases of tuberculosis of the lungs at Leysin several years ago found only 1 patient with ocular tuberculosis

The explanation of this difference between the state of the eye and that of the lungs is at present far from clear. There must be some special reason for this immunity, which at present is not known but which certainly depends, as already observed, on the general constitution of the body. Nevertheless, intraocular tuberculosis must always be treated not only by local medication but by general measures. A stay in the mountains for many months often brings about surprising results. I have seen this in many cases. The former, and even present day, use of tuberculin without definite indication should be avoided.

The importance of early complete healing of the local process in the eye is seen in table 10 (Achermann). It shows the correlation between the duration of the ocular disease and the diminution of the

TABLE 10—*Decrease of Visual Acuity with Increasing Duration of Tuberculous Disease of the Eye (After Achermann)*

Visual Acuity												
6/5	4	3	3	—	—	—	—	—	—	—	—	—
6/6	15	6	2	1	—	—	—	—	—	2(1)	—	—
6/8	10	2	2	1	—	—	—	1	—	(1)	—	—
6/12	9	5(1)	—	2	1	—	1	—	—	1	—	—
6/18	7	4(1)	2	1	1	—	—	—	—	2(1)	1	1
6/24	2(1)	—	2	1	—	—	—	1	—	1	1	1
6/36	1	—	—	—	—	—	1	—	—	1	—	1
6/60	1	3(2)	1	?	—	—	1	1	—	2	1	1
Counting fingers at 12 m	—	3(1)	3	4	—	4	2	—	—	4	3	7
Light sensation	—	—	—	1	4	1	1	3	2	1	1	7
Duration of the disease, yr	1	2	3	4	5	6	7	8	9	10	15	20

visual acuity in the cases in which relapses occurred. Good visual acuity is to be found chiefly in cases of involvement of less than four years' duration. The visual acuity is seen to decrease as the duration of the illness increases. This is due to the exudates formed by the inflammation and later often to complicated cataract. The table proves that by every means available complete cure of the condition in the eye must be attempted at its commencement.

Scrofula of the Eye—When one considers the relation between the local tuberculous condition in the eye and the primary focus in the chest, a certain degree of antagonism is found. In contrast to this fact are the conditions which are to be found in the cases of scrofula. This illness is also one involving the whole body. It manifests itself principally on the skin and on the surface of the eye (the conjunctiva and cornea).

Scrofula is at present regarded as a tuberculotoxic condition with a hypersensitiveness of the integument, caused by the toxins of the tubercle bacillus. Children especially are affected, apparently in con-

nection with a primary infection in the lungs. Therefore, the typical phenomenon of scrofula of the eye, the phlyctenule, is not due to the tubercle bacillus but is to be regarded as a specific reaction to the toxin. Admittedly the endogenous factor is not the only one, for exogenous factors, such as uncleanness, bodily strain and lack of fresh air, can all play their part in producing the condition.

In scrofula of the eye, a closer connection exists between the severity of the local focus in the eye and that in the chest than in true tuberculosis. Table 11, published by Stalder,¹¹³ shows the results in 100 cases of scrofula of the eye in which the state in the lungs was determined by roentgen examination in the General Polyclinic in Basel. In only 17 cases were there negative or uncertain findings in the chest. The examinations were made with the fluoroscope for reasons of economy,

TABLE 11—*Intrathoracic Condition in Patients with Scrofulous Inflammation of the Eye (After Stalder)*

Age in Years	Negative or Uncertain Diagnosis	Hilus		Condition of Lungs Without Physical Confirmation	Condition of Lungs with Physical Confirmation	Total Number	
		Condition of Hilus	With Calcification			Positive	Altogether
0-5	3	7	1	1	8	16	19
6-10	3	10	5	2	7	19	22
11-15	2	11	6	3	7	21	23
16-20	5	3	2	4	1	8	13
21-25	1	2	2	4	3	9	10
Over 25	3	0	3*	3	4	10	13
	17	33	19	17	30	83	100

* Without enlargement of hilus

but the same results would have been obtained had photographs been made.¹¹⁴

The patients with severe scrofulous conditions of the eyes are often found to have marked alterations in the lungs. A typical enlargement of the hilus was observed in children under 5 years of age. In patients more than 5 years old this was combined with calcified foci or was absent, indicating a tendency toward healing. After the sixteenth year the number of patients having lesions of the lungs increases. In the third decade and later the number of positive findings in the lungs diminishes.

Generally, one may say that scrofula of the eye is always associated with alterations in the chest of no small degree but apparently of no great malignancy. Therefore, some physicians have recommended mar-

¹¹³ Stalder, H. Zur Kenntnis der Lungenbefunde bei Augenskrophulose, *Schweiz med Wchnschr* 56 618, 1926

¹¹⁴ It may be mentioned that the dark-adapted eye when looking at the fluorescent screen has a very low visual acuity. When the light-adapted eye looks at photographs it can distinguish considerably more detail.

riage with women who had had scrofula in their childhood, because of the protection which this gives in later life against progressive tuberculosis of the lungs. This seems to be only partly justified.

DISEASES OF THE EYE IN RELATION TO WEATHER AND CLIMATE

The significance of climate or of definite influence of the weather on the incidence of some general diseases has been noted occasionally. But in recent years the interest in this problem has increased.

Among others, Hellpach,¹¹⁵ in Germany, studied the influence of the weather on man. In Switzerland, Fritzsche¹¹⁶ affirmed that under the influence of the foehn wind the frequency of apopleptic seizures, embolisms and thromboses increased. At Innsbruck, in Austria, for instance, where the foehn wind is very strong, the physical and mental energy of those who are sensitive to this phenomenon of the atmosphere is considerably diminished. Surgeons do not operate during this time unless it is absolutely necessary, because of the decreased resistance both of the patient and of the physician.

The typical foehn is a so-called falling wind (*Fallwind*), which approaches the heights as a warm wind from the south. On the ridge of the mountains it becomes colder and therefore heavier. It then descends rapidly and in doing so again becomes warmer. Violent storms, accompanied by manifold disturbances in the atmosphere are frequent in consequence.

The precise way in which the foehn affects the human body is still unknown. No opinions in regard to this problem have been confirmed.

The factors which change with the weather and which are ordinarily registered by the meteorologic stations are temperature, intensity and direction of the wind, clouds, precipitation and atmospheric pressure. Recently the degree of cooling (*Abkühlungsgrosse*) also has been recorded by some institutes, for instance, in Basel and Davos.

The degree of cooling takes account not only of the temperature but of all factors which cause loss of warmth to a small black sphere of metal which is heated electrically to 36.5 C. The sphere is placed, for instance, on the roof of a house. The amount of electric current necessary to keep the black sphere at a constant temperature of 36.5 C. is then measured.

Cooling depends on several factors: the temperature, the wind, the rate of emission of heat from the black sphere into the surrounding air and the absorption by it of heat from the sun. It is clear that the same factors which are of importance in such an example of cooling influence the living organism in a similar manner. Therefore, this index may be expected to be of greater biologic interest than that of temperature or of the wind alone.

115 Hellpach, W. *Geopsychische Erscheinungen*, ed. 3, Leipzig, Wilhelm Engelmann, 1923.

116 Fritzsche, E. *Witterung—Thrombose und Lungenembolie*, Schweiz. med. Wchnschr. 60: 889 and 1055, 1930.

Some authors have suggested that the free ions in the atmosphere also influence the human body, especially the vessels. But determinations made in the meteorologic laboratory at Davos, Switzerland, under the direction of Morikofer¹¹⁷ proved that the number of positive and negative ions, both large and small, changes rapidly in a room, for instance, without any influence on the human body. Therefore, this factor is probably not of importance.

I recently investigated the correlation between the state of the weather and the incidence of certain diseases in the ophthalmologic clinic in Basel. I obtained some interesting facts. The observations extend over a period of nine years, from 1926 to 1934.

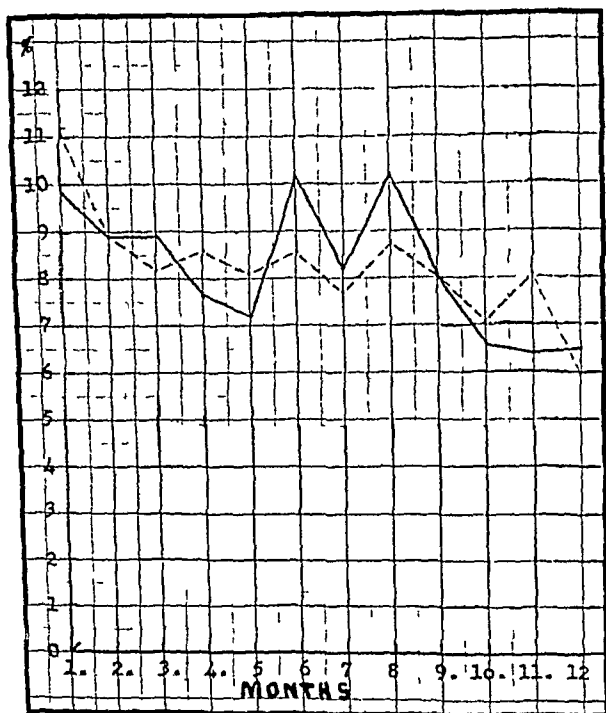


Fig. 59—Hordeolum from 1926 to 1934. Frequency in percentage for a month of thirty days. The dash line indicates the frequency curve for the polyclinic.

Hordeolum—I shall refer briefly to hordeolum. I have not distinguished here between disease of the large meibomian glands and that of the small Zeiss glands in the outer surface of the lid at the seat of the eyelashes. These glands have almost the same function and are also generally exposed to the same noxious conditions.

In regard to the fluctuation in the frequency of this disease in relation to the seasons, figure 59 shows that there are three periods of incidence, namely, in January, June and August. A minimal number of cases are to be found in May and in the last three months of the year.

¹¹⁷ Morikofer, W., and Chorus. Ergebnisse von Ionenmessungen in Davos, Verhandl. d. Schweiz. naturforsch. Gesellsch., 1932, p. 324.

That this increased frequency is not dependent on the number of patients attending the polyclinic at Basel is easily seen from the diagram in figure 59. Therefore, the variation in frequency of this disease must be significant, all the more so as the number of cases (about 1,000) seems sufficient for conclusions to be drawn.

On correlation of the factors concerning the weather with the onset of hordeolum in each case, an undoubted dependence on temperature was found, especially as regards fall in temperature or a high degree of cooling. Figures for the degree of cooling are available in Basel only since 1929. Table 12 shows that in two thirds of all cases of hordeolum the condition is related in onset to a fall in temperature and an increase in cooling rate. The fall in temperature occurred one or two, seldom three, days before the appearance of the sty. This

TABLE 12—*Hordeolum, Dependence on Lowering of Temperature and Increase in Degree of Cooling*

	Temperature			Cooling			
	+	—	Total	+	—	Total	
1929	62	34	96	65	31	96	
1930	88	47	135	99	36	135	
1931	81	45	126	94	32	126	
1932	82	44	126	82	44	126	
1933	91	43	134	83	51	134	
1934	81	45	126	84	42	126	
Total	485	258	743	507	236	743	
Increase of degree of cooling 1929 1934				507	236	= 68 2%	31 8%
Decrease of temperature 1929 1934				485	258	= 63 9%	34 7%
Decrease of temperature 1926 1934				698	347	= 66 9%	33 2%
				(1,045)			

time, as is to be expected, corresponds to the period of incubation. Considering the large number of cases, coincidence can be ruled out. In my opinion, the skin of the eyelids, being exposed to the influence of weather, is cooled and consequently has a lowered resistance to the bacteria, especially staphylococci, which are present in the skin. The lowering of the resistance of the tissues permits the onset of inflammation. This explanation accords with the opinion, often expressed by the patient, that he had been chilled before the hordeolum appeared.

It is to be noticed that long periods of cold do not increase the number of cases. It is only in the beginning of the period that cases become more frequent, especially when the cold sets in after a period of warm weather.

It seems to me that this fact proves that the skin and its organs were previously adapted to a higher temperature and are not able to accommodate themselves quickly enough to the lower temperature.

But there remain about one third of all cases which cannot be explained in this way. Here other influences may be at work. It is

especially noticeable that during the summer an extremely high temperature frequently coincides with an outbreak of hordeolum. This may be due to the patients' having exposed themselves to a higher degree of cooling, say in a draught, with the intention of seeking relief from the heat. The onset of the hordeolum may be explained in the same way as before. But it seems more probable that copious perspiration of the face leads to a rubbing or touching of the lids with the fingers or pocket handkerchief and a consequent infection of the glands. This suggestion would be in accord with the fact that many persons have general inflammation of the follicles in the skin after perspiration.

Catarrhal Ulcer of the Cornea—Conditions similar to those which favor the occurrence of hordeolum also accompany the onset of the so-called marginal or catarrhal ulcer of the cornea. This small ulcer, which lies from 0.5 to 1 mm. from the margin of the cornea, is benign but tends to relapse for some years afterward. It occurs mostly in middle-aged or elderly persons. The relapses suggest that some constitutional factor may be at work.

It was noticed that often during the summer when a few cold days occurred suddenly this disease was fairly frequently encountered. The statistical treatment of about 300 cases confirms these observations, because the highest frequency is to be found from July to October, while during the winter it is relatively low. There are two minima, one during May and June, and the second in November and December (fig. 60).

Consideration of the meteorologic factors has made it possible to establish here too a definite connection between a fall in temperature or an increase in the degree of cooling and the onset of the ulcer. There is therefore a strict positive correlation between the beginning of this ulcer and the state of the weather. In about three fourths of the cases there is such a connection (table 13).

This relation is especially clear in the summer months. It may be explained by the absence of vessels in the cornea and the lower temperature of that membrane in relation to the rest of the body. When the cornea during the warm season has been adapted to a high temperature, cool weather causes an exaggerated loss of warmth and thereby diminishes its resistance. Since there are no specific bacteria which cause the ulcer, a constitutional factor must certainly be responsible, of what nature it is difficult to say. Perhaps defective adaptation of the cornea to extreme temperatures is due to an insufficient regulation of the sensitiveness of the vessels in the immediate neighborhood of the cornea. It would be of interest to determine whether these patients show any similar alteration of the general vessels. The method of Otfried Muller, the so-called microscopy of the capillaries, might be useful for this purpose.

Acute Conjunctivitis—In contrast to these ocular diseases, investigations concerning acute conjunctivitis gave other results. This disease is certainly in some cases due to local cooling, it is, for instance, often observed after a person has driven a motorcar with open windows, so that a draught of air struck the eye. Since in cases of acute conjunctivitis pneumococci are usually found, it is justifiable to suggest that the damage to the anterior part of the eye facilitated attack by the bacteria through diminishing the defensive power of the organ.

But examination of the cases of acute conjunctivitis for nine years shows no strict dependence on the temperature, although there is a

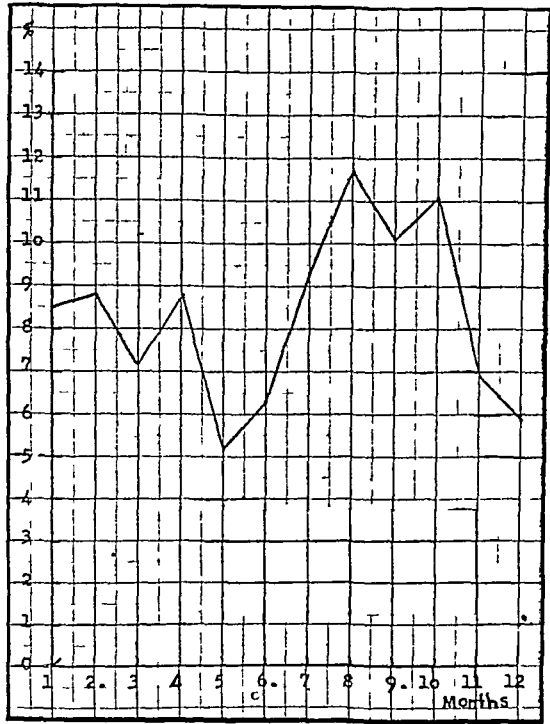


Fig. 60—Ulcus catarrhale from 1926 to 1934. Monthly frequency in percentage.

TABLE 13—Ulcus Catarrhale, Dependence on Lowering of Temperature and Increase in Degree of Cooling

	Temperature			Cooling			
	+	—	Total	+	—	Total	
1929	30	15	45	33	12	45	
1930	25	6	31	23	8	31	
1931	34	2	36	27	9	36	
1932	27	6	33	21	12	33	
1933	28	4	32	28	4	32	
1934	27	11	38	30	8	38	
Total	171	44	215	162	53	215	
Increase of degree of cooling 1929-1934				162	53	= 75.3%	24.7%
Decrease of temperature 1929-1934				171	44	= 79.5%	20.5%
Decrease of temperature 1926-1934				238	68	= 77.9%	22.2%

(306)

summit of incidence in May and another in October. The latter could be taken as produced by a fall of temperature. But closer consideration does not support this suggestion. On the contrary, about a half or two thirds of the cases have no relation to the temperature or the degree of cooling. I think it must be concluded that the onset of acute conjunctivitis is determined by different factors, partly bacteriologic and partly of other kinds. It may be especially stated that the cases mentioned do not comprise instances of severe infections, such as diphtheria, gonorrhea and inflammation with the so-called inclusion bodies (*Einschlusskörperchen*), namely, trachoma and swimming pool conjunctivitis.

This negative result seems to me to prove that the dependence of the two previously mentioned diseases on the weather is real.

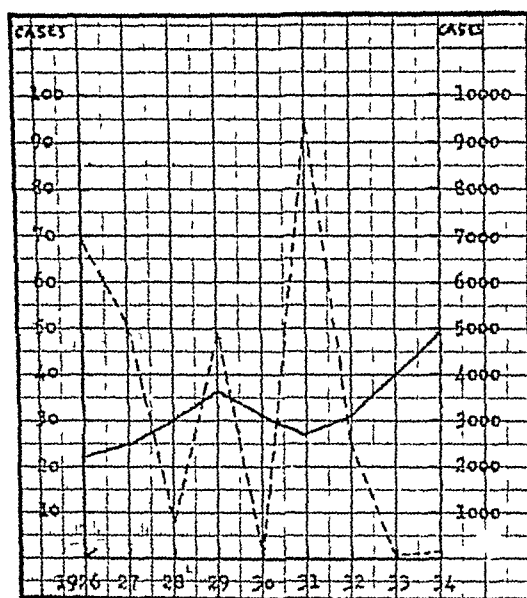


Fig 61—Relation of the frequency of herpes corneae to the frequency of influenza. The continuous line indicates cases of herpes, the dotted line, cases of influenza.

Herpes Corneae—Another ocular disease which probably depends on the weather is herpes corneae, the eruption of small vesicles on the surface of the cornea, which frequently is followed by an extension to the deeper parts of the membrane (keratitis disciformis). Although it was learned from the experiments of Gruter and Doir that the condition must be caused by an infective ultramicroscopic virus, it is conceivable that the weather may influence the frequency, for instance, by increasing the disposition to the disease. However, a more detailed investigation demonstrated no relation to the temperature in general. Certainly in some cases a cold may be the cause.

The statement in textbooks that herpes corneae follows influenza hardly seems to be justified by statistics. Figure 61 shows the frequency

of the cases of herpes observed in the clinic in Basel and the number of cases of influenza reported to the sanitary department in Basel during a period of nine years. There is no correlation. This proves, as some authors have believed, that the causes of influenza and herpes corneae, at present unknown, are not identical. However, there may be some relation.

Hinrichs,¹¹⁸ of Greifswald, after investigations extending over a period of only two years expressed the opinion that the weather, especially "the fronts," has an influence on the appearance of herpes. The results of his investigation do not seem to me to be sufficiently conclusive.

Scrofula of the Eye—Of more interest is the relation of scrofula of the eye to the seasons. It has been seen that this disease is due to a tuberculous infection and that the manifestations on the skin and in the eye are not really tuberculous, but only toxic. The incidence of this

TABLE 14—*Scrofulous Conjunctival and Corneal Conditions*

Years	Number of Cases	Total Number of Outdoor Patients
1926	191	4,754
1927	155	4,837
1928	193	5,225
1929	254	5,589
1930	192	5,642
1931	190	6,000
1932	195	7,061
1933	261	6,670
1934	151	6,796
Total	1,782	52,574

disease has greatly diminished in all countries of middle Europe during the last fifteen years, perhaps in connection with the improved hygienic conditions of the dwellings and with better nourishment. Nevertheless, in Basel a change in its frequency has been observed during recent years which is by no means explained (table 14). A relation to the change of the weather from year to year is not established. But the dependence on the season is remarkable (fig 62). An increase of the cases in the first half of the year, especially from March to June, is followed by a decrease during the last months of the year. It is due not only to a decrease in the number of patients in general, but apparently to the influence of the seasons, which effects a change in the resistance of the body.

Nearly the same course of the curve was to be found in Palermo, Graz, Greifswald and Helsingfors. The summit varies according to

¹¹⁸ Hinrichs, H. Einfluss von Wetter und Jahreszeit auf die Entstehung von Augenkrankheiten, Ztschr f Augenh 86 269, 1935

the geographic situation of the town, whether it is in the south or in the north. There must be a common cause which affects the frequency, dependent on the seasons (Rohrschneider¹¹⁹). A similar distribution during the different seasons was also found by Essen-Møller in Lund¹²⁰.

I suggest that the cause is to be found in the favorable effect of the air and sun in summer on the tuberculous process in the chest. The diminution of the tuberculous toxins produced reduces the frequency of scrofulous manifestations. On the contrary, the dark winter, with the small amount of sunshine and confinement to the house, diminishes the resistance. These influences last until the late spring. Rohrs-

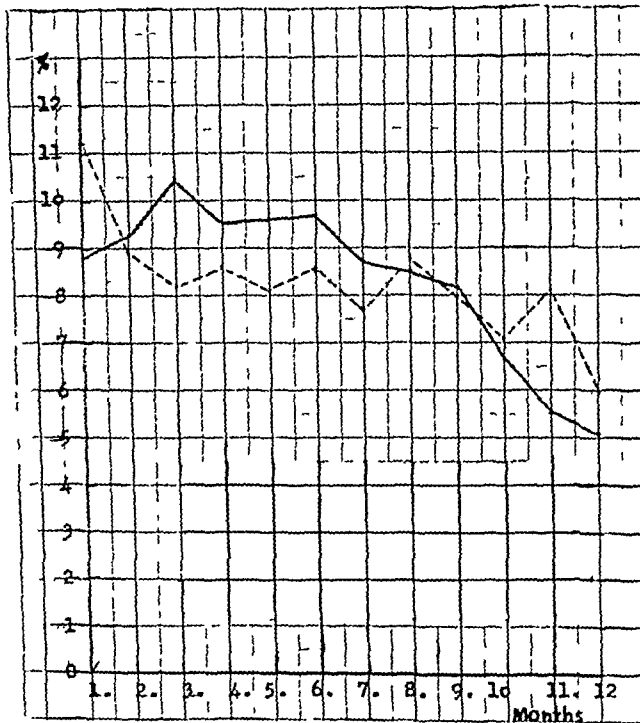


Fig. 62—The continuous line indicates scrofula from 1926 to 1934 (frequency in percentage for a month of thirty days). The dash line indicates the frequency curve for the policlinic.

schneider¹¹⁹ expressed the same opinion. One might believe that the increasing insolation in the spring excites the efflorescence of the scrofula in the eye. Occasionally after strong doses of artificial sunlight (the mercury lamp) an increase of recent scrofulous phenomena is seen in the eye. Perhaps both causes are effective. In considering the

119 Rohrschneider, W. Ueber den Frühjahrsgipfel der skrofulösen Augenerkrankungen, *Ztschr. f. Augenh.* 86:281, 1935.

120 Essen-Møller, L. Eine Studie über die Häufigkeit der Phlyctänen, *Acta ophth.* 14:414, 1936.

decrease of the resistance during the winter, account must also be taken of the lack of fresh vegetables and the consequent lack of vitamins

These questions are still theoretically unsolved, but the practical consequences are simple. If the relapses so frequent in this disease are to be avoided, scrofulous patients should go to the high mountains during the second half of the winter, thus they will escape the mist and the fog in the plains. If the financial situation does not allow this, a thorough treatment by artificial sunlight is to be recommended. Indeed, after this treatment I have observed good results even in serious involvement, especially in the absence of relapses.

Acute Glaucoma—Finally, of great interest is the relation of attacks of acute glaucoma to the weather. In the beginning of this century Steindorff¹²¹ called attention to the fact that such attacks were more frequent in the winter than in the summer. He stated that this was due to the cooling of the body, which produces a disturbance in the regulation of the flow of the blood. Other authors said that the darkness in winter dilates the pupil, which increases the chances of an attack. There is also the fact that economic cares, which are greater in winter than in summer, increase the disposition to an acute attack from alterations in the blood vessels.

At present there is no certain knowledge about the mechanism of acute glaucoma. That the season has great influence on the frequency is shown in figure 63. It clearly demonstrates that from November to February this disease is much more frequent than in the summer.

The frequency changes also with the years in general (fig. 64). Especially in the last few years the number of cases of acute glaucoma observed in Basel has increased. Whether social factors caused by the economic crisis operate here is still uncertain.

De Rudder¹²² emphasized some years ago that acute glaucoma occurs especially at the passing of the so-called cool and warm fronts. Modern meteorology teaches that the antagonism of the warm tropical and the cold polar air is of the greatest importance to the general type of the weather. These bodies of air (*Luftkörper*) are, it seems to me, of interest to medical science also. De Rudder suggested that no special factor accompanying the change of the body of air is effective, but all factors combined. Especially it is not the pressure of the air which has an influence in producing disease.

Dr Bider, of the Meteorological Institute in Basel, helped me to evaluate cases of acute glaucoma with regard to the weather. In 150 cases we were able to fix the time of the beginning of the attack exactly. As shown in table 15, in 87 cases the onset coincided with the passage

121 Steindorff, K. Ueber den Einfluss von Temperatur und Jahreszeit auf den Ausbruch des akuten primären Glaukomanfalles, *Deutsche med. Wchnschr.* 28:924, 1902.

122 de Rudder, B. Wetter und Jahreszeit als Krankheitsfaktoren. *Grundriss einer Meteoropathologie des Menschen*, Berlin, Julius Springer, 1931.

of a warm or cold front Only 17 cases were observed with foehn and 19 with severe cold, and only 11 of 150 cases occurred without any remarkable disturbance in the atmosphere

The weather is certainly to be considered as influencing the flow of blood by producing decompensation Acute glaucoma is in my opinion



Fig 63—Acute attacks of glaucoma Curve of the totals for the months compiled for the years 1920 to 1934

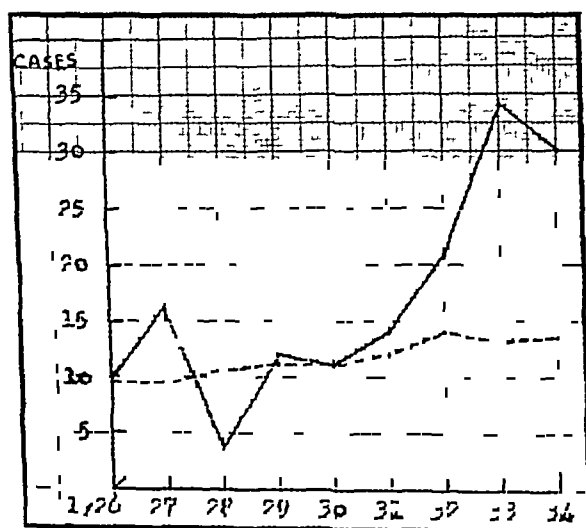


Fig 64—Glaucoma Curve of the totals for the years 1926 to 1934 The dash line indicates the frequency curve for the polyclinic

related to an insufficiency of the vessels, although in what manner is not known

It may be of interest in this connection to note the following observations made on 6 patients attacked by different illnesses at the same time, from Oct 26 to 28, 1935, during the passage of an extreme warm front All these illnesses, ophthalmic migraine, apoplexy, extrasystoles, and so on, result from disturbances of the vessels, which must have a common cause

On October 26 the air on the continent was cold, while warm air was to be found above the ocean. On October 27 a definite warm front passed across the continent and reached Basel in the evening, a storm from the northwest arose there, accompanied by very warm air. Next day the front disappeared and heavy rain fell.

It is not known what factor associated with the movement of the air bodies is effective. I have recently begun investigations with a physicist and a physiologist to try to solve this complicated and important problem. Nevertheless, some practical conclusions may be deduced from the facts already known. All patients suffering from simple

TABLE 15—*Acute Attacks of Glaucoma in the Years 1926 to 1936*

Meteorologic Condition	Number of Attacks
Cold front	42
Warm front	45
Foehn	17
Cold without front	19
Snow	2
Other disturbances	14
No disturbances	11
Total	150

TABLE 16—*Influences of a Sudden Approach of a Warm Front on the Vasomotor System During the Period from Oct. 26 to 28, 1935*

- 1 Attack of acute glaucoma in an elderly man during the night of October 27, intense pains at 3 o'clock in the morning
- 2 Woman of 75, several years ago acute attacks of glaucoma, at present regularly taking pilocarpine, on October 27 typical headache on the right side, passing without attack, apparently a slight increase of the intraocular pressure
- 3 Physician, hemicranial migraine from October 27 to 28
- 4 Physician, suffering from habitual asthma, during night of October 27 serious attack of asthma
- 5 Elderly physician, on October 27, extrasystoles and irregularities of pulsation
- 6 Woman nearly 70 on October 27 sudden diminution of the vision, occasioned by thrombosis in a branch of the retinal vessels

glaucoma should, in order to minimize the danger of an acute attack, use more pilocarpine when the weather changes, especially when a "front" approaches. This can readily be ascertained from the meteorologic data published daily by the newspapers.

In general, it may be emphasized that this sensitiveness to the weather or to several meteorologic factors, which have been established for glaucoma, is caused by an individual insufficiency in the regulation of some parts or functions of the body. For those who are not sensitive, the weather does not matter. The sensitiveness perhaps changes with increasing years. I know many inhabitants of Basel, which in this respect has not a good climate, who have become more and more sensitive to the weather. Probably the faculty of adaptation of the blood vessels diminishes with their increasing rigidity.

SUMMARY

As in all other respects, so also from the special standpoint of the ophthalmologist, different persons show different reactions to normal and abnormal stimuli. Adaptation of the living substance is possible within a wide range. In relation to adequate stimuli, the visual organ is of an individual variability which is sometimes so extreme, for instance in cases of congenital color blindness, that a mutual understanding between abnormal and normal persons is impossible.

Adaptation to low intensities of light also shows considerable individual variability, so that persons with extreme degrees of insufficiency can exist only by the protection which human civilization affords.

In his environment, in a narrow sense, the individual changes by adapting himself to the given circumstances, but there are remarkable differences in reaction to some definite meteorologic changes. Human civilization allows man to protect himself against the inclemency of the weather, but this defense is only relative. There are some factors against which the power of adaptation of many persons is insufficient.

Also in tuberculous infection of the eye, which I have mentioned, there are a variety of reactions according to the hereditary or acquired constitution of the individual which led, in spite of the ordinarily equally infective virus, to different courses of the illness.

The human organism and one of its organs, the eye, react in a most variable manner. This fact teaches how surprisingly variable human life is, even in this small province.

For the purpose of classification, be it in a scientific, social or political sense, this variation in life may be disturbing or inconvenient. Therefore, it has often been overlooked or intentionally neglected. But each organism is and always will be an individual.

SIMPLE DACRYOCYSTORHINOSTOMY

LOREN GUY, M D

NEW YORK

The birth of the idea of the dacryocystorhinostomy occurred when the lacrimal sac and ducts were shown to be conducting structures and not secreting organs. Antoine Maître-Jan¹ appears to have been the first to have recognized the true anatomic structure and physiologic function of the lacrimal duct and sac. His work formed the foundation of rational therapy of the part. Before his time it was believed that these structures were glands.

The first surgeon to take advantage of the basic physiologic and anatomic knowledge of the lacrimal structures was the Englishman Woolhouse. Woolhouse himself left no known description of his method of operating, but his technic is mentioned by Platner² in an article published early in the eighteenth century. Metal cannulas and frequent irrigations were employed in the earlier technic.

Modern work began with the contributions of the Florentine rhinologist Toti,³ in 1904. All subsequent work has been along the lines set down by Toti. Following him, the work has consisted mostly in individual preferences in method and in the introduction of special instruments.⁴

West⁵ and Halle^{5a} have devised an intranasal operation which appears well suited to one trained in intranasal work. It has the advantage of eliminating the possibility of a visible scar.

From the New York Eye and Ear Infirmary

Read at the New York Academy of Medicine, Section of Ophthalmology, Jan 18, 1937

1 Maître-Jan, A. *Traite des maladies de l'oeil et des remedes propres pour leur guerison*, Troyes, Jacques le Febvre, 1707

2 Platner, J. Z. *De fistula lacrymali*, respondente, Henrice Godofredi Heyland, Lipsiae, lit. I. Titu, 1724

3 Toti, A. *La dacriocistorinostomia come cura radicale conservatrice delle suppurazioni gravi e delle fistole del sacco lacrimale. Risultati ottenuti nei primi sette casi operati*, Policlinico (sez. chir.) **11** 545, 1904

4 Weeks, W. W. *The Technic of Dacryocystorhinostomy*, Arch. Ophth. **7** 443 (March) 1932. Dupuy-Dutemps and Bourguet. *Procede plastique de dacryocysto-rhinostomie et ses resultats*, Ann. d'ocul. **158** 241, 1921

5 West, J. M. *The Clinical Results of the Intranasal Tear Sac Operation*, Tr. Sect. Ophth., A. M. A., 1931, p. 69

5a Halle, M. *Intranasale Tranensackoperation bei Sauglingen*, Med. Klin. **24** 1699, 1928

With the objective of developing a method of doing a dacryocystorhinostomy that would be effective and yet simple, work was started on cadavers four years ago. Three years ago the operation was applied to living subjects. A small group of patients was carefully studied and such changes were made in the method as the results indicated. In doing this work, Dr. Francis Shine, Dr. Clyde McDannald and Dr. Hugh Blackwell have been most helpful.

It has been concluded that the operation is indicated in cases of chronic dacryocystitis in adults. So many children respond to non-surgical treatment that it should be deferred, if possible. Owing to its effectiveness in eliminating epiphora and infection in upward of 85 per cent of cases, dacryocystorhinostomy is preferable to extirpation of the sac. In cases in which the operation has not been a success, no more serious damage has been done other than the inconvenience of a minor local operation.

The operation should not be done in cases in which an intraocular operation is likely to be necessary. The dangers of an ascending infection from the nasal cavity appear too great. Obstruction in the canaliculi and in the nasal passages might render the operation useless. These structures should be examined before the operation is undertaken, and any polyps, sinus disease or anatomic malformations should be corrected. The operation should not be done in the presence of acute infection.

The technic as finally accepted is as follows:

Anesthesia is obtained by infiltrating the area with a 2 per cent solution of procaine hydrochloride, 8 parts, and epinephrine hydrochloride in a dilution of 1:1,000, 2 parts. Intranasal application of cocaine hydrochloride was found not to be necessary. Depending on the temperament of the patient, a preoperative sedative is given, usually $\frac{1}{8}$ grain (0.008 Gm.) of morphine sulfate.

Before the incision in the skin is made, the angular vessels are located by palpation, and by avoiding them much troublesome bleeding is eliminated. The incision, *A* in the illustration, is made from a point 3 mm. above the inner canthal ligament downward and outward just nasal to the anterior lacrimal crest. The length depends on the size of the sac and the anatomy of the parts, but averages 20 mm. The incision is made down to the bone through the periorbital skin. The periorbital skin is then elevated temporally, exposing the lacrimal fossa and the sac. In the lower half of the fossa a window about 4 by 6 mm. is made. A chisel, a pair of scissors or a curet is used to make this window. Such an instrument allows for anatomic variation and seems to be more desirable than a fixed trephine or other similar instrument.

The lower half of the fossa is emphasized because the ethmoid cells are rarely, if ever, found, according to Whitnall. While it does not appear to make much difference whether or not these cells are entered, it would seem best not to disturb them.

After the window is cut through the bone of the fossa, the corresponding mucous membrane is excised, and then the corresponding portion of the sac is also excised. The tissues are allowed to fall back into their natural positions,

the excised portions coming in apposition, thus forming a new line of communication from the conjunctival sac to the middle meatus of the nose. The tissues are held in place by a pressure bandage. No sutures are used except for the skin. Fine interrupted sutures are taken there. The insertion of sutures to hold the mucous membrane of the nose and sac in place takes considerable time and, in addition, such sutures do not seem necessary. It is doubtful if the introduction of a foreign material, such as suture material, in inflamed tissues, such as chronic dacryocystitis presents, is desirable.

On the second day after the operation the pressure bandage is replaced by a dressing of collodion. The skin sutures are removed on the fourth or fifth day. It requires less than thirty minutes to perform the average operation.

In the series of cases in which this operation was performed no complications have thus far been encountered. In the literature severe

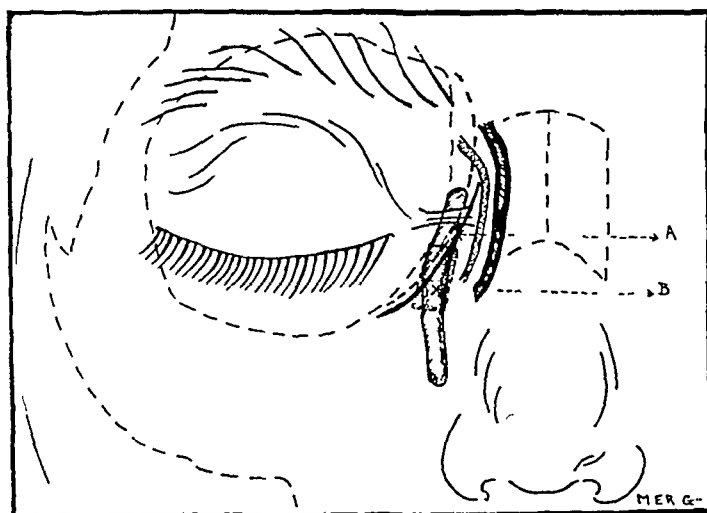


Diagram illustrating the simple dacryocystorhinostomy operation. *A* indicates the incision and *B*, the window cut in the lower half of the lacrimal fossa. Note the relation of the angular vessels.

hemorrhage from severing the nasociliary artery has been reported. For this reason it might be desirable to be prepared for such an emergency. Hematoma of the orbit and infection have also been reported, occurring with about the same frequency as with extirpation of the sac.

To date, 54 patients have been operated on by seven different operators. Only one of the operators had had previous experience with any type of dacryocystorhinostomy. Fifty of the patients were relieved of both epiphora and empyema of the sac. One failure was due to faulty technic, and one resulted from nasal obstruction due to neglected sinus disease. The failures occurred in the first half of the series.

It is possible to tell by the fifth day whether the operation is successful. All patients in whom communication has been established

by that time have maintained it up to the present, a period in some instances of over three years

From these observations it appears that in selected cases of chronic dacryocystitis, dacryocystorhinostomy is indicated because in a worth while number it will relieve epiphora and infection. If a few simple rules are observed, the operation is easy to perform, taking on an average of less than one-half hour. It can be done by the average ocular surgeon with the instruments found in the ordinary ophthalmic operating room.

570 Park Avenue

ANGIOMATOSIS RETINAE

REPORT OF A CASE WITH A PATHOLOGIC STUDY OF THE ENUCLEATED EYE

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Angiomatosis retinae was described as early as 1882 by Fuchs¹ and subsequently by several other investigators, but it was von Hippel² in 1904 who first recognized it as a clinical entity. Since then his name has been associated with the disease. Scarlett³ reported the first case in this country in 1925. Cushing and Bailey⁴ and Bedell⁵ have subsequently reviewed the literature, but studies on an enucleated eye employing differential staining technics have not been recorded in the American literature and rarely in the literature of other countries. Because of this fact and the rarity of the condition, it has been considered worth while to report a case.

REPORT OF A CASE

A boy aged 15 years was first seen and admitted to the Wills Hospital, to the service of Dr Frank C Parker on May 24, 1937. His parents stated that when he was 5 years of age he had been hit above the right eye with a stone. Shortly after this he lost all useful vision in that eye. When 11 years old he became aware of failing vision in his left eye. A local oculist prescribed glasses, and the patient carried on for four years. His vision became rapidly worse in August 1936, and he was admitted to the Mercy Hospital in Pittsburgh the same month. It was learned, while preparing this report, that the condition was diagnosed as angiomatosis retinae at that time by Dr Rhodes and that the case was presented before the Pittsburgh Ophthalmological Society⁶. After the child's discharge from the Mercy Hospital, his vision became progressively worse, decreas-

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1 Fuchs, E. Aneurysma arterio-venosum retinae, Arch f Augenh **11** 440, 1882

2 von Hippel, E. Ueber eine sehr seltene Erkrankung der Netzhaut, Arch f Ophth **59** 83, 1904

3 Scarlett, H. Angiomatosis of the Retina, Arch Ophth **54** 183 (March) 1925

4 Cushing, H, and Bailey, P. Hemangiomas of the Cerebellum and Retina (Lindau's Disease), with Report of a Case, Arch Ophth **57** 447 (Sept) 1928

5 Bedell, A J. Angiomatosis Retinae, Am J Ophth **14** 389 1931

6 Rhodes, D H. Angiomatosis Retinae, Am J Ophth **20** 522, 1937

ing from 20/80 to perception of light on his admission to the Wills Hospital. The remainder of his personal and family histories, especially with regard to visual disturbances, was found to be unimportant.

Physical Examination—The results of the physical examination were essentially negative, but the following findings are recorded because of their bearing on the case. The skin and mucous membranes were normal, with no evidence of nevi, papillomas or nevus flammeus. The neurologic examination did not reveal any evidence of organic disease. The spleen and kidneys were not palpable. The laboratory tests showed 4,590,000 red blood cells and 7,300 white blood cells (46 per cent neutrophils, 48 per cent basophils, 5 per cent eosinophils and 1 per cent monocytes), a coagulation time of three minutes, a negative Wassermann reaction of the blood, a negative reaction to the Mantoux test with purified protein derivative (0.000001 and 0.001 mg) and normal urine. A roentgenogram of the right eye did not reveal any opaque foreign body.

Ocular Examination—*Right Eye*. At the junction of the outer and the inner third of the eyebrow there was a small scar, the site of the wound from the stone. The lids, cilia, lacrimal passages, conjunctiva and sclera were normal. The eye was divergent about 20 degrees. The cornea was clear, the corneal reflex was not impaired. There was no evidence of any old penetrating wound. The anterior chamber was of normal depth, the iris was atrophic and was bound down to the anterior capsule of the lens, which was opaque. Examination with the slit lamp showed a few fine pigment deposits on the posterior surface of the cornea. The aqueous was clear. The iris was markedly thinned, and many new blood vessels were visible in the stroma, some of them could be seen to anastomose on the thickened anterior capsule of the lens. Transillumination of the eye was equal in all meridians and showed a red reflex in the pupillary space. The tension was 18 mm (Schiotz). Vision was nil.

Left Eye. The lids, cilia and lacrimal passages were normal, and the external ocular movements were full. The conjunctiva and sclera were normal. The cornea was clear, with a normal sensory reflex. The anterior chamber was of normal depth. The pupil was round and reacted to light. The lens was clear. The media showed a faint haze, but there were no large opacities. The fundus presented a striking and bizarre picture. Most impressive were the changes in the vascular system. The disk could not be seen, but just above the posterior pole an enormously dilated vein was visible in the upper temporal quadrant. It coursed in a serpentine fashion over the many folds of the detached retina, the displacement of which forward increased temporally and below. The vein ended abruptly in a large reddish cyst in the extreme periphery about 1 disk diameter above the horizontal meridian. The vein was followed above by a similar dilated vessel of slightly smaller size, it was hidden in part of its course by the many folds of the retina, but it could be seen to enter the cyst just above the vein. There were two other cystlike dilatations, one above about 1 o'clock and another below at 7 o'clock. The artery in the latter could not be seen among the retinal folds.

The vessels, especially those communicating with the cysts, were enormously dilated and tortuous and had a peculiar reddish reflex which gave the impression that they were about to burst. The entire temporal and lower half of the retina was detached, the detachment being uneven, with many undulating folds. Its color varied from red to yellow, and in places it was of a greenish hue. No normal area was seen in the fundus. A small sector of retina above appeared to be attached with normal vessels, but the retina was yellow and atrophic. No areas of choroid or any choroidal vessels were visible. Examination with the slit

lamp showed the cornea to be clear, the aqueous to contain a few fine pigment granules and the iris to be normal. The lens was clear, and the vitreous contained many fine floating opacities.

The tension was 20 mm (Schiotz). Vision was limited to perception of light, with projection limited to the nasal field.

The patient was hospitalized for five weeks while these procedures were carried out. He was given 1,405 mg hours of radium over the left orbit, four treatments one week apart being given. There was no apparent change after the radium therapy. This therapy was instituted because it had been recommended by Bedell⁷ and others but with the belief that this was an inherently benign tumor and the treatment would therefore be of no avail.

The patient was shown to several examining members of the American Board of Ophthalmology, and all but two concurred with our diagnosis of angiomatosis.



Fig 1—Photograph of a painting of the fundus of the left eye showing the cyst-like dilatations in the periphery, the dilated vessels and detachment of the retina.

retinae. The case aroused considerable discussion, but to exclude the possibility of a malignant tumor of the blind right eye, the latter was enucleated on June 14. The patient was discharged from the hospital on July 3.

Macroscopic Report on the Right Eye—The globe was of normal size. When it was sectioned, the anterior chamber was found to be deep and almost completely filled with a serofibrinous exudate. There were an occlusion and seclusion of the pupil, with an accompanying iris bombé. The lens showed cataractous changes, with many calcareous deposits, and was surrounded posteriorly by an

⁷ Bedell, A. J., in Berens, C. *The Eye and Its Diseases*, Philadelphia, W. B. Saunders Company, 1936, p. 779.

organized exudate, which had caused complete detachment of the retina. The subretinal space occupying almost two thirds of the posterior chamber was filled with a serous exudate.

Microscopic Report on the Right Eye—The cornea showed practically no change except at the limbus, where there were many new blood vessels and a moderate degree of round cell infiltration. There was some degeneration of the endothelium. The anterior chamber was deep, and at the filtration angle there was a complete anterior synechia. The canal of Schlemm was completely obliterated. The pupillary space was filled with an organized exudate, there was seclusion of the iris by posterior synechia, and the iris was pushed forward into the anterior chamber, producing iris bombé.

The iris and ciliary body showed loss of cellular structure, with fibrous tissue proliferation and many newly formed blood vessels. The pigment layer, though atrophic, was intact. The posterior chamber was deep and filled with a sero-fibrinous exudate.

The lens was surrounded by a fibrinous exudate and showed advanced hyaline and calcareous changes. The entire retina was detached and extended from the posterior surface of the lens to the optic nerve as a V-shaped band. Anteriorly it merged with the atrophic ciliary processes by many strands of connective tissue. The normal retinal structure had been replaced by the tumor. The tumor lay in this stalk of tissue extending from the posterior pole to the lens, and a large dilated vessel could be seen extending almost its entire length. Many large cyst-like dilatations could be seen lined by endothelium, some of them contained red blood cells. The whole structure was permeated by thin strands of connective tissue and many newly formed blood vessels.

The choroid had been almost completely replaced by fibrous tissue, but a few dilated blood vessels were visible posteriorly. Anteriorly the choriocapillaris was barely distinguishable, and there was some round cell infiltration. On both sides of the nerve head there were areas of bone formation lying in the band of fibrous tissue that had replaced the choroid.

The sclera appeared normal. There was some dipping of the lamina cribrosa, but the nerve head itself did not show any change. The intervaginal space was moderately well filled.

Microscopic Report on the Tumor of the Right Eye—The section of tumor stained with hematoxylin and eosin did not present a uniform appearance. The matrix was cellular, and under low power magnification this background, which was chiefly glial, was most prominent. It appeared to form the major part of the tumor. The Bielschowsky stain accentuated the glial framework, the cells and fibers of which stained deep gray while the reticulum appeared as a fine black webbing sparsely distributed. Similarly, with the Holzer stain glial tissue was recognized by the deep blue-staining fibers. These fibers occasionally formed a dense network, although individual, smaller wavy fibrils could be seen arising from one or both poles of elongated and deeply nucleated cells. While the glial tissue formed the greater part of the background, it assumed no definite arrangement. It was compact or loosely arranged in whorls. These whorls, however, should not be mistaken for rosette formations, for the latter do not occur, nor does actual palisading of cells as is seen in tumors of neurogenic origin.

In addition to the typical and odd monster glial cells and fibers, there were other far less numerous and conspicuous cellular elements. These could be seen to advantage by the toluidine blue method of staining, which sharply delineated them. Slender, flat endothelial cells could be seen, and in one focal area they

appeared in a bizarre pattern of crosses and chains of closely packed cells. In the less dense cellular areas, ovoid, vesicular nucleated cells were found. Polygonal cells with central nuclei were seen. Occurring but rarely were ganglion cells, some of which were degenerating, and mast cells with red granules in their cytoplasm. Isolated peripherally was a group of cholesterol crystals with foreign body giant cells about them, but no pseudoxanthoma cells were noted. Blood pigment and retinal pigment were observed in small deposits.

Even though these cellular components seemed to constitute the greater part of the tumor, other structures attracted attention. Of these, most striking were the

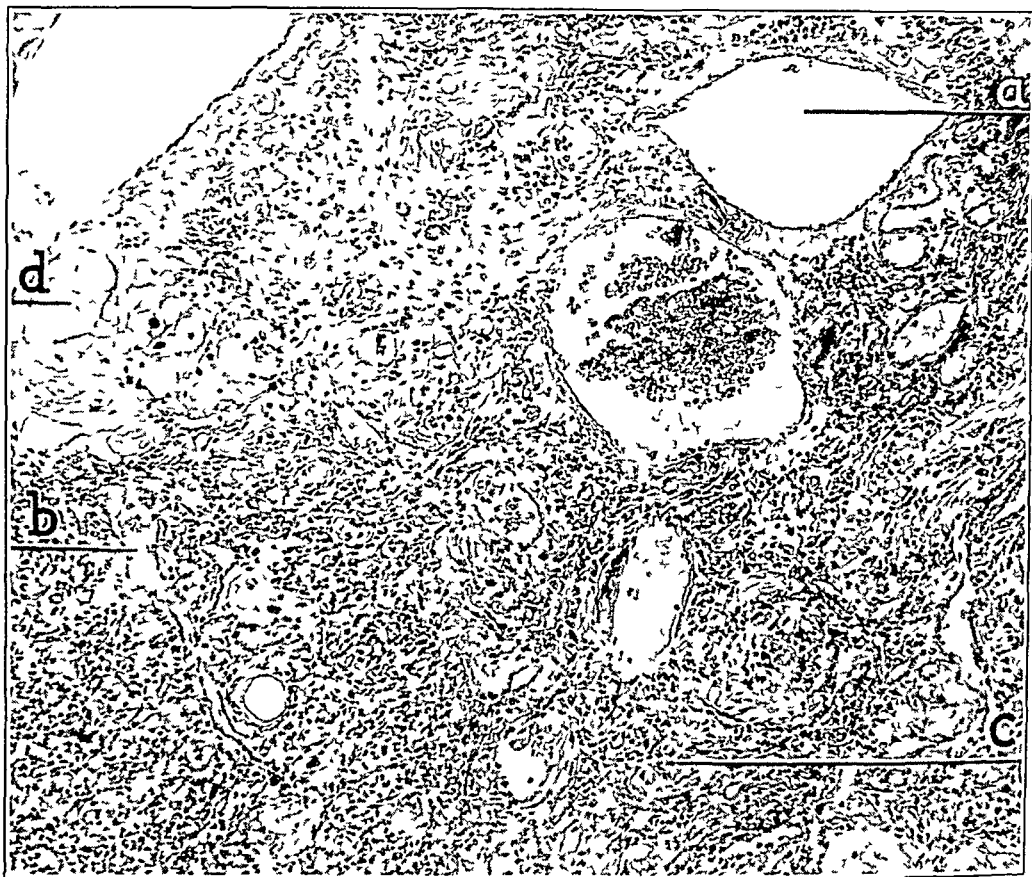


Fig 2—Low power magnification of a section of the tumor of the right eye, showing cysts (a), capillaries (b), connective tissue (c), mostly glial, and degenerative connective tissue (d). Hematoxylin and eosin, $\times 112$.

cystic dilatations, the largest of which occupied a low power field. Many were much smaller and more perfectly circular. They were completely denuded of lining cells, which might be due either to pressure over a long period or to simple degeneration. Of great importance were the capillaries that characteristically were long and narrow and frequently had red blood cells within them. They occasionally appeared like sinusoids when cut transversely. Their walls were formed by tapering slender cells. Not uncommonly the entire lumen was filled by roughly circular layers of endothelial cells,



Fig 3—Section showing the variation in size and shape of the capillaries (a) in the tumor Hematoxylin and eosin, $\times 220$

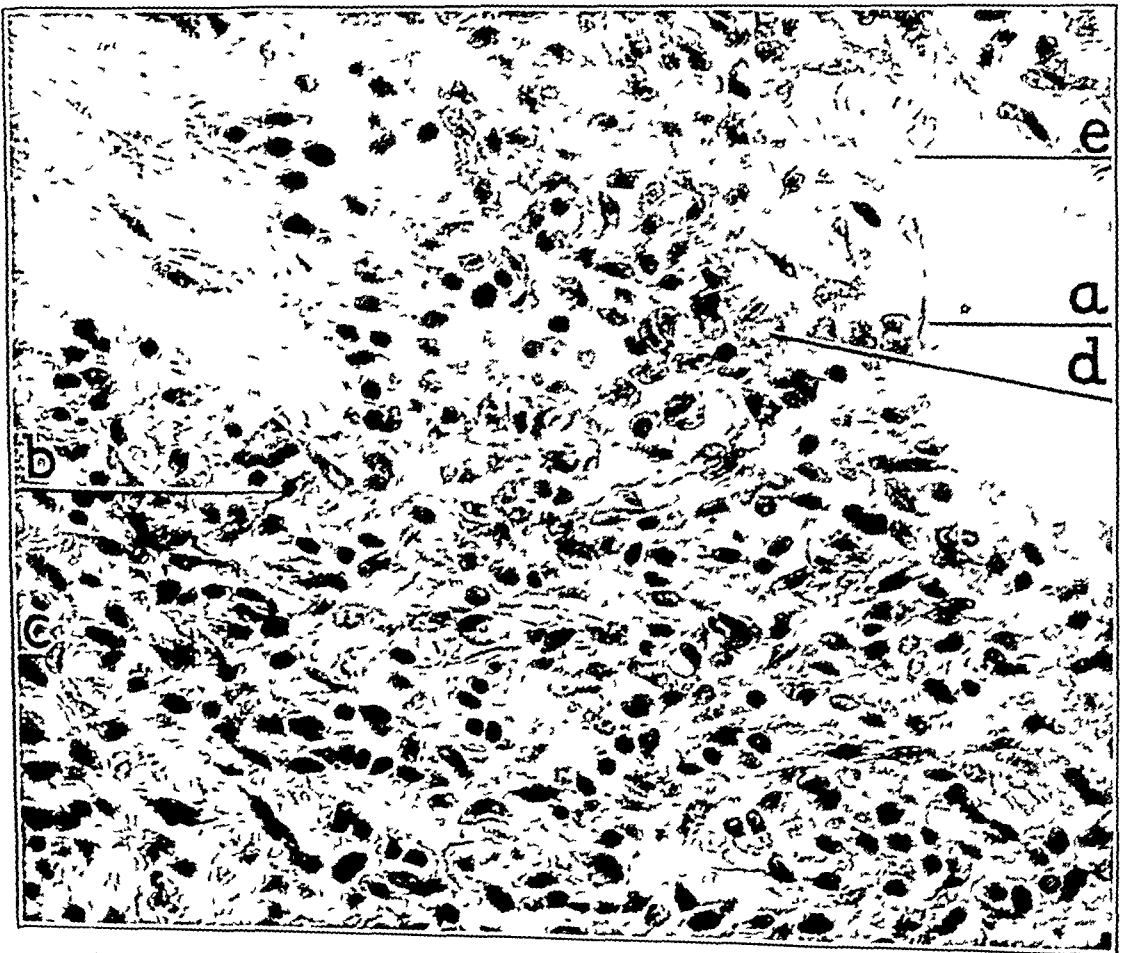


Fig 4—Section showing the cellular background in which can be distinguished endothelial lining cells of a capillary (a), glial cells with tapering fibers (b), fibroblast (c), a degenerating ganglion cell (d) and red blood cells (e) Toluidine blue, $\times 436$

which as they approached the center became more plump but did not exhibit any mitotic figures. About the cystic dilatations and larger capillaries, the Masson stain showed circular bands of green fibers. They did not penetrate into the substance of the tumor but coiled about the vessels and were composed of collagen and connective tissue. In studying the section stained by the Van Gieson method for further evidences of collagenous connective tissue fibers, the same distribution as was seen in the preparation stained by the Masson method was here recognized by the deep red fibers. Furthermore, smaller red fibers wove delicately between some of the more dense glial fibers.



Fig 5—Section showing the deeper staining collagenous fibers about the vessels and woven in the matrix. Van Gieson's stain, $\times 112$

In reviewing the structural composition of the tumor, it was evident that the background was primarily composed of glial cells and fibers with but little intertwining collagenous connective tissue. Enmeshed in this matrix were cystic dilatations and numerous capillaries. Other varied, but infrequent, cellular elements were present. It is our belief, however, that the initial event was the formation of the angioblastic derivatives, represented by the cystic dilatations and capillaries and that the gliosis was secondary and of long standing. This, then, was not a tumor of glial derivation but a tumor of vascular origin which should be considered as a congenital malformation.

COMMENT

We have presented a case of angiomatosis retinae. The diagnosis was made in the left eye on the ophthalmoscopic examination and confirmed by histologic examination of the blind right eye. The early onset of the disease is interesting, having been present in the right eye apparently since the child was 5 years of age. It is doubtful whether the injury had any bearing on the case, except to attract attention to the loss of vision in the right eye.

The clinical diagnosis in this case was either angiomatosis retinae or Coats's exudative retinitis. The clinical differentiation is not always easy. Both diseases are characterized by extensive subretinal exudate and subsequent detachment of the retina, but the singular changes in the retinal vascular system in this case favored a clinical diagnosis of angiomatosis retinae.

Characteristically in the disease, dilated vessels first appear, running to a cystlike formation or aneurysm in the periphery of the fundus. Later, secondary changes occur, namely, exudation, hemorrhages, detachment of the retina, iridocyclitis, secondary glaucoma, cataract and finally hypotension with phthisic changes.

The enucleated eye shows the secondary features. The tumor of the right eye has been shown to be of vascular origin, representing a congenital malformation, even though the gliosis is so marked that it nearly masks the true nature of the derivation of the tumor.

LESION OF THE OPTIC TRACT

PROBABLY THE RESULT OF INFECTED SPHENOID SINUSES

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The article of Onodi entitled "The Optic Nerve and the Accessory Cavities of the Nose"¹ in 1908 laid the anatomic foundation for the theory of blindness as a result of disease of the accessory sinuses. Smith, Van de Hoeve and Loeb reported the clinical application of this theory and some results obtained by surgical intervention on the ethmoid and sphenoid sinuses. White² published reports of 2 cases of retrobulbar neuritis due to disease of the accessory sinuses in 1916 and of 3 cases in 1917. These articles stimulated a wave of enthusiasm, with the result that most cases of retrobulbar neuritis not due to purely exogenous toxins, such as tobacco, alcohol, lead, thallium acetate, etc., were attributed to disease of the posterior sinuses. Since that time in this country the pendulum has swung back and forth from disease of the posterior sinuses to multiple sclerosis or foci of infection as being the predominating etiologic factor in these cases. European ophthalmologists constantly attributed most of these cases of retrobulbar neuritis to multiple sclerosis.

A case in which the lesion was similar to retrobulbar neuritis but was back of the chiasm is reported here. The findings and course of the disease suggest that sphenoid sinusitis was the cause of the condition.

REPORT OF A CASE

B B S, a married man aged 39, who had no children, first consulted Dr V H Bergmann on Dec 18, 1937, complaining of a dull pain about the waist extending to the knees. He had an enlargement of the prostate, with a slight amount of residual urine that had made it necessary for him to get up on an average of twice a night since 1932. All this time he had been under the care of a urologist. In 1935, at the age of 37, he became impotent. The past history preceding this trouble was essentially unimportant.

Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 10, 1938.

1 Onodi. The Optic Nerve and the Accessory Cavities of the Nose, *Ann Otol, Rhin & Laryng* **17** 1, 1908.

2 White, L. Loss of Sight from Retrobulbar Neuritis Due to Accessory Sinus Disease, with Report of Two Cases, *Boston M & S J* **174** 790, 1916, Loss of Sight from Posterior Sinus Disease, with Report of Three Cases, *ibid* **176** 891, 1917.

Laboratory and physical examination at that time gave entirely negative results except that transillumination of the sinuses showed a cloudy right antrum, and rectal examination revealed "enlargement of the prostate with the right lobe firmly bound down and slight fibrosis. The left lobe and central raphe were full and indurated." The diagnosis was chronic prostatic hypertrophy. Dr Bergmann³ expressed the belief that most of these cases are due to a deficiency of the anterior lobe of the pituitary gland, so the patient was given 1 cc of anterior pituitary extract every four days.

By December 31 the patient noticed definite improvement in his well-being, emotional state and sexual outlet. This improvement continued until Jan 14, 1938, the time of the onset of the present illness.

On January 14 pain developed in the right eye which was aggravated when the patient moved his eyes. Five days later a blurring of the vision developed, most marked in the right eye. The patient consulted an oculist, who concluded that the condition was due to multiple sclerosis and gave him 15 grains (0.97 Gm) of potassium iodide three times a day. At this time a careful neurologic examination gave entirely negative results. Complete laboratory examination revealed the following values: blood sugar, 89.2 mg per hundred cubic centimeters, non-protein nitrogen, 28.2 mg, creatinine, 2.1 mg, chlorides, 417 mg, cholesterol, 152.2 mg, uric acid, 3.6 mg, red blood cells, 4,590,000, white blood cells, 8,000, hemoglobin, 90 per cent, polymorphonuclear cells, 72 per cent, nonfilamented forms, 6 per cent, and large and small lymphocytes, 22 per cent.

The impairment of vision gradually became worse. The patient was referred to me on January 29. At this time vision in the right eye was limited to counting fingers at 1 foot (30 cm), vision in the left eye was 20/60 + 2. The right pupil was larger than the left and reacted sluggishly to light. The left pupil reacted sluggishly to light. The fundi were normal with the exception of a slight temporal pallor of the disk and slight blurring of the margins of the disk. Both fields showed a homonymous defect of the left inferior quadrant (fig 1) involving the maculas. Roentgenographic studies were ordered. Ephedrine sulfate was instilled into the nose, the instillation was repeated two days later. On the fourth day the sector defect (fig 2) was much smaller, and the vision improved to 6/400 in the right eye and 20/20 — 1 in the left eye.

The roentgenographic examination, made by Dr E. W. DeWeese (fig 3), revealed the following facts:

The examination included the entire head and revealed no demonstrable intrinsic disease of the bone or secondary manifestations of an intracranial lesion. The inner and outer tables of the skull were smooth and regular, and the calcium content was not locally or generally impaired.

There was no exaggeration of the vascular markings over the inner table or abnormal widening of the suture lines.

The sella turcica was unusually small, and the clinoid processes were much flattened. Another unusual finding was the degree of development of the sphenoid sinuses. There was an aerated cellular structure extending around the sellar fossa on all surfaces with the exception of the dorsal. This was obviously a developmental variation. No true erosion of the osseous structure of the sphenoid walls was found, nor were there definite pathologic changes within the cells or the clinoid processes.

3 Bergmann, V. H. Prostatic Hypertrophy as a Definite Endocrine Problem, Missouri State M. J. 34:119, 1937.

Further roentgenographic studies to include the paranasal sinuses showed large, well formed cells, symmetric in outline. No gross blocking opacities of any of the cells were found. There was present a definite mucocoele within the right antrum, and the sphenoid sinuses did not appear to be normally ventilated. There was also a rather marked general increase in the intranasal tissue, with the turbinates apparently much hypertrophied. There were some thickening and mild deviation of the nasal septum to the right. A questionable small spur formation was present on the nasal septum on the right side.

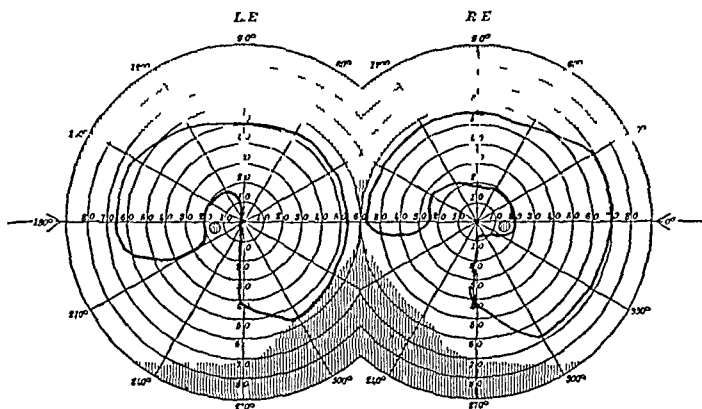


Fig 1—Peripheral fields of vision on Jan 29, 1938, the day of the first examination. Vision in the left eye was $20/60 + 2$, that in the right eye was limited to counting fingers at 1 foot (30 cm). A 1 degree target was used.

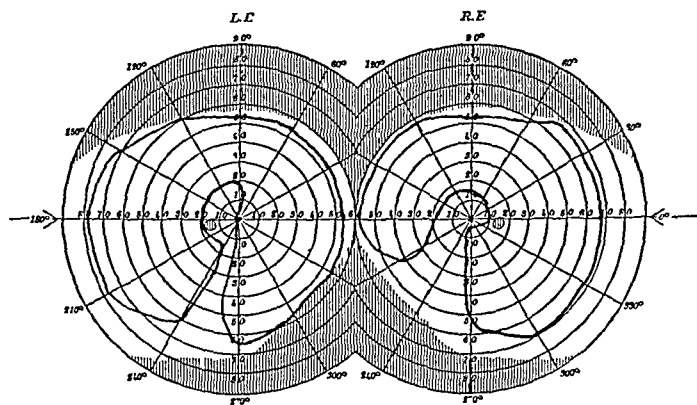


Fig 2—Peripheral fields of vision on Jan 31, 1938. There was improvement due to shrinking the mucous membrane of the nose with ephedrine sulfate. Vision in the left eye was $20/20 - 1$, that in the right eye was $6/400$. A 1 degree target was used.

The roentgenographic picture was interpreted as being indicative of a significant pathologic process within the sphenoid cell.

After February 2 the vision rapidly failed, so that on February 4 the vision of the left eye had become so poor that it was difficult for the patient to get around. He was examined by Dr. Paul Lux (rhinologist), who reported that there was no pus or crusting in the nasal passages and no change in the nasal mucosa.

The vision rapidly became more impaired, and on the evening of February 7, when the patient went to the hospital, he could not see his bed

The hematologic picture had been undergoing a gradual change in the number and proportion of the white cells from the time the patient was first examined, as indicated by the following findings

On January 19 the hemoglobin content was 90 per cent There were 4,590,000 red blood cells and 8,000 white blood cells, with 73 per cent polymorphonuclear cells and 27 per cent large and small lymphocytes

On January 31 there were 9,700 white blood cells, with 78 per cent polymorphonuclear cells and 22 per cent large and small lymphocytes

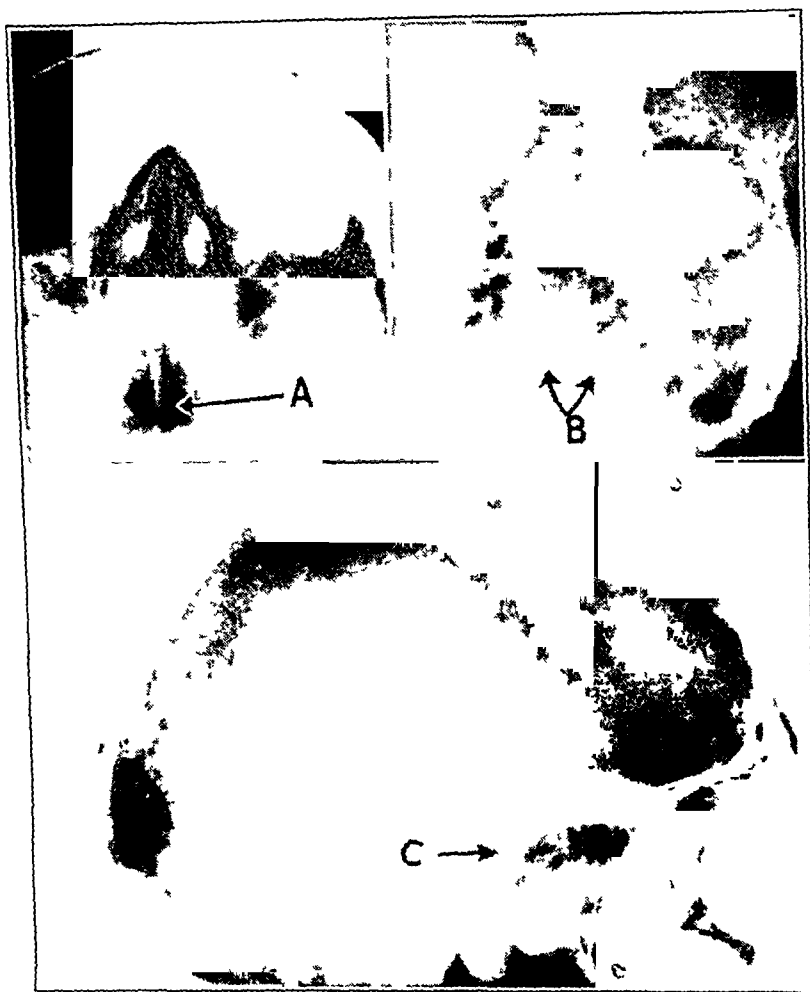


Fig 3—*A*, anterior posterior view of the sphenoid sinuses taken through the mouth Note the definite pathologic process as revealed in the veiled opacities *B* shows the marked blocking of the nasal passages caused by general hypertrophy of the turbinates *C* shows the aerated sphenoid sinus extending into the posterior clinoid process with a veiled opacity

On February 7 there were 10,600 white blood cells, with 81 per cent polymorphonuclear cells and 19 per cent large and small lymphocytes

It will be noted that the white blood cells and the polymorphonuclear cells were gradually increasing

In view of the roentgenographic picture, coupled with the changes in the fields that indicated that the lesion was in close proximity to the nasal pathologic

process, and the rapidly failing vision of twenty-one days' duration, associated with hematologic changes that pointed to increased activity of infection, I recommended draining the sphenoid sinuses

On February 8 Dr Lux performed a submucous resection and a radical operation on both sphenoid and ethmoid sinuses, a window was made in each of the antral walls under the corresponding inferior turbinate

Operation revealed a thickening of the mucoperiosteal lining of both sphenoid sinuses with a hemorrhage into the mucoperiosteal lining of the posterior superior wall of the right sphenoid sinus. There was no pus in either sinus. The bony walls

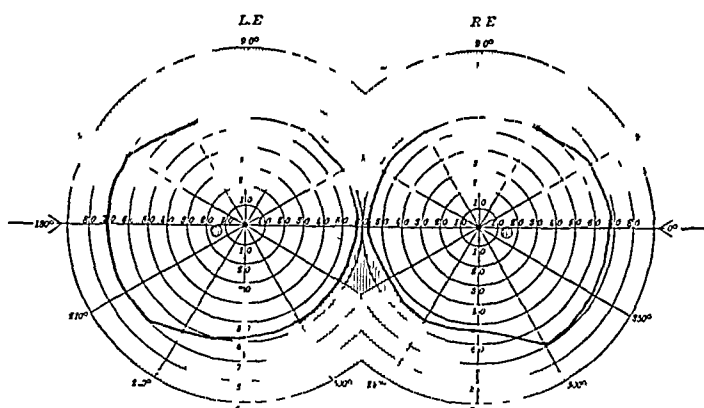


Fig 4—Peripheral fields of vision thirteen days after operation (Feb 21, 1938) on the sphenoid sinuses. Vision in the left eye was 20/20—2, that in the right eye was 20/200. A 1 degree target was used. There was concentric contraction to all colors.

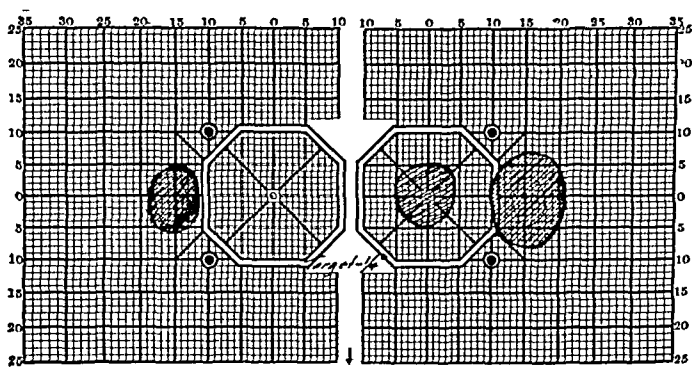


Fig 5—Peripheral fields of vision thirteen days after operation

of both anterior and posterior ethmoid cells on the right and left sides were destroyed, probably the result of a chronic catarrhal process. There was no visible change in the mucoperiosteal lining of the antrums.

The day after the operation the vision was much better, and on the second postoperative day (February 10) the patient could read large print. His vision rapidly improved.

On February 21 vision in the right eye was 20/200 and in the left eye 20/20—2. The sector defect (fig 4) in the fields had disappeared, but a large central scotoma was still present in the right eye (fig 5).

On March 12 the vision in the right eye was 20/30—2, that in the left eye was 20/20—2

The fundi were normal except a slight pallor of the temporal quadrant of the disks. The peripheral fields had returned to nearly normal with a small pericentral scotoma.

On February 12 the hemoglobin content was 96 per cent, there were 4,710,000 red blood cells and 7,900 white blood cells, with 65 per cent polymorphonuclear cells, 32 per cent lymphocytes and 3 per cent eosinophils.

The patient was given 1 cc of anterior pituitary extract every five days after the operation. On March 28 vision in the right eye was 20/30+, that in the left eye was 20/15—2. The visual fields were normal. The blindspot in the right eye enlarged 1 degree (fig 6), and it required a 1 degree target to recognize green centrally with the right eye. The patient was then sexually normal and had no nocturia. There was a marked diminution in the size of the prostate.

On May 25 vision in the right eye was 20/20, that in the left eye was 20/15—1. The peripheral fields and the blindspots were normal, and the patient could recognize colors in each eye with a $\frac{1}{2}$ degree target. He had received no treat-

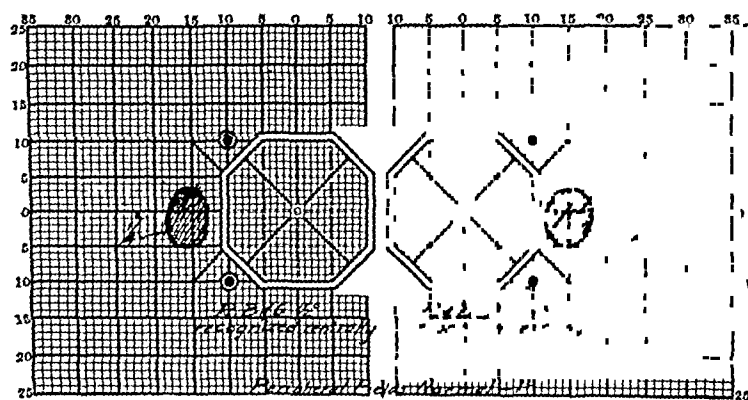


Fig 6—Peripheral fields of vision twenty days after operation on sphenoid sinuses. The only defect was 1 degree of enlargement of the right blindspot. Green was recognized only with a 1 degree target.

ment since March 28 and stated that he felt better in every respect than he has since 1932.

COMMENT

In this case hypertrophy of the prostate developed in a healthy person at the age of 33 and impotency at the age of 37. This condition was attributed by an endocrinologist to a deficiency of the anterior lobe of the pituitary gland. Improvement was taking place with the administration of anterior pituitary extract, when a lesion of the right optic tract developed, which progressed to complete loss of central vision in twenty-one days. The failing vision was temporarily improved by the use of ephedrine in the nose, as had been observed by Frost.⁴ The roentgenograms revealed poorly aerated sphenoid sinuses extend-

4 Frost, A. Papilledema, with Special References to Papilledema Associated with Sinus Disease, *Tr Am Ophth Soc* 33:480, 1935.

ing into the posterior clinoid processes. At operation the right sphenoid sinus was found to have a hemorrhagic area on the posterior wall, which was the portion extending into the posterior clinoid process and in close proximity or possibly in apposition to the superior portion of the right optic tract involving the fibers corresponding to the defect in the visual field, according to the scheme of Traquair.⁵

After the operation the vision and fields of vision returned to normal, and with six weeks of therapy with anterior pituitary extract the patient returned to sexual normality with no recurrences to date.

The logical explanation of this picture is that the patient had chronic sinusitis in an anomalous sphenoid sinus that extended into the posterior clinoid process back of the optic chiasm, causing a retrobulbar neuritis of the right optic tract. The *modus operandi* of involvement of the optic tract is problematic. It might have been the result of an actual invasion of the tract by a virus or toxins by direct continuity.

Since there was improvement temporally in the defects of the visual fields and the vision after shrinking the mucous membrane of the nose and since the disease progressed rapidly with an equally rapid improvement immediately after the opening of the sphenoid sinuses, it is not likely that the condition was due to thrombi, as was pointed out by Putnam.⁶

If the whole process had been due to invasion of the optic tract by a virus and if the theory is accepted that multiple sclerosis is due to a virus, it is possible that the infection will again become active and that multiple sclerosis will develop in the future. However, if one accepts the theory of thrombi or toxins coming from the sphenoid sinus, the patient will most likely remain permanently cured.

I feel that all patients with retrobulbar neuritis should be thoroughly studied, especially those in whom the condition seems to be secondary to disease of the posterior nasal sinuses. Such studies may lead to the solution of the perplexing problems of the underlying causes of multiple sclerosis and other demyelinating diseases.

DISCUSSION

DR A. J. BEDELL, Albany, N. Y. I wish to call attention to the observation of choked disk in 3 cases of arachnoiditis. (Slides were used to demonstrate the appearance of the fundi in the 3 cases.) Examination of a 12 year old girl revealed pale disks with indistinct margins, surrounded by retinal edema, retinal hemorrhages and exudates. Before operation the vision was reduced to 2/200. Recovery

5 Traquair, H. M. *An Introduction to Clinical Perimetry*, ed 2, London, Henry Kimpton, 1931, p. 66.

6 Putnam, T. *Etiological Factors in Multiple Sclerosis*, *Ann Int Med* 9: 854, 1936.

was complete without defects in the visual field. Ultimate vision was better than 20/15.

A man, aged 39, complained of headache. Examination showed swollen disks and retinal hemorrhages and exudates. Recovery after operation was complete, with full restoration of the visual fields and normal visual acuity.

In a 17 year old boy examination showed swollen disks and retinal exudates. After operation, which revealed arachnoiditis, vision was fully restored.

DR ALBERT D. FROST, Columbus, O. I observed 2 cases of arachnoiditis two years ago and made my studies the subject of my thesis presented to the American Ophthalmological Society. In these cases papilledema and loss of vision were accompanied by headache. The symptoms were relieved by shrinkage of the nasal mucous membrane. Full restoration of vision was accomplished by repeated shrinkage of the nasal membrane and the opening of the sphenoid and ethmoid sinuses. The first response to the nasal treatment was improvement of vision and reduction of the size of the blindspot within thirty minutes. The swelling of the disk disappeared within three days.

DR DERRICK VAIL, Cincinnati, O. I agree fully with Lemoine. The remarkable case that he reports supports with overwhelming logic and evidence the premise that the sphenoid sinuses and chiasmal cisterns have a close relationship, much closer than has been realized in the past. Whether this relationship is entirely vascular or lymphatic remains to be demonstrated. It likewise remains for the future to determine why opening and draining the sphenoid sinuses in cases of retrobulbar optic neuritis have resulted in so many instances of dramatic restoration of vision, as reported in the literature. In 1930 Cushing, discussing the chiasmal syndrome (ARCH OPHTH 3 704 [June] 1930), stated "that a mild meningeal reaction may be produced in the cerebellopontile cisternae by an inflammatory process in the middle ear with symptoms suggesting those of an acoustic tumor, is well known. There is no reason, therefore, why a similar process might not occur in the chiasmal cistern in association with inflammatory processes in the accessory paranasal sinuses." The clinical significance, therefore, of Dr. Lemoine's case assumes a great importance. Here is a patient who exhibits definite pituitary dystrophy, characteristic defects in the visual field and an extraordinary condition of the sphenoid sinuses, as shown roentgenographically. Exploration showed an absence of pathologic material in the sphenoid sinuses, except for a hemorrhagic area on the posterior wall of the right sphenoid sinus, which was the portion extending into the posterior clinoid process. The temporal pallor, the slight blurring of the margin of the disk, the characteristic sector defects and the rapid improvement of symptoms following nasal drainage are to be carefully considered as evidence pointing toward the chiasmal cistern as the seat of the disturbance. This significance will not be lost if multiple sclerosis were to develop later.

EVENTS OF VASCULARIZATION AND DEVASCULARIZATION SEEN IN CORNEAS

P F SWINDLE, PH D
MILWAUKEE

The Rocky Mountain bighorn sheep (*Ovis canadensis* Shaw) is the only animal, so far as my observations go, in which the apparently normal cornea becomes extensively invaded by blood vessels. This animal, like others, has the pericorneal network, but at or about the time of birth a number of bouquets of vascular loops (figs 14 and 15) develop between various corneal lamellae. Each of these bouquets originates from a scleral arteriovenous anastomosis which loops into the cornea (figs 8 and 9) and then metamorphoses into a principal artery, an accompanying vein, many paired arterial and venous branches and many capillaries which connect the tips of the smallest arteries with the tips of the smallest veins (figs 14 and 15). The corneal tissue remains extremely clear during the invasion of it by the scleral arteriovenous anastomoses. It is possible, therefore, to observe the metamorphosis of these anastomoses in the cornea of the lamb during early postnatal life.

The metamorphosis of any of the arteriovenous anastomoses in the cornea of the lamb does not cease until the lumen of the principal arterial trunk becomes spontaneously obliterated. This obliteration can be observed from time to time at various parts of the cornea during the very early postnatal life of the animal. The vascularization of the cornea takes place rapidly, and the bouquets of vascular loops vanish rapidly after the lumens of the principal arterial trunks become obliterated. The vascularization ceases four or five days after birth. The corneal tissue is extremely clear during the entire time of the vascularization and devascularization of it.

Other corneas may become vascularized if they become inflamed (figs 1 to 7 inclusive). In cases in which there are some persisting scleral arteriovenous anastomoses these vessels may loop into the inflamed corneal tissue, and the metamorphosis of these anastomoses may then be either identical or almost identical with the metamorphosis of the arteriovenous anastomoses in the apparently normal cornea of the lamb. For instance, such bouquets of vascular loops as those in the inflamed cornea of the black panther (fig 5) are similar to the most

extensively developed ones in the cornea of the lamb (figs 14, 15, 16 and 17) In the panther's cornea, however, the capillaries of the various bouquets of vascular loops united to form a general network of capillaries A general capillary network uniting two or more bouquets with one another was not found in the corneas of any of the lambs I shall accordingly speak of the complete metamorphosis of the scleral arteriovenous anastomoses, as in the panther's cornea, and of the incomplete metamorphosis of the scleral arteriovenous anastomoses, as in the lamb's cornea The metamorphosis of these anastomoses in a tissue is not complete until or unless capillarization progresses to such a stage that a general network of capillaries exists

In the absence of scleral arteriovenous anastomoses corneas may also become vascularized if they become inflamed (figs 1, 2, 3, 4 and 7), but the bouquets of vascular loops, like those in figures 5, 14 and 15, cannot be found in these corneas For instance, the inflamed cornea of the adult sheep may become extensively vascularized, but bouquets of vascular loops, like those in figures 5, 14 and 15, cannot be found in it The probable reason is that the supply of scleral arteriovenous anastomoses became exhausted during the original vascularization of the apparently normal cornea In other animals, such as the cat, the supply of scleral arteriovenous anastomoses may become exhausted during a first vascularization of the cornea (fig 5), so that no bouquets of vascular loops can be found in the tissue at a later vascularization of it In the absence of scleral arteriovenous anastomoses an inflamed cornea may become invaded by preexisting blood vessels (figs 1, 2, 3, 4 and 7), which are normally located at and near the limbus This is a vicarious vascularization of the cornea, and it is a poor imitation of the much more systematic vascularization by the scleral arteriovenous anastomoses Each scleral vessel, or in some instances each plexus of scleral vessels, which vicariously invades the cornea might appropriately be called a substitute for a scleral arteriovenous anastomosis, but it is a poor substitute because the cornea is often scarified and shrunk after the vessels in it vanish A cornea may be entirely normal, however, after it has been extensively vascularized by scleral arteriovenous anastomoses and the corneal vessels have vanished

In some earlier papers the metamorphosis of arteriovenous anastomoses was discussed principally in connection with the vascularization of the nasal lining and ear,¹ the nasal lining and brain² and the brain

1 Swindle, P F The Architecture of the Blood Vascular Networks in the Erectile and Secretory Lining of the Nasal Passages, *Ann Otol, Rhin & Laryng* 44 913, 1936

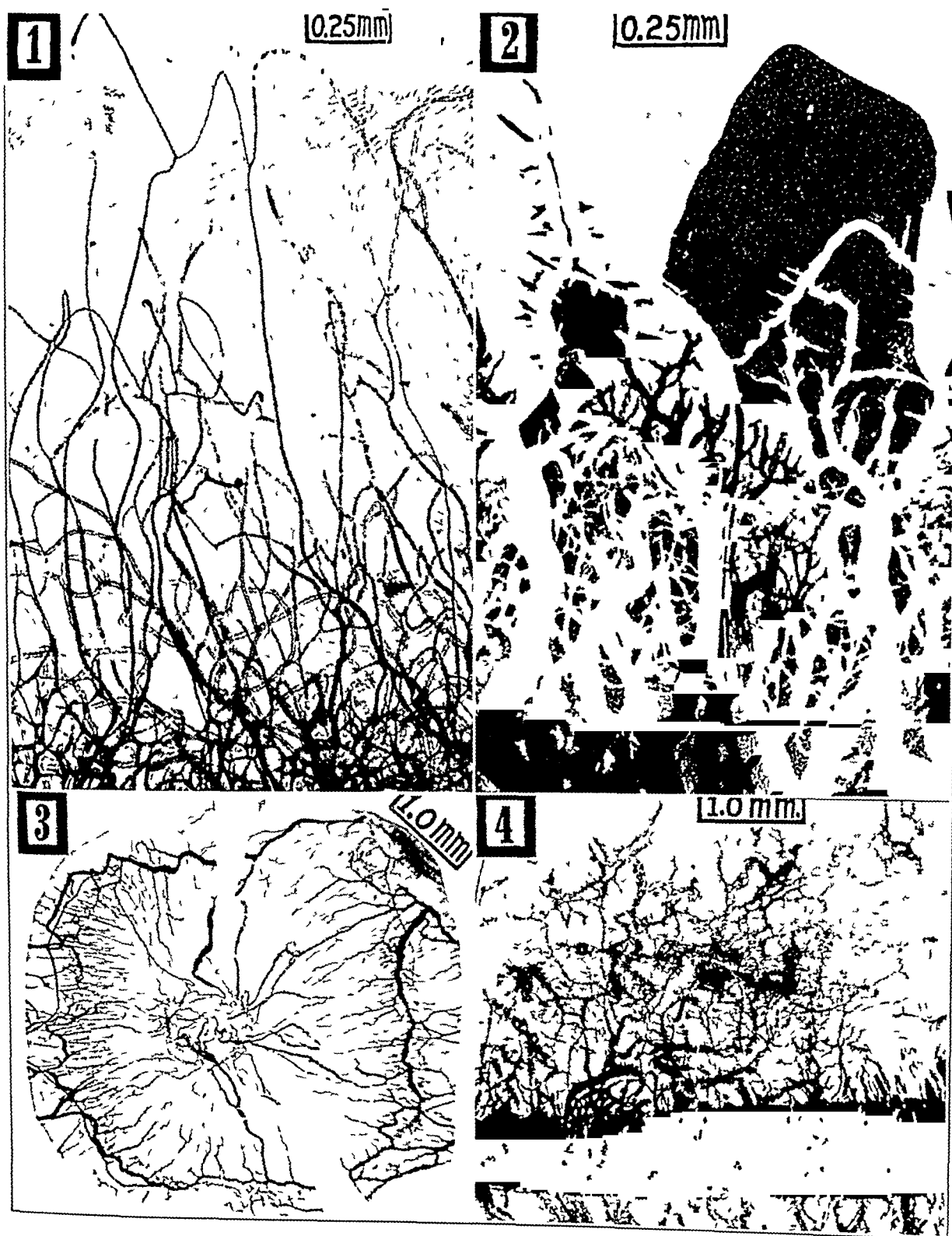
2 Swindle, P F Nasal Blood Vessels Which Serve as Arteries in Some Mammals and as Veins in Some Others, *Ann Otol, Rhin & Laryng* 46 600, 1937

EXPLANATION OF PLATE

Figs 1 to 4—Figures 1 to 3, sections from the inflamed eyes of adult albino guinea pigs. The inflammation was caused by injecting a small amount of red mercuric sulfide (in water) into the vitreous humor. Each of the eyes was observed almost constantly for several hours each day after the first signs of inflammation were detected. Each of the animals was killed by first anesthetizing it with ether and then severing the abdominal aorta. India ink was then injected rhythmically into the peripheral stump of a common carotid artery until either all or the greater number of the scleral and corneal vessels were filled with the ink. These vessels were observed with a binocular microscope during the injection. The animal was then suspended, head down, for about four hours before the eye was removed.

In figure 3 three lines of invading loops of blood vessels can be seen. The crests of the loops of the first line met at the center of the cornea. No loops of the second line extend entirely to the center, and the loops of the third line (the large vessels of the circle of Hovius) extend only short distances into the cornea at only some places. Figure 1 shows principally the vessels of the first line of invasion. Figure 2 shows loops of the first line of invasion and also many of the scleral vessels which would have looped into the cornea and been recognized as the loops of the second line of invasion if the animal had lived. Figure 2 also shows many stumps of vessels which restrained the movement of the vessels of the first line of invasion for a certain time and then broke and permitted some loops to jump farther toward the center of the cornea.

Figure 4, section from the left eye of a half-grown wallaroo kangaroo (*Macropus robustus* Gould). On gross examination the animal seemed to have actinomycosis, but the fungus could not be found. Principally the throat and the left side of the head and neck were involved. The animal was killed by quick decapitation after it lay in a profound comatose state for five days in my laboratory. The right eye appeared to be normal. The conjunctiva of the left eye was severely inflamed, and a light gray substance was deposited irregularly on the inner surface of the cornea, but the corneal tissue was clear. Blood cells could be seen circulating in some narrow and broad channels deep in the cornea at various points near the limbus. The circulation was at times erratic and at times steady. The vessels were observed for several hours each day for four days. As soon as the animal was killed, india ink was injected rhythmically into the left common carotid artery while the other severed vessels in the neck were clamped with hemostats. Before the specimen was mounted, the uvea, some of the outer lamellae of the cornea and some of the superficial tissue of the sclera were removed, so that the deep vessels at and near the limbus could be seen more clearly. The portion selected for mounting was one in which some of the blood vessels had been carefully observed while the animal was living. The dark horizontal band in the lower part of the figure is a picture of the highly developed anterior portion of the cavernous plexus of scleral blood vessels (Swindle, P. F. *The Principal Drainage Channels of the Eye*, Arch Ophth 17 420 [March] 1937). The vessels in the cornea originated from this cavernous plexus. They lie between the membrane of Descemet and the posterior lamella of the cornea. The left cerebral hemisphere was found to be almost completely devascularized.



Figures 1-4

and epiphyses³ The vascularization of the cornea by scleral arteriovenous anastomoses is similar to that of the brain and epiphyses It is also similar to the final stage of the vascularization of the nasal lining and various other parts of the body The vicarious vascularization of a cornea by substitutes for arteriovenous anastomoses is similar to the vicarious vascularization of necrotic areas at various parts of the body where these anastomoses do not exist

At the 1933 session of the American Medical Association I exhibited some corneas which had become partially vascularized by substitutes for arteriovenous anastomoses Some of the preexisting vessels, substitutes for these anastomoses, at and near the limbus were observed merely to move or loop toward the center of the cornea as they grew in length Bruckner⁴ and Kreiker⁵ had already called attention to this type of invasion of corneal tissue by blood vessels I spoke of "vascularization without neovascuogenesis" Some other corneas shown at the same meeting illustrated a combination of "vascularization with and without neovascuogenesis" At the 1934, 1936 and 1937 sessions of the Wisconsin State Medical Society and at the 1938 session of the North Central Section of the American College of Surgeons I exhibited some of the specimens and also some others In addition to the terminology used in 1933, I spoke of the "complete and incomplete metamorphosis of arteriovenous anastomoses and some of the substitutes for arteriovenous anastomoses into small arteries, small veins and capillaries"

The inflamed corneal tissue is rarely clear enough that the events of vascularization and devascularization can be clearly seen at all parts of the pannus It is tempting, therefore, to examine such pathologic corneas only or primarily after they have been injected and cleared By examining the vessels in the dead cornea only, or even by examining the living cornea for only a short period each day or each week, it is often impossible to differentiate between some of the generative and degenerative events There is a disconcerting confusion of these events in the literature In order to avoid confusing observations with assumptions, I shall describe only the events which were seen The photomicrographs presented as figures will serve to show certain features of the corneal vessels and to facilitate the descriptions of the observed events of vascularization and devascularization of normal and inflamed corneas

3 Swindle, P F Occlusion of Blood Vessels by Agglutinated Red Cells Mainly as Seen in Tadpoles and Very Young Kangaroos, *Am J Physiol* **130** 59, 1937

4 Bruckner, A Klinische Studien über Hornhautgefässe, *Arch f Augenh* **62** 17, 1909

5 Kreiker, A Die Entwicklung der Gefässbildung in der Hornhaut an der Hand von Spaltlampenbeobachtungen, *Ztschr f Augenh* **50** 115, 1923

MATERIALS AND GENERAL METHODS

The specimens herein presented as figures were selected from a large number which were collected during the past fourteen years. During this time I have performed autopsy on the animals which died in the Washington Park Zoo in Milwaukee and have had many opportunities during these years to study various normal and ailing animals under favorable conditions at the zoo. For instance, the events of both vascularization and devascularization of the cornea were observed in four healthy lambs, and the corneal vessels were studied in prepared specimens from three late fetuses and two early postnatal lambs. A number of animals from the zoo were treated and incidentally studied in my laboratory in the medical school. Some common laboratory animals were used in special experiments. The observations on the living corneas were made almost always with a binocular microscope with magnifications ranging from 10 to 216 diameters. Panophthalmitis with keratitis vasculosa was produced in some common laboratory animals by using Arnold's method⁶ of injecting red mercuric sulfide (cinnabar), in water, into the vitreous mass.

Except when it was necessary or advisable to inject some of the corneal vessels individually, all of the injections were made from a common carotid artery. India ink was injected rhythmically into this artery until the corneal arteries, capillaries and veins were conspicuously black. In some instances red mercuric sulfide (in water) was injected rhythmically into the same artery until it could be seen in many of the scleral and corneal arteries. Excessive injection of the dye was avoided so that the water would not wash the ink out of the capillaries. The vessels labeled A in figures 5, 6, 9, 13, 14, 15, 16 and 17 are amuscular arteries. Some of these arteries contain a small amount of the mercuric sulfide mixed with ink. Some of the others contain ink only, but they can be unmistakably traced back in the specimens to scleral arteries which contain some of the dye. The vessels labeled V in the same figures contain either ink only or ink and blood. These vessels are amuscular veins. The absence of muscle fibers between the tunica intima and the tunica adventitia was demonstrated by using Arnold's methods of staining and microdissection.^{6a}

All of the specimens were mounted in dammar after they were cleared, and certain parts of them, such as the uvea, and in some instances parts of the cornea, sclera and conjunctiva, were removed by dissection.

⁶ Arnold, J. Experimentelle Untersuchungen über die Entwicklung der Blutcapillaren. II Die Entwicklung der Capillaren bei der Keratitis vasculosa, Virchows Arch f path Anat **54** 1, 1872.

^{6a} Arnold, J. Beiträge zu der Entwicklung der Blutcapillaren im embryonalen Glaskörper, Virchows Arch f path Anat **54** 408, 1872.

The specimens were cleared by the Spalteholz method of dehydrating with alcohol, displacing the alcohol with either benzene or xylene and then displacing the benzene or xylene with methyl salicylate. All of the photomicrographs were taken by using transmitted light.

Some special details concerning the materials and methods are stated in the explanations of the figures and also in the body of the paper.

EVENTS OBSERVED IN CORNEAS BY DIRECT AND INDIRECT MEANS

Vascularization Unaccompanied by Neovasculogenesis—It is common for the crests of such vascular loops (substitutes for arteriovenous anastomoses) as those shown in figures 1 and 2 to move either steadily or in a jerky manner toward the center of the cornea as the legs of the loops increase in length. The steady movement of the tip of a loop could rarely be observed directly, but it was demonstrated in many corneas by indirect means. For instance, the surface of the cornea was gently pricked with a sharp needle immediately above the tip of a loop, and a small speck of either ink or red mercuric sulfide was tamped into the small wound, so that it was possible to determine whether the tip moved or remained stationary. In some instances the speck of ink or mercuric sulfide was tamped into a small wound at or near the center of the cornea, so that it was possible to measure repeatedly the distances between the speck and the crests of various loops. The distances between the speck and the points of origin of various anastomoses connecting the arterial and venous legs of some loops with one another were also measured systematically in many instances. The measurements showed not only that the crests moved toward the center of the cornea but that either parts or all of some of the cross anastomoses moved in the same direction. Some simple networks of vessels accordingly moved toward the center of the cornea.

The erratic or jerky movements of the vessels are most common during the earlier part of the invasion, as can be explained best by referring to figure 2. I was especially interested in the unusual dilatation of the limbic vessels of this eye and happened to be observing the portion of it shown in figure 2 when a vessel leaped out of the network of limbic vessels and quickly assumed the hairpin-like form, which it still has. The two conspicuous loops shown in the upper right portion of the picture also leaped farther into the cornea as well as somewhat to the left. Immediately before and also during the time that these vessels leaped away from their original positions I saw many small vessels break. The retracted stumps of the broken vessels are shown clearly in the picture. Many of them bled freely from their broken ends. These stumps are the vestiges of some capillaries and other small vessels which should be called restraining vessels, because they temporarily retarded or

restrained certain other vessels in their movements toward the center of the cornea. The cornea was clear until the restraining vessels broke and bled. I have never been able to recall the exact vascular connections of the present hairpin loop with the general network of limbic vessels before the restraining vessels broke, but I saw it assume its present form as it leaped from a relatively posterior position to its present one. The free ends of the vascular stumps attached to this loop were directed sharply toward the limbus for a few seconds after the loop developed. It is remarkable how quickly the open ends of the stumps became sealed so securely that ink did not escape from them. Ink did escape, however, from some similar stumps in certain other parts of this cornea.

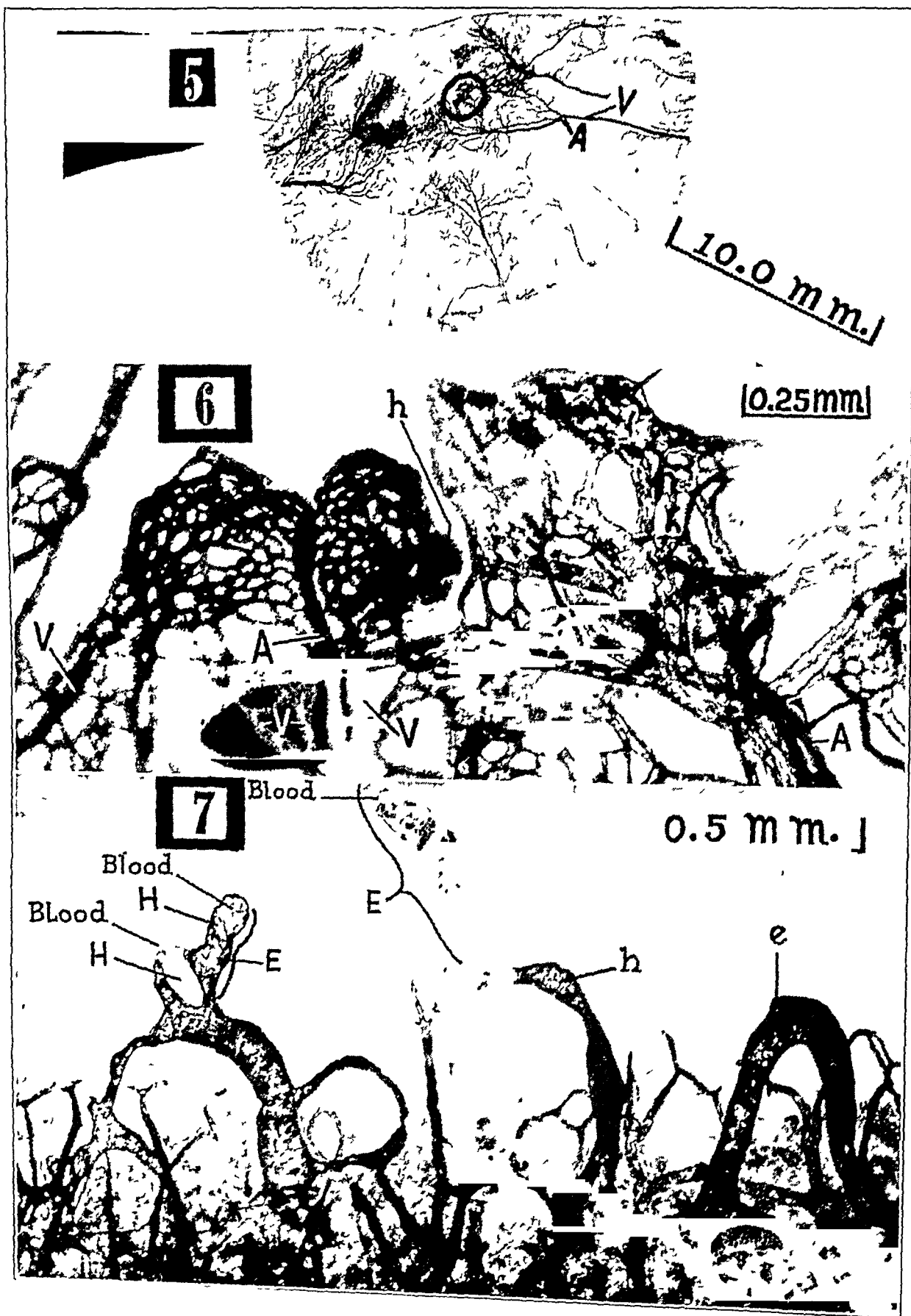
The restraining vessels (fig 2) broke so rapidly that I am unable to describe accurately the breaking in this instance. In other instances, however, I saw restraining vessels become so slender at or near the middle, as they became longer, that blood ceased to flow through their narrowed lumens shortly before the vessels broke. In some instances the lumen of a restraining vessel became obliterated near both ends, but not at the middle, so that an ampuliform pocket containing either plasma or whole blood existed at or near the middle of the stretched vessel. These pockets rarely contained blood cells. Some of the restraining vessels which I watched persisted much longer than others. In fact, some of them did not break. Their lumens became either obliterated or almost obliterated and then large enough that blood again circulated freely through them. Direct observation showed that at least in some instances the breaking of the restraining vessels was responsible for the jerky or erratic movements of vascular loops and simple networks of vessels toward the center of the cornea. The movements of the substitutes for scleral arteriovenous anastomoses (loops and simple networks) became more gradual as the cornea became depleted of the greater number of the restraining vessels. Many of the stumps of the restraining vessels became filled with static blood and then vanished.

On many occasions I saw straight and relatively straight vessels at and near the limbus become hairpin and ampuliform loops. The crests of these loops moved toward the center of the cornea as the legs of the loops increased in length. I have watched parts of the relatively large veins and arteries of the circle of Hovius as they moved farther into the cornea after they had moved (unobserved) through the limbus into the relatively clear corneal tissue (fig 3). I have also observed that the limbic region became almost completely depleted of its relatively small vessels as some of them broke and as others moved far into the cornea (fig 3). In a few instances I saw small ulcers that developed in the relatively devascularized region near the base of the cornea.

EXPLANATION OF PLATE

Figs 5 to 7—Figures 5 and 6, sections from the eye of an adult black panther which died in uremic coma. The darkest spot in the cornea (fig 5) is a small foreign body (probably a piece of corroded metal) deeply embedded in tissue. The head of the animal was removed before the blood coagulated in the vessels. The corneal vessels were injected in a rhythmic manner from a common carotid artery, while the other severed vessels in the neck were clamped with hemostats, but the greater part of the ink escaped from the corneal veins while the eye was being enucleated. These veins were then reinjected with ink individually and antidromically from points posterior to the limbus while the vessels were observed with a binocular microscope. At one region of the cornea, the upper right region (fig 5), some loose ink flowed between two corneal lamellae, but this was later removed by dissection. Parts of the trunks of some injected vessels were inadvertently destroyed while removing the ink. The upper part of the cornea was cut away, and some of the vessels in it were carefully dissected under the microscope in order to examine the tunics and especially the amorphous, jelly-like substance which separates the adventitia from the intima of many corneal vessels. The bouquets of vascular loops are situated at four different levels in the cornea. Figure 6 is a photomicrograph of some capillaries and other small vessels shown within the small elliptic area in figure 5. The ellipse was drawn on the photographic print with pen and ink. The individual vessels in the cornea were not studied while the animal was living. The vessels labeled *A* in figures 5 and 6 are amuscular arteries, those labeled *V* are amuscular veins, and the large vessel labeled *h* in figure 6 is one which assumed its present size and form during an injection with india ink. Many small vessels coalesced to form the large one labeled *h*.

Figure 7, section of an eye from a domestic cat (two-thirds grown). The cause of the keratitis could not be determined. The animal was found by the side of a road. It was so emaciated and weak that it could not stand, but it showed some improvement before it was killed. Six days after it was brought to the laboratory it was anesthetized and killed by cutting the abdominal aorta. This time was chosen to kill the animal because I was ambitious to secure a picture of the vessels in the particular stages of development shown in figure 11. I had observed these vessels carefully for many hours each day for five days. They were injected from a common carotid artery, and the specimen was otherwise prepared as described in connection with figures 1 to 3 inclusive. The structures labeled *E* are diverticula or evaginations which were invading the cornea. Some blood was driven to the ends of these evaginations by the ink. The short structure labeled *e* is a retracted evagination. The clear areas labeled *H* are tissue islands or holes which developed when opposite walls of the evagination met and fused. At the point labeled *h* a small hole or tissue island vanished while the ink was being injected. In this case two small vessels coalesced to form a larger one. The large vessels and also the small ones in figure 7 are epicorneal vessels in the sense that they are on the cornea, although they are between the bulbar conjunctiva and the anterior lamella of the cornea.



Figures 5-7

Vascularization Accompanied by Neovasculogenesis—The cornea presented as figure 7 was extremely clear. I saw the holes or tissue islands labeled *H* develop and grow larger. I saw a hole or tissue island develop at the point labeled *h*, although this hole disappeared under my eyes while the vessels were being injected with ink. One of the difficult events to observe is the development of such an island in a blood vessel. The only warning that it is about to develop is that the blood at the area is reduced to a thin film. As soon as no blood cells can be seen in the area the island has developed, as can then be demonstrated beyond reasonable doubt by killing the animal and injecting ink mildly into the vessels. It is often the case that the hole is at first very small. In some instances it remains small, but in other instances it becomes larger. I saw similar holes develop in some of the more anterior swamp-like vessels shown in figure 4, in some of the vessels which reached the center of the cornea (fig 3) and also at and near the tips of some of the loops in the corneas of the lamb (as illustrated at and near the tips of the simple and complex loops in figs 9, 11 and 18). Every time I observed the development of a tissue island in a simple vessel, in a dilated portion of a vessel or in an evagination from a vessel, I witnessed the genesis of a new blood channel or vessel. In some instances, as in the kangaroo's eye (fig 4), it seems appropriate to speak of the resulting vessels as swamp channels and of the tissue islands as swamp islands in diminutive blood swamps.

In the cornea of the lamb I watched some evaginations as they assumed different shapes. At times an evagination was long with a sharp tip, and at other times it was shorter and had a relatively blunt tip. I saw some small evaginations vanish by retracting. In some instances the original site of the evagination was marked by only a slight distortion of the wall of the mother vessel. The retraction of such a process can be illustrated by referring to figures 12 and 13. When the photomicrograph presented as figure 12 was taken, the small, sharp process which passed to the right from the lower right hand corner of the upper parallelogram (fig 12) was somewhat longer than the similar process which passed to the right from the upper right corner of the lower parallelogram, as can be seen in the picture. The photomicrograph presented as figure 13 was taken about six months later from the same mounted specimen. In figure 13 the evagination associated with the upper parallelogram is much shorter than the process associated with the lower parallelogram. The vestige of the evagination labeled *e* in figure 7 is another instance of the partial vanishing of an evagination by retraction.

Vanishing of Corneal Blood Vessels by Migration, by Transformation and by Obliteration—Blood vessels may vanish from a small or large area by moving out of it into some other part of the cornea. For

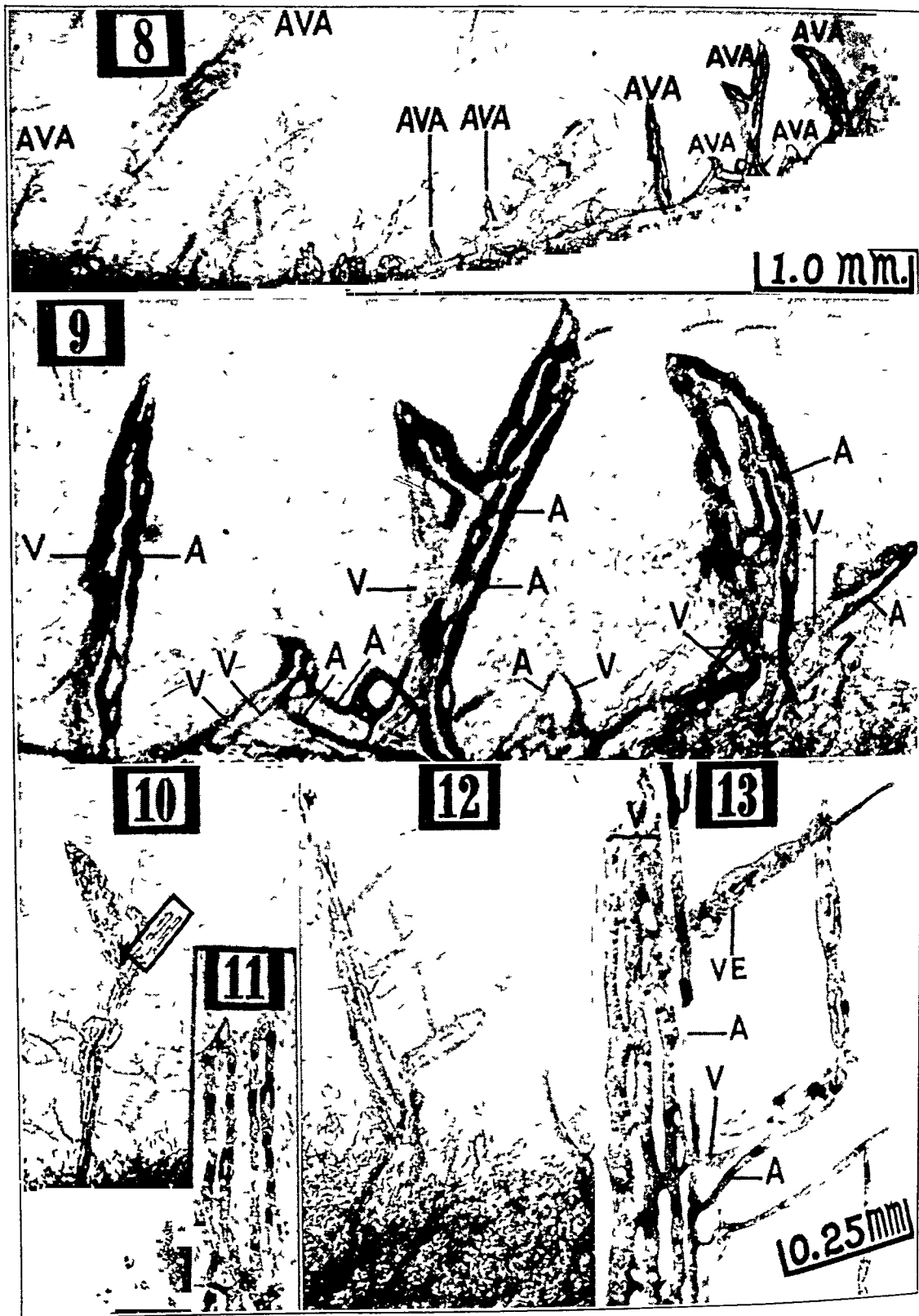
instance, as illustrated by figure 3, many vessels vanished from the limbic region by moving toward the center of the cornea. A simple rungl-like anastomosis connecting the two legs of a vascular loop with one another may be said to vanish partially in the sense that it develops into a simple or complex hairpin or arciform loop with its tip directed toward the center of the cornea. By indirect means already described I have repeatedly observed this kind of vanishing of parts of vessels, entire vessels and even simple networks of vessels from parts of living corneas. Many vessels may vanish from the sclera by moving into the cornea, and they may then vanish from the base of the cornea as they continue to move toward the center of the cornea.

Vanishing of corneal blood vessels by sudden transformation of small vessels into a larger one was observed as a spontaneous event in some inflamed corneas of rabbits. This sudden transformation means only that the holes or tissue islands which have recently developed in a vessel become suddenly eliminated as the mildly fused, opposite walls of the original vessel become separated from one another. As such islands vanish, the small blood vessels diminish in number. In a special sense the small vessels coalesce to form the original one in which the tissue islands had earlier developed. This type of coalescence was successfully brought about in a few instances by clamping the external and internal jugular veins in the neck so that the corneal vessels became so greatly distended with blood that some of the recently fused, opposite walls became passively separated from one another.

Contributory evidence of such a transformation of small vessels into a larger one can be obtained by injecting the substances into the vessels of dead corneas so strongly that some of the recently fused walls are forced apart. For instance, I saw a hole develop at the point labeled *h* in figure 7, and then I saw this hole or island vanish while the vessels were being injected with ink. In another cornea (fig. 6) I saw and studied a network of small vessels in the location of the large vessel labeled *h*. The small vessels were already injected with ink. On injecting more ink I saw many of the tissue islands among these vessels vanish, so that one relatively large vessel existed in the place of the smaller ones. This large vessel is comparable to one shown in the article published by Spicer.⁷

Vanishing of corneal blood vessels as a result of spontaneous obliteration of their lumens is an ordinary event. In the cornea of a Rocky Mountain bighorn lamb a slender stream of blood was circulating, at times rapidly and at times slowly, in the lumen of the arterial leg of a metamorphosing arteriovenous anastomosis which reached from the

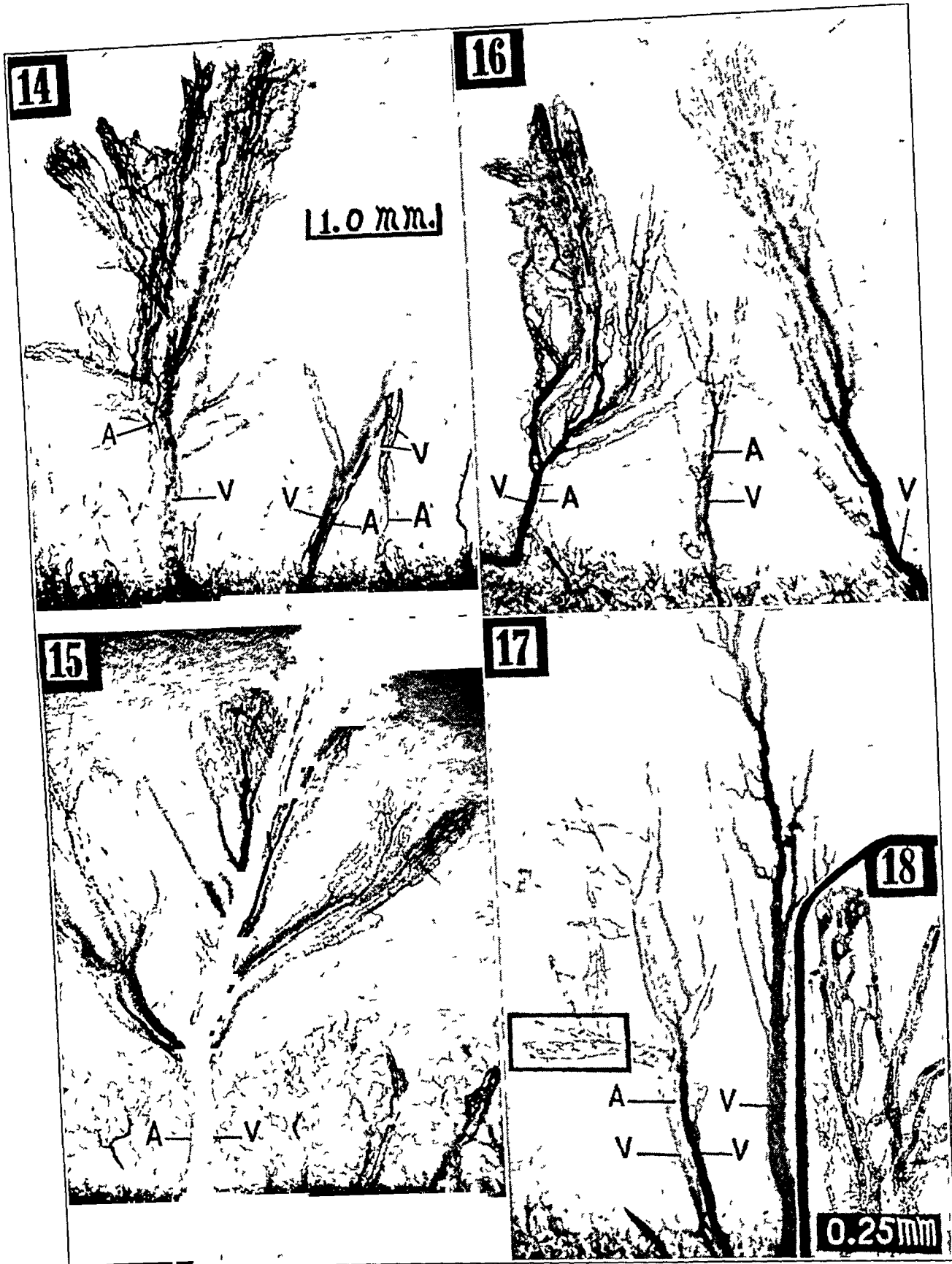
⁷ Spicer, W. T. H. Parenchymatous Keratitis, Interstitial Keratitis, Uveitis Anterior, Brit. J. Ophth., 1924, supp. 1, p. 29, fig. 30.



Figs 8 to 18 (inclusive) —Sections from the eyes of a Rocky Mountain bighorn sheep (lamb) which died (probably of starvation) about fifty hours after it was born. The blood vessels in the eyes of this animal were not studied before it died. The injections were made from a common carotid artery (first with India ink and then with a small amount of red mercuric sulfide), and the eyes were further prepared as described in connection with figures 1, 2 and 3.

Figure 9 is a higher magnification of the vessels shown to the right in figure 8. Figure 11 is a higher magnification of the two loops shown in the rectangular area in figure 10, figure 13 is a higher magnification of some vessels shown in figure 12, figure 18 is a higher magnification of the vessels shown in the

(Legend continued on next page)



rectangular area in figure 17 The magnification for figures 10 and 12 is the same as for figure 8 and that for figure 9 and 11 is the same as for figure 13 The magnification is the same for figures 15, 16 and 17 as for figure 14 The letters on the figures have the following meanings *AVA*, arteriovenous anastomosis, *A*, amuscular artery, *V*, amuscular vein, *VE*, venous evagination

The arteriovenous anastomoses in a single cornea of the lamb were found to be situated at five different levels in the tissue The epicorneal plexus of blood vessels was removed from all of the preparations except those shown as figures 10, 14 and 15 Parts of this plexus can also be seen in figure 8 The fine epicorneal meshwork of vessels is not in clear focus in any of the figures These vessels were developmentally at a standstill None of the conspicuously sturdy bouquets of vascular loops originated from the epicorneal plexus

limbus almost to the center of the cornea. Some unilateral irregularities or invaginations of the intima almost obstructed the blood flow at some points. A few minutes later the flow became completely obstructed by at least one of these intimal invaginations. The blood was then static in all parts of the arteriovenous anastomosis. I have not found it possible to reopen the lumen of such a vessel in a dead animal by injecting a liquid into a common carotid artery, although the liquid may enter the venous leg, as shown in the arteriovenous anastomosis to the right in figure 16 and also in the anastomosis to the right in figure 17. Ink was observed to pass antidromically into both of these veins. Incidentally, the venous side of the arteriovenous anastomosis to the right in figure 16 is filled principally with blood. Only the lower part of the main trunk of this venous leg is black with ink. I have failed to find any trace of these bouquets of vascular loops (as in figs. 14, 15, 16 and 17) in the corneas of adult sheep.

It was appropriate to describe the destruction of restraining vessels in connection with the erratic movements of corneal loops. In a general way that description is applicable to the destruction of some loops. This destruction of corneal loops has been described by Julianelle and Bishop.⁸ At or near the crest of a loop the vessel becomes first too slender to conduct blood, and then it breaks into two pointed stumps which retract.

According to my observations the tips of one or both of the stumps of a broken loop may be slender and slightly curved for minutes and even hours after the break occurs. The stumps often have the appearance of the "capillary sprouts" which Arnold found in his prepared specimens. Arnold assumed that these "sprouts" arch toward one another, as if by positive chemotaxis, preparatory to fusing to form an arched structure which then becomes an arched "capillary" as soon as "canalization" takes place.⁹ Immediately before the break occurs, the arched structure is often identical in appearance with the "capillary sprouts" which Arnold assumed had arched toward one another and fused but had not become completely canalized. The constriction is at times irregular, so that ampulliform blood pockets or plasma pockets exist. Some of the pockets seem to be identical in appearance with pockets which Arnold described but mistook for beginning "canalization" of the "fused" structure at or near the middle, as well as at one or both ends. I observed this destruction of loops in the inflamed corneas of some guinea pigs and rabbits, but I was unable to observe it in the cornea of the lamb. I likewise failed to observe the destruction of restraining vessels in the cornea of the lamb. I was also unable to find any red blood cells free in the cornea of the lamb.

8 Julianelle, L. A. and Bishop, G. H. The Formation and Development of Blood Vessels in the Sensitized Cornea, *Am J Anat* **58** 109, 1936.

9 Arnold, J. Experimentelle Untersuchungen über die Entwicklung der Blutcapillaren, *Virchows Arch f path Anat* **53** 70, 1871, footnotes 6 and 6a.

COMMENT

There are probably several dependent factors which contribute collectively to the looping of scleral vessels into the cornea. One is an appropriate concentration of hydrogen ions in the scleral and corneal tissue to promote rapid proliferation of the cells of the tunics¹. As this proliferation of the cells takes place, the vessels loop away from their normal positions rapidly if the surrounding tissue is so necrotic (as in certain cases of panophthalmitis) that it offers little resistance to the movement of the vessels and is rapidly eroded by the growing vessels. The partial loss of elasticity of the walls of the vessels in necrotic tissue is a factor of minor importance. Studies of the vascularization of the cornea of the lamb show that the capacity of growing arteriovenous anastomoses to erode the intralamellar scleral and corneal tissue is an important factor contributing to the invasion of the cornea by the scleral vessels. The energy imparted to the vessels by the heart is also an important factor contributing to the looping of scleral arteriovenous anastomoses into the cornea.

If the arterial blood pressure is low, the brunt of the vascularization is often borne by the veins. In this connection I wish to call attention to the inflamed corneas of the kangaroo (fig 4) and the domestic cat (fig 7). I am unable to say that an artery invaded the cornea in either of these instances. The arterial blood pressure of the cat was not determined, but it was probably low, at least some of the time, because the heart action was often feeble. The pressure in the left brachial artery of the kangaroo was determined many times with a Tycos sphygmomanometer. The systolic pressure was never found to be higher than 55 or lower than 22 mm of mercury while the animal was in the comatose state. It is conceivable that the arteries did not move and that the veins moved away from their normal positions because the arterial blood pressure was too low to urge the arteries into the cornea and that the pressure of the blood in the veins was at times great enough to urge these vessels away from their original positions in the sclera. If this reasoning is correct, the venous pressure is in exceptional cases a factor contributing to the invasion of the cornea by veins.

The vascular evagination is an incomplete or imperfect vascular loop. The development of an evagination means that only a local portion of one side of a vessel invades the tissue by moving into it, while the neighboring portions of the vessel either remain stationary or move less rapidly than the tip of the evagination. Local proliferation of cells may promote the incomplete or abortive vascular looping similarly as the proliferation of cells of the tunics of a loop may contribute to movement of the crest of this loop toward the center of the cornea. However, it is not safe to explain the development of the evaginations entirely on the

basis of cell proliferation, because some evaginations become longer and shorter and even vanish by retracting. If proliferation of cells in the walls of an evagination takes place before the retraction occurs, the wall of the mother vessel may have a local thickening due to the presence of an extra number of cells. When such a wall is examined in histologic sections a person might erroneously conclude that he is observing an incipient stage in the development of a vascular "bud" or "sprout" when he is actually looking at a vestige of an evagination.

It is only a relatively small number of the evaginations which meet and fuse with one another to form new blood vessels. Some of them vanish by retracting. Others become distended with static blood and then vanish. The greater number of them divide one or more times so that they become thereby converted into vascular loops as was suspected but not seen by Julianelle and Bishop⁸.

The development of holes or tissue islands is also a result of imperfect or incomplete vascular looping. A local portion of only one side of a vessel moves into the blood stream, while the other parts of the vessel remain either stationary or relatively stationary. The local portion that moves into the lumen of the vessel is an invagination which may touch the opposite wall and fuse with it to form a hole or tissue island, which may be easily destroyed shortly after it has formed. When the intravascular pressure is increased about the newly formed island the mildly fused walls may become separated from one another, and the same island may never develop again. The new vessels which are formed as a result of the fusion of opposite walls of a vessel may or may not form loops which grow in different directions through the tissue.

It is undoubtedly the case that in some instances during life the surrounding tissue may press against a vessel in such a manner that opposite walls of the vessel come passively in contact with one another and fuse, but I am aware of some instances (as in fetal epiphyses) in which the tissue pressure is probably not a factor in the development of the tissue islands. The greater number of the holes or islands which develop in the vessels are probably due to the invaginations of the growing walls of the vessels. Such invaginations are not more remarkable than the diverticula or evaginations which move through the tissue and form new blood vessels and concomitantly new tissue islands by meeting and fusing with one another. It is a common occurrence for at least one invagination to develop in an evagination, as is clearly illustrated in figures 7, 9, 11 and 13. An evagination may become a network of vessels before any part of it meets and fuses with any part of another evagination.

The diverticula shown in figure 7 are essentially the same as the venous diverticulum which forms the upper side of the parallelogram in figure 13. The differences in form are not important, because a single

evagination may assume various forms. The sharpness or bluntness of the tip can scarcely be looked on as being an important feature in any instance because, so far as I have been able to determine, a sharp tip, like a blunt one, has a lumen, although a part of the lumen of a sharp tip may be so small that only plasma and some blood debris can enter it. Such tips often seem to be solid structures when they are seen in prepared specimens. It would be inconsistent to speak of the evaginations as "buds" or "sprouts" without committing the obviously ridiculous act of calling the invaginations "buds" or "sprouts" also.

It is amazing how bald hairpin loops (loops which do not possess evaginations) avoid touching one another in growing through normal tissue, as at and near the center of a highly vascularized cornea of the guinea pig or that of the lamb. A bald hairpin loop may be growing in the direction of another one but either stop growing or merely swerve to one side of the second one in time to avoid touching it. Loops which are provided with or crowned with evaginations behave in exactly the same manner as the bald ones, except that the evaginations of approaching loops may not succeed in avoiding one another and may accordingly fuse to form anastomotic connections between the loops. If the loops consist of capillaries, the haphazard fusion of the evaginations may eventually be responsible for the existence of a complex network of capillaries which connects all of the smallest tips of the arteries and veins of many bouquets of vascular loops with one another. Each of these bouquets should then be spoken of as a completely metamorphosed arteriovenous anastomosis.

The bouquets of vascular loops shown in figures 14, 15, 16 and 17 are incompletely metamorphosed arteriovenous anastomoses. It is true that evaginations are present in these bouquets, but in the living cornea the arteriovenous anastomoses were at so many different levels in the lamellated or stratified corneal tissue, the number of metamorphosing arteriovenous anastomoses at any level was so small and the distribution of these anastomoses at any level was such that the evaginations of any one anastomosis did not have an excellent opportunity to come in contact with and fuse with evaginations of any other anastomosis. It is probable, however, that if some of the metamorphosing arteriovenous anastomoses at a certain level had reached the center of the cornea the metamorphosis would have become complete, as it did in the cornea of the panther (figs 5 and 6) or as in the eye of the guinea pig presented as figure 3. After the crests of several loops reached the approximate center of the cornea (fig 3) they avoided one another by twisting and turning in various directions. I then saw (for the first time in this eye) diverticula develop and fuse with one another, and I also saw holes or tissue islands develop in some of the vessels which became distorted

after they reached the center of the cornea. A result of the distortion of the crests of the corneal loops, the development and fusion of evaginations and the development of new blood vessels due to the invaginations is the hemangioma-like tangle of vessels seen at the center of the cornea (fig 3). Many of the arteries vanished, so that the tangle eventually consisted principally of veins and capillaries. It seems correct to speak of the diverticula or evaginations as fusion evaginations, because, so far as my observations go, vascular loops do not become united with one another if evaginations are not present.

My observations that evaginations rarely fuse with one another in the cornea and that the fusion is haphazard when it occurs (as illustrated in fig 13) are opposed to the notion of Arnold,⁹ Augstein¹⁰ and many others that the tips of "capillary sprouts" regularly approach one another, as if by positive chemotaxis, and then fuse to form straight and arched structures, which soon become "canalized." I am convinced that Arnold's alleged instances of systematic meeting of the tips of his "capillary sprouts" was greatly overestimated owing to his frequently mistaking degenerative events for generative ones while observing his prepared specimens. He undoubtedly mistook stretched portions of restraining vessels and stretched crests of some vanishing loops for fused tips of "capillary sprouts" which had not become completely "canalized," and he undoubtedly mistook the greater number of slightly retracted stumps of vanishing vessels for "capillary sprouts" which were in the act of approaching one another, as if by positive chemotaxis preparatory to the meeting and fusing of their tips. Arnold did recognize a small number of the stumps as being probably the retracted stumps of broken "capillaries." This last interpretation is remarkable in view of the fact that Arnold did not see a "capillary" break.

One of Arnold's findings is important in connection with the phenomenon of the final devascularization of the cornea. This is his observation that each of the greater number of the "capillaries" and even many of the "capillary sprouts" is enclosed in a cylinder of adventitial cells and fibers. He demonstrated this fact by reflecting a part of the adventitial wall to expose the intima (which he considered to be the capillary). In fresh specimens there is a relatively clear space (intramural space) between the walls of the two cylinders of many of the corneal vessels. Some of my microdissections revealed that this space is filled with a jelly-like mass. This is especially evident in the largest arterial trunks. After one of these vessels has existed for a considerable time, the lumen becomes progressively smaller as the intramural space becomes progressively larger. The lumen continues to decrease in

10 Augstein. Gefasstudien an der Hornhaut und Iris, Ztschr f Augenheilkd 8 317, 1902

size until it becomes completely obliterated at one or more points along its course. This obliteration can undoubtedly be attributed to such an increase in the amount of the jelly-like mass that the walls of the intima are forced together at one or more points. In other parts of the body the intramural space can be found, but it becomes invaded by muscle and connective tissue and in some instances also by small blood vessels (*vasa vasorum*).

Some simple loops in the cornea become closed off at or near their crests. This is probably due to a postinflammatory shortening of the legs of the loops, because as soon as a loop breaks at or near its crest the legs sometimes retract to such an extent that the ends of their pointed tips are separated from one another by a considerable distance. In a healing cornea the legs of a loop may be in a relatively healed area near the limbus, while the crest is in a relatively unhealed area and may accordingly be the weakest and most inelastic portion of the loop. If the legs retract, as the surrounding tissue contracts the lumen becomes obliterated at or near the crest before the vessel breaks.

It is certainly incorrect to explain the movements of vascular loops in terms of positive chemotaxis, because the crests of the loops tend to avoid touching one another. I believe that it is equally incorrect to speak of negative chemotaxis as a factor contributing to the tendency of the crests to avoid one another. These crests merely grow best in the avascular parts of the tissue, where the concentration of hydrogen ions is greater than it is in the immediate vicinity of another vessel, where the exchange of respiratory gases and nutrient and waste substances in general is relatively perfect. Loops may accordingly become interdigitated without touching one another. The evaginations are not so successful in avoiding one another, because these blind structures contain static or relatively static blood, so that the exchange of respiratory gases and other substances is not good in the immediate vicinity of these structures. The hydrogen ion concentration is accordingly somewhat high about them, but it is low enough that they show an unmistakable tendency to retract, to stop growing or to swerve gently away from one another when their paths of growth cross or meet. However, some of the evaginations do meet and fuse in some corneas.

SUMMARY AND CONCLUSIONS

An inflamed cornea may become almost completely vascularized by preexisting scleral and conjunctival vessels before a new blood vessel develops in it. In fact, many vessels may become destroyed in the process. This is vascularization unaccompanied by neovasculogenesis. Single and interconnected vascular loops move first erratically and later steadily toward the center of the cornea. The irregular or erratic movement is due to the breaking of some stretched vessels which were called

restraining vessels because they temporarily retard the motion of other vessels in relatively more advanced positions in the cornea. The restrained vessels may leap or rush rapidly toward the center of the cornea every time some of the restraining vessels break. After the cornea becomes depleted of the greater number of the restraining vessels, many parts of the remaining vessels move steadily through the tissue. As the legs of a loop become longer, owing to loss of elasticity of the vessel but principally owing to the proliferation of the cells of the tunics, the crest of the loop slowly erodes the restraining tissue ahead of it and moves farther into the cornea. In some instances the scanty interlamellar tissue is so necrotic that the erosion by the crest of a loop is a negligible factor.

In the apparently normal cornea of the Rocky Mountain bighorn lamb and in some traumatized corneas the crest of a loop may erode the tissue so slowly that at least one incomplete loop may develop and advance beyond the major portion of the crest. This incomplete loop is a diverticulum or evagination with a lumen. It has been erroneously spoken of as an "endothelial bud" or a "capillary sprout." It may have at one time a pointed tip and at another time a blunt one. It may even vanish by retracting, or it may grow longer and meet and fuse with a similar evagination either from the same vessel or from a different one. These incomplete loops should be called fusion diverticula or fusion evaginations, because they are the only vascular structures which were observed to meet in the tissue and fuse with one another. They are excellent tissue invaders, because they are so small and pliable that they can enter and move through extremely small interlamellar and even intralamellar spaces. These diverticula are not excellent fusion structures, because they tend to avoid one another by retracting, by merely ceasing to become longer or by gently swerving away from one another. However, when these fusion evaginations are plentiful some of them may fail to avoid one another and accordingly fuse to form new blood vessels.

Another incomplete vascular loop is the invagination which also originates from only one side of a vessel and increases in length. It does not invade the tissue. It invades the blood stream instead. Its tip touches and fuses with the opposite wall of the same vessel, so that a bloodless area, a hole or a tissue island develops in the vessel and two blood channels or vessels concomitantly develop out of the original vessel. One or more of the tissue islands may develop in the crest of a loop, in a leg of a loop and even in an evagination or diverticulum. Some of the new vessels thereby developed form hairpin or arciform loops. These move into or invade nonvascularized portions of the corneal tissue where they become new sources of development of more evaginations and more invaginations.

Spontaneous destruction of corneal vessels is a common event. For instance, certain vessels (restraining vessels) at and near the limbus become stretched and then broken by some others which are moving relatively rapidly in relatively advanced positions in the cornea. It is also the case that the legs of certain loops retract, so that the crests of these loops become stretched and even broken. This destruction of loops takes place when the crests are in the relatively unhealed portion of the cornea near its center and the legs are in the relatively healed portion near the limbus. As the relatively healed corneal tissue contracts, the legs of some of the loops in this tissue become so short that their crests break. It is also true that the lumens of corneal arteries eventually become small and that such intimal invaginations develop in these arteries that their already narrowed lumens become locally obliterated. If the obliteration takes place in the principal arterial trunk of a bouquet of vascular loops which is not connected by anastomoses with vessels of neighboring bouquets, the entire bouquet (including the principal venous trunk) vanishes. However, if the tips of several bouquets are joined with one another, the greater number of the principal arterial trunks may vanish without bringing immediate destruction to the greater number of the capillaries and veins of the several associated bouquets. The narrowing of the lumens and the development of the intimal invaginations are due to the increase in volume of a jelly-like substance between the intimal and the adventitial cylinders of the amuscular arteries.

ORBITAL HYPEROSTOSIS

ITS OCCURRENCE IN TWO CASES OF MENINGIOMA OF THE SKULL

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Thickening of the bony walls of the orbit was formerly classified as hyperostosis circumscripta, as described in Naito's atlas ¹ On the basis of the knowledge of meningiomas following Cushing's ² investigations, a new interpretation of these bony changes arose and a new classification became necessary In a recent personal communication Schuller ³ stated

In the past ten years, since Naito's book appeared, we have observed some cases where the x-ray diagnosis of osteoma or hyperostosis circumscripta had to be corrected for hyperostosis in meningioma But there are certain reasons that we cannot abandon the group of circumscribed hyperostosis of the skull, because on one side there are cases without progression which are clinically harmless, and on the other side the circumscribed hyperostosis is the beginning of leontiasis ossea, which gives a bad prognosis and is not operable

Osteoma of the orbit is quite a different process, as these bony tumors (generally called ivory exostoses) arise on the inner wall of the orbit, usually from the frontal or the ethmoid sinus

Meningiomas occur between the third and the fifth decade of life, at least a decade later than the pituitary adenomas They are frequent and occurred in Cushing's series in a ratio of 1 to 4 gliomas

Cushing has divided meningiomas into two groups, in one the growth is large and more or less irregularly lobulated, with a small stalk of attachment to the dura, and in the other the tumor is termed "en plaque" and is round, flat and slightly elevated and tends to spread over the inner dural surface The latter type of growth is less frequent and arises usually from the basal meninges Meningioma is found in the places where the arachnoid villi and their associated cell clusters are situated The arachnoid villi and the cell clusters act as filters for the cerebrospinal fluid between the subarachnoid spaces and the dural sinuses They become hypertrophied and are the seats of predilection for the development of meningioma The capping clusters of endothelial cells

Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 9, 1938

1 Naito, I Die Hyperostosen des Schädels, Vienna, Julius Springer, 1924

2 Cushing, H The Meningiomas (Dural Endotheliomas) Their Source and Favoured Seats of Origin, Brain **45** 282 (Oct) 1922

3 Schuller Personal communication to the author

frequently show a tendency to the familiar whorl formation, and psammoma bodies are present

Aoyagi and Kyuno,⁴ two Japanese investigators, have studied the usual situation of these cell clusters

Meningiomas, therefore, have certain definite seats of predilection, and each group, on account of its anatomic seat of origin, possesses a definite characteristic symptomatology. The growths abound along the major sinuses and also in the basal meninges, especially over the tip of the temporal lobe and in the region of the gasserian ganglion. In the latter region the meningioma is of the flat (*en plaque*) variety and appears to rise from near the pterion, where the meningeal vessels and sinuses so commonly channel the bone.

The overlying bone is involved in about 25 per cent of the cases of meningioma. While this hyperostosis occurs in both varieties, it is particularly prevalent in the flat variety.

It is the following groups of meningiomas with which this paper is concerned: the tumors of the sphenoid ridge and tumors of the sylvian cleft (temporofrontal). In these locations a meningioma is particularly prone to push through the dura and later to invade the overlying bone, causing striking hyperostosis. The bone may be involved in a number of ways. Frequently the bone is enormously thickened and the canaliculi are filled by tumor cells.

Concerning tumors of the sphenoid ridge, Cushing⁵ stated

The growths which lie astride the sphenoidal ridge, with a portion of the tumor resting on the orbital plate under the frontal lobe and a portion in the middle fossa indenting the temporal lobe, are fairly common and characteristic. Most of them have been unexpected findings, though one or two have been recognized because of an absorption of the sphenoidal ridge shown by an antero-posterior roentgenogram. They may cause uncinate seizures and not infrequently encroach on the side of the chiasma and produce an homonymous hemianopsia.

Cushing's description of tumors of the sylvian cleft (temporofrontal) follows:

These are, on the whole, similar to the above and doubtless arise from the same sinus, but they lie sufficiently far lateralwards to be brought into view by a subtemporal decompression. Two of them which gave no localizing symptoms were unexpectedly disclosed in this way and were subsequently removed by an osteoplastic exposure. In this region, too, for some unexplained reason, the tumors *en plaque* are common, and as these meningiomas are apt to provoke hyperostosis and cause a palpable thickening of the bone at the temporo-sphenoidal junction, they are easily recognized through this indication of their presence, as well as by the marked exophthalmos which occurs if the hyperostosis involves the outer orbital wall.

4 Aoyagi and Kyuno. *Neuroglia* 11 1, 1912

5 Cushing, H. Cranial Hyperostoses Produced by Meningeal Endotheliomas, *Arch Neurol & Psychiat* 8 139 (Aug) 1922

The microscopic examination of the tumor tissue shows typical clusters of arachnoid cells arranged in whorls and lying within the dural layers. The bony tumors show bony channels filled with connective tissue and tumor masses.

Meningioma in this location runs a characteristic slow course, with no neurologic symptoms and no increased brain pressure. The principal ocular symptom is exophthalmos, and the eyeground is frequently not changed until late in the disease.

Cushing⁵ in 1922 wrote an article on cranial hyperostosis produced by meningeal endothelioma, in which the hyperplasia of the adjacent bone and the possibility that it may overlie a meningeal endothelioma were emphasized. The report showed that this type of tumor may be circumscribed and massive or flat, and if it were not for the associated hyperostosis and its consequences the tumor would not be recognized. In the case reported a smooth hard tumor could be palpated in the left temporal fossa, and the only ocular symptoms were exophthalmos and paresis of the abducens nerve. The roentgenogram revealed increased density in the region of the squamous wing of the temporal bone and of the adjoining part of the sphenoid bone. At operation the temporal muscle was found invaded, and the underlying bone was thickened. This was removed as well as the outer side of the orbit and of the sphenoid wing down to the region of the anterior clinoid processes in the depth of the sylvian groove. At the depth of the operative field the dura was toughened, and when it was opened a flat endothelioma was exposed.

In an excellent article with a good bibliography, Alist Stender⁶ described 3 cases in which the meningioma arose from the posterior surface of the greater wing of the sphenoid bone. He also found that meningioma with its origin on the posterior surface of the sphenoid bone frequently presents a characteristic clinical syndrome, with swelling of the temporal and sphenoid bones and extension to the bony orbit, with exophthalmos. The process progresses slowly and gives rise to few symptoms, and there is no increase in the brain pressure. The tumors are usually found in the form of plaques and infiltrate the sphenoid bone and the adjoining bones and occasionally the temporal muscle. Associated with the tumor there is a new formation of bone.

The report of the cases on which this paper is based follows.

B. L., a 44 year old woman, was first seen on Jan. 23, 1922. She stated that the left eye had bulged for one year. There was some headache, vision in the left eye was 20/20, and an exophthalmos of 5 mm was present. The visual field and interior of the eye were normal, there was weakness of the external rectus muscle. The bone of the outer wall of the orbit was thickened, within the outer

6 Stender, A. Ueber das Meningiom des Keilbeinrucksens, *Ztschr. f. d. ges. Neurol. u. Psychiat.* **147** 244, 1933.

orbital margin a firm resistance was encountered on palpation, and the whole temporal region was swollen and tender

The condition was believed to be a circumscribed hyperostosis and an operation was undertaken to remove the excess bone in the orbit. A Kronlein operation was attempted, but the bone was found so hard that an osteoplastic flap could not be prepared, and the bone was removed piecemeal by a chisel and hammer. The postoperative recovery was slow but uneventful. Moderate exophthalmos remained.

On September 15, two years later, the exophthalmos of the left eye was unchanged, but the optic nerve showed neuritic atrophy. Vision was 20/30.

The patient was not seen again until 1924, when the vision in the left eye was found to be lost and the optic nerve was atrophic. Pain about the left orbit had continued.

In 1929 the left eye became more prominent. The bony swelling in the temporal region was greater. The pain about the orbit was still present. The vision in the right eye was normal.

The patient was then lost from observation until 1935, six years later, when the left eyeball was more prominent and the cornea was inflamed from exposure. The eye was removed, and the orbit was found filled with bony masses. After operation there was persistent edema of the lids, and there seemed to be a soft mass in the depth of the orbit. The patient was referred to the Memorial Hospital, and Dr. Hayes E. Martin reported that though a number of roentgenograms were studied no diagnosis was made. On account of the swelling of the lids and edema of the conjunctiva and the patient's pain, an operation was done on December 20, the skin was peeled back from the upper eyelid, and the edematous mass was excised. When the orbital cavity was entered it was found to be filled with a corded, nodular fibrous mass which had the gross appearance and consistence of a hemangioma hypertrophicum. Along the outer wall of the orbital cavity this process seemed to have invaded and destroyed the cortex of the bone or to spring from it. As much as possible of the mass in the orbital cavity was removed. The incision was then extended laterally, and a specimen of bone was removed from the main tumor mass for histologic examination. The incision was closed, and the excess skin of the upper eyelid was pushed back into the orbital cavity and a gauze pack applied. Healing was without incident and the orbital cavity is now entirely healed and covered with skin.

According to Dr. Martin, it was surprising to find that the histologic diagnosis of the material removed from the orbital cavity and also from the bone specimen was meningioma.

The photomicrographs showed meningioma typical in form and structure with new bone formation (figs 1 to 4).

The roentgen findings in 1935 showed the marked changes in the bones of the skull (fig 5).

The patient was given roentgen treatments three times a week for a total of fifty treatments. The vision in the right eye remained normal.

The patient was seen on Sept. 8, 1937, when the bony swelling (fig 6) measured roughly 8 by 6 by 4 cm. The shooting pains were entirely relieved by roentgen treatment.

In this connection it is interesting to note that Velter⁷ stated that roentgen treatment of meningioma may have a harmful effect by produc-

⁷ Velter, E. Le diagnostic des affections de la région chiasmatisque et sellaire, *Arch. d'opht.* 53: 593 (Aug.) 1936.

ing local anatomic changes (adhesions and vascular changes) which interfere with operation

The patient in the case reported here presented the characteristic symptoms of unilateral bony deformity of the skull and exophthalmos, with no changes in the optic nerve in the early stage and slight paresis of the external rectus muscle. Roentgen examination showed increased

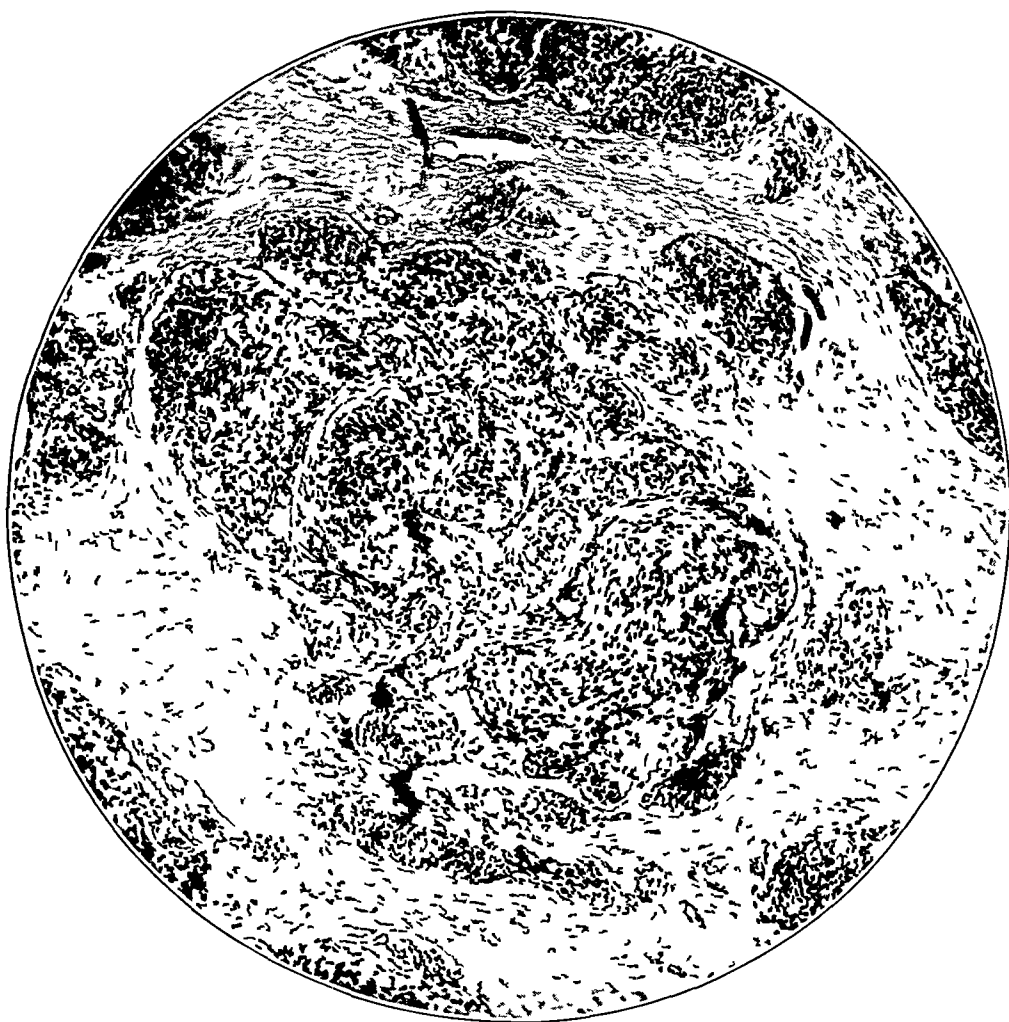


Fig. 1—Photomicrograph showing the appearance of the tumor in the dura, low power magnification

density and enlargement of the squamous portion of the temporal bone and of the large wing of the sphenoid bone, with involvement of the outer and upper walls of the orbit. There is no reason that with the present knowledge the correct diagnosis should not be made at an early stage and the condition relieved by operation when the operative procedure is not formidable.

Elsberg, Hare and Dyke⁸ reported a number of cases similar to the one described here and observed 10 meningiomas in 15 tumors associated with unilateral exophthalmos. They found that the protrusion of the eye was due either to perforation of the tumor into the orbit or to thickening of the bony orbital walls or extension of the tumor through the superior orbital fissure and not to pressure on the cavernous sinus or the ophthalmic veins.



Fig 2—Photomicrograph showing the appearance of the tumor in the bone, low power magnification

Cases of this type have been but rarely reported in the ophthalmic literature, though this condition is not unusual and should be recognized by the ophthalmologist to whom the patient is likely to apply because of the striking exophthalmos.

⁸ Elsberg, C. A., Hare, C. C. and Dyke, C. G. Unilateral Exophthalmos in Intracranial Tumors with Special Reference to Its Occurrence in the Meningioma, *Surg., Gynec. & Obst.* **55** 681 (Dec.) 1932

Cohen and Scarff⁹ recently reported a case of meningioma of the middle fossa

The second case was somewhat similar though the condition has shown no progression

L. E. B., a woman aged 38, was seen on Dec 13, 1924 Six years before she met with an accident and was thrown against the left side of the head A roentgenogram showed a bony tumor of the outer wall of the left orbit to be

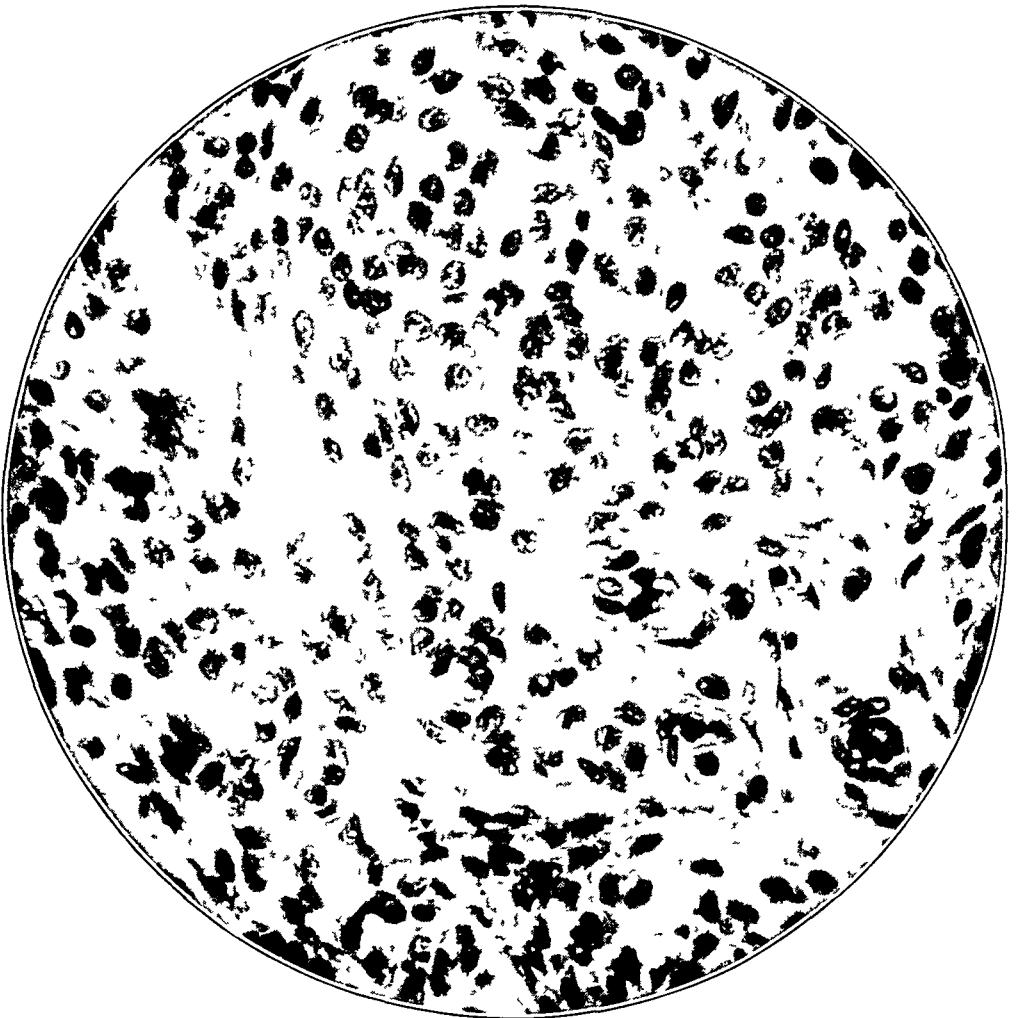


Fig 3—Photomicrograph showing the appearance of the tumor in the dura, high power magnification

present There has been no change in the past six years The left eye showed an exophthalmos of 8 mm, and the eye was pushed straight forward The vision was 20/20— Motility of the left eye was normal except that the external rectus muscle was weak There was thickening of the upper outer orbital margin, and a definite tumor could be palpated within the orbit posterior to the external canthus The optic nerve showed some swelling and neuritic atrophy

⁹ Cohen, M, and Scarff, J E Unilateral Exophthalmos Produced by a Meningioma of the Middle Cranial Fossa Report of a Case, Arch Ophth 13 771 (May) 1935

The roentgenogram revealed increased density of the bone of part of the left orbit, especially of the greater wing of the sphenoid bone and the outer surface of the frontal bone. The malar bone was slightly involved, the sella turcica was normal. The patient was given roentgen treatment.

In 1936 the condition had not changed. The vision in the left eye was 20/30. The exophthalmos measured 8 mm. The visual field was normal. The optic nerve was pale, and the external rectus muscle remained weak. In reply to a recent inquiry, the patient stated that the condition is unchanged.

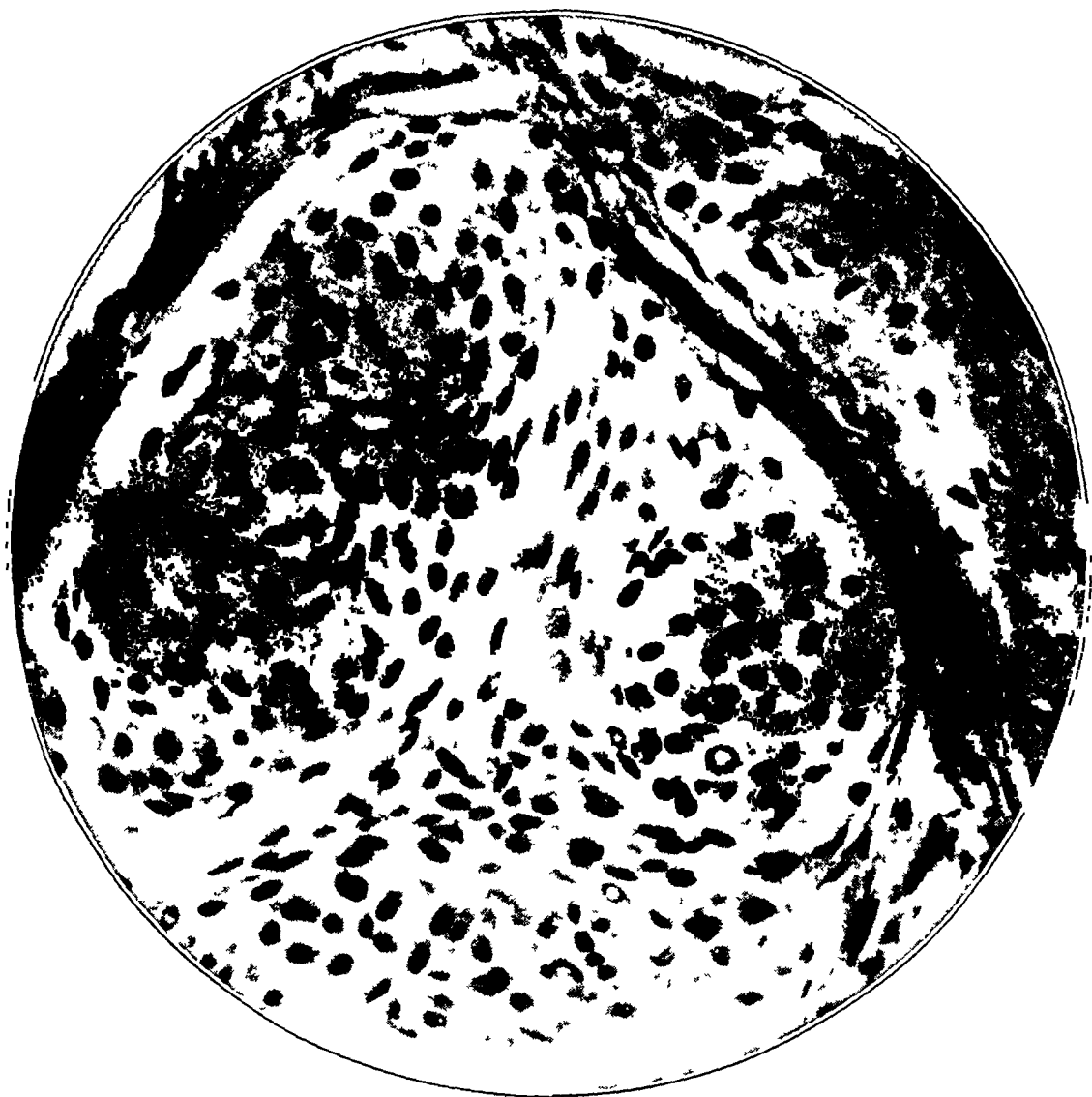


Fig 4—Photomicrograph showing the appearance of the tumor in the bone, high power magnification.

There is but little doubt that the meningioma originated at the temporal half of the fissure of Sylvius and involved the greater wing of the sphenoid bone and the temporal part of the lesser wing, extending to the anterolateral part of the frontal, and the anterior part of the temporal, bone. According to Goalwin, in the earlier stages of osseous hyperostosis caused by meningioma characteristic small erosions may be

present on the surface of the hyperostosis, which correspond to the points where the blood vessels enter from the tumor into the bone and are the points where the bony invasion begins



Fig 5—Roentgenographic appearance of skull in 1935

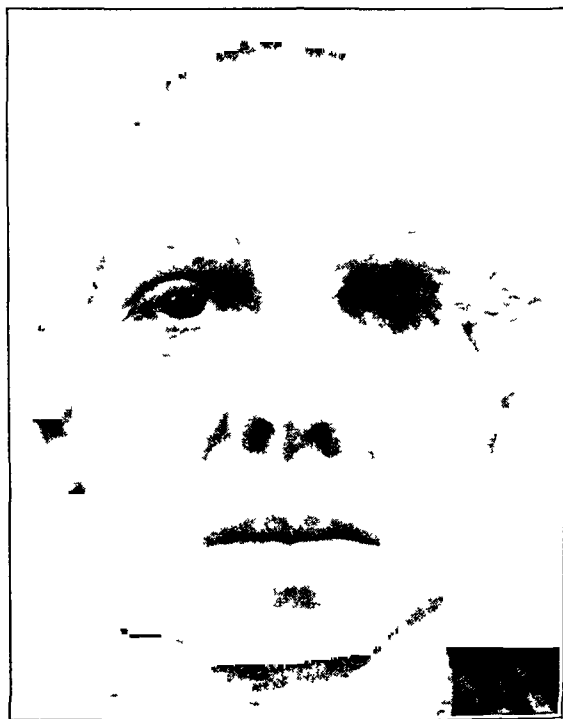


Fig 6—Appearance of patient on Sept 8, 1937

This case is reported to show that the process may remain stationary, whether this is due to the roentgen treatment or not cannot be determined

DISCUSSION

DR W L BENEDICT, Rochester, Minn (Roentgenograms were shown to demonstrate the three classes of bony tumor of the orbit, the exostoses, enostoses and hyperostoses) The distinction between these types of tumor is always clear enough to meet the needs of diagnosis and consideration of surgical intervention. The most common cause of orbital hyperostosis is meningioma, but it may occur in cases of primary bone disease due to constitutional dyscrasia or to inflammation or to tumor other than meningioma. When it is believed that vision can be helped or that the condition can be helped by surgical intervention, the method of approach must be carefully considered. Hyperostosis of the sphenoid ridge or of the lesser wing of the sphenoid bone, when accompanied by visual disturbance or by exophthalmos, usually points to meningioma about the sella turcica. Tumors in this position are favorably situated for surgical removal by the transcranial route. Meningioma of the optic nerve in the intraorbital portion has been described (Roentgenograms and slides showing tumor with hyperostosis of the orbital walls before and after operation were used to illustrate the advantage of the transcranial as against the transorbital approach.)

DR DANIEL B KIRBY, New York I wish to add my experience in a single case in order to stress the matter of early diagnosis, so that the neurosurgeon may operate before the meningioma invades the bones and produces hyperostosis and secondary changes. Hyperostoses are not sensitive to radium or roentgen rays, so the only recourse is surgical treatment. Dr Knapp pointed out that the meningioma has special sites of origin. Two of these favorite sites are the tuberculum sellae and the sphenoid ridge. My patient was a 65 year old woman. Her first symptom was that of blurred vision in the left eye, which started twenty years ago. There was reason to believe that the changes started in the region of the sphenoid ridge, and rather atypical changes were produced in the visual field. After one year her eye became prominent, and after twenty years it had increased to 32 mm, as measured by the exophthalmometer, and the entire eye was anterior to the lateral orbital margin. The vision and the field of vision in the right eye were normal. All the central vision and the field of vision in the left eye were gone two years after the beginning of the exophthalmos.

Roentgenograms showed an amazing degree of hyperostosis extending through all the walls and invading all the bones, entering into the formation of the orbit. It extended from the roof of the orbit posteriorly and covered the entire floor of the middle fossa, laterally it extended from the region of the tuberculum sellae over the sphenoid ridge out to the lateral wall. The optic canal was obliterated. Only a narrow free space remained in the nasal portion of the orbit. Keratitis developed, and it was necessary to remove the eye. In palpating the orbit one could feel only this narrow space in the nasal portion. Such an extensive bony change cannot be removed surgically. Neurologically, the patient had no objective signs of involvement other than the ocular changes.

The moral in a case like this is that one should exercise more care in obtaining visual fields and roentgenograms in every case of obscure loss of vision and in every case of exophthalmos.

INTRAOCULAR NEMATODE WORMS

REPORT OF A CASE AND REVIEW OF THE LITERATURE

LESTER T JONES, M D

LEWIS W JORDAN, M D

AND

NICHOLAS P SULLIVAN, M S

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Many parasites, such as tapeworms, fly larvae and nematodes, are reported in the medical literature as having been found "in the eye" In most instances, however, the parasites have been in the extraocular tissue or conjunctival sac, rather than actually intraocular Nearly all of the intraocular nematode worms found have been of the genus *Filaria* and were reported from the tropics

One of the most interesting cases is that described by Nayar and Pillai,¹ of India

On June 10, 1932, a native man 34 years of age came to the Government Ophthalmic Hospital at Madras, India, because of blurring of vision and a wormlike object floating about in front of his left eye for five days Examination of the fundus revealed a "threadlike wriggling worm, coiling itself and moving briskly, especially when light was thrown directly on it There was an oval clot of blood, about three fourths of the size of the optic disk, near the macula, to which one end of the worm was attached The worm appeared to be roughly about 3 mm long " Fourteen days later the worm disappeared, and after another five days it was found lying in the bottom of the anterior chamber, moving briskly when light was used Microfilariae found in the blood were of the *Filaria bancrofti* type After the worm was removed from the anterior chamber, it was lost

In 1934 Wright² reported the case of a Hindu aged 25 who consulted him for iritis of the right eye

A fine exudate was seen on the pupillary border, and there were flocculent deposits at the bottom of the anterior chamber with fine keratitis punctata Fine opacities of the vitreous were present Details of the retina were not clear Two round hemorrhages $\frac{1}{4}$ disk diameter from the macula were seen Twelve days later a worm was observed moving rapidly in the anterior chamber A fluorescent sheen gave rise to scintillations as it rapidly lashed and wound itself into a

Read before the Oregon Academy of Ophthalmology and Otolaryngology in Portland, May 17, 1938

1 Nayar, K K, and Pillai, A K A Case of Filariasis Oculi Brit J Ophth **16** 549-551 (Sept) 1932

2 Wright, R E Adult *Filaria* (*Wuchereria*) *Bancrofti* in the Anterior Chamber, Brit J Ophth **18** 646-650 (Nov) 1934

loose tangle of coils. It was translucent and faintly opaque. The tail was carried in the position characteristic of the male *F. bancrofti*, a sharp complete loop being maintained just in front of the anus. Punctate brownish deposits were seen all over the endothelium, and there were fine dustlike floaters in the aqueous. The worm was removed through a small keratome incision. The parasite was 22 mm in length and 0.09 mm in width, and it tapered toward the anterior end, which was slightly enlarged and bulbous. The posterior end was characteristically coiled. The worm was identified as *F. bancrofti*.

Fernando³ reported from Africa in 1934 the removal of an adult female of the species *F. bancrofti* from the anterior chamber of the eye. No microfilarias were found in the blood of this patient, who was not from an area in which *Filaria* was endemic.

*F. bancrofti*⁴ normally inhabits the lymphatics of the pelvis and lower part of the abdomen, where it causes lymphatic obstruction. Here it gives birth to living microfilarias, which are about 200 microns long and are present in the peripheral blood, usually only at night. Some of the microfilarias are taken up by the *Culex fatigans* mosquito, which acts as the most important vector. However, many different types of mosquito may transmit the disease. After a definite stage of development in the mosquito, the larvae are injected into a new host by a bite of the insect. In the new host the larvae quickly find their way to the lymphatics, where they develop into adults. After about twelve months microfilarias appear in the peripheral blood of the host. Typically, the patients may show lymphangitis of the involved lymphatics, which is followed later by the symptoms of chronic lymphatic obstruction, such as elephantiasis. However, the symptoms of lymphatic obstruction are usually not present in temperate climates. Occasionally the young larvae find their way into the blood stream, thus accounting for their presence in many different regions of the body.

The term *Filaria oculi humani*⁵ or *Agamofilaria oculi* Stiles⁶ has been applied to the organisms in a group of reported cases in which the worms were lost or were not definitely classified and identified after their removal from eyes. One of the earliest cases on record was reported by Mercier in 1771, the involvement occurred in the eye of a Negress Barkan,⁷ of San Francisco. In 1876 reported the case of a patient from

3 Fernando, S. E. Ocular Filariasis (Adult *Wuchereria Bancrofti* in the Anterior Chamber of the Human Eye), *J. Trop. Med. & Hyg.* **38** 17-18 (Jan 15) 1934.

4 Craig, C. F., and Faust, E. C. *Clinical Parasitology*, Philadelphia, Lea & Febiger, 1937.

5 Elliot, R. H. *Tropical Ophthalmology*, New York, Oxford University Press, 1920, p. 173.

6 Wood, C. A. *The American Encyclopedia and Dictionary of Ophthalmology*, Chicago, Cleveland Press, 1918, p. 9316.

7 Barkan, A. A Case of *Filaria Medinensis* in the Anterior Chamber, *Arch. Ophth. & Otol.* **5** 151, 1876.

Australia who had a threadlike worm attached to the lower part of the iris. After removal, the worm was thought by Knapp to be *Filaria medinensis*. However, it is now believed that this worm was probably an unknown Australian species. López,⁸ in Havana, saw an active, white threadlike worm 25 mm in length in the anterior chamber of the eye of a European woman 61 years of age. It had caused slight keratitis and iritis. The parasite passed into the posterior chamber, where it died. Permission for removal was refused by the patient. Von Nordmann⁹ found two dead filarias, which were threadlike and 2 mm in length, in a lens sent him by von Graefe. Gescheidt¹⁰ discovered three filarias, varying in length from 1.5 to 4.5 mm, in a cataract sent him by von Ammon, of Dresden. Scholer¹¹ in 1875 showed the members of the Medical Society of Berlin a living worm in a human lens. Virchow pronounced it a nematode, it was from 12 to 15 mm long and moved continuously.

REPORT OF A CASE

On Nov. 12, 1937, H. F., a widow aged 42, was seen complaining of pain in her right eye and epiphora. There was mild iritis, the lacrimal ducts were obstructed, and a large amount of fluid pus was expressed from both tonsils. Atropine was instilled into the conjunctival sac. Salicylates were used at home, and the iritis cleared in seven days. Vision was 20/15 in each eye. A diagnosis of iritis, probably due to tonsillar sepsis, was made.

On April 7, 1938, the patient stated that she woke up with some redness in the right eye, but no pain. At noon she had a sudden sharp pain in the eye. It was an agonizing pain, stabbing in character, and kept recurring at intervals of five minutes. She had severe headache. The redness and swelling of the eye increased. The patient consulted us that afternoon. We found circumcorneal injection, iritis and a contracted pupil. The fundus was normal. A large amount of pus was again expressed from the tonsils.

A 2 per cent solution of homatropine hydrobromide was instilled, and the patient was advised to use hot packs and to take salicylates. The diagnosis was iritis, probably due to tonsillar sepsis.

On April 8 the pain was still severe. The pupil had again contracted. A 1 per cent solution of atropine was instilled at the office. After the patient waited one-half hour, the lower third of the iris was still adherent to the lens. Epinephrine in a dilution of 1:100 was applied to the limbus, and the iris was retracted from the lens without evidence of pigment deposits. The application of a 1 per cent solution of atropine was prescribed three times a day.

On April 11 the eye was greatly improved.

On April 13 the patient felt worse again. The tension was found to be 55 mm (Schiotz). The use of the atropine was discontinued. Physostigmine salicylate was instilled at intervals of five minutes, until the pupil contracted moderately.

8 López, E. *Filaria en la camara anterior*, Rev. de cien. med. **6**: 269 (Dec. 5) 1891.

9 von Nordmann, A. *Mikrographische Beiträge zur Naturgeschichte der wirbellosen Tiere*, Berlin, G. Reimer, 1832.

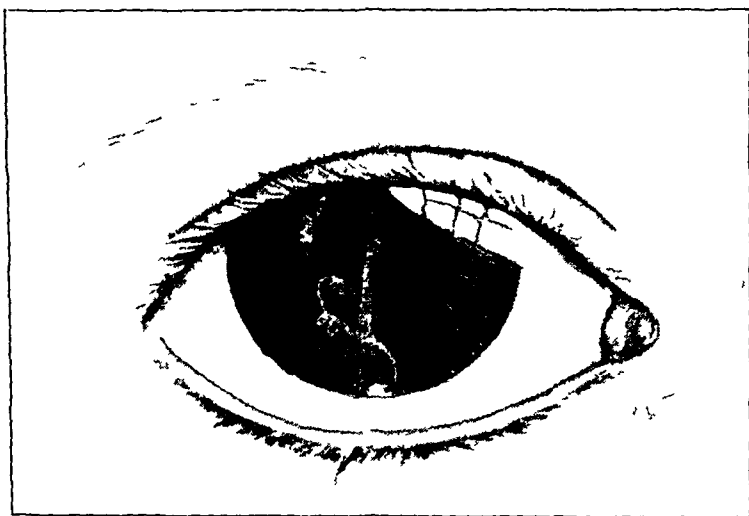
10 Gescheidt. *Die Entozoen des Auges. Eine naturhistorische, ophthalmomonosologische Skizze*, Ztschr. f. d. Ophth. **3**: 405, 1833.

11 Scholer. *Berl. klin. Wchnschr.* **12**: 682, 1875, **13**: 8, 1876.

On April 14 the tension was still increased. Dr Clinton Cooke was called in consultation and discovered a worm in the anterior chamber.

On April 18 the patient returned to our office. An adult type of nematode worm was easily seen in the anterior chamber of the right eye. Moving pictures of its rapid coiling movements were obtained. The visible part was about 7 to 9 mm in length and 0.1 to 0.2 mm in diameter. It seemed to emerge from an oval pocket at the base of the iris, in which it was attached. Like *F. bancrofti* of Wright, it seemed to taper toward its free end and to throw itself into a "loose tangle of coils," especially when strong light was thrown on it. The patient was referred to Dr Merle Taylor, who gave the following report:

"The anterior chamber is of normal depth. There is a slight increase in cell content in the aqueous. Six keratic precipitates are seen on the posterior surface of the cornea at the lower border of the pupillary area. The iris is clear, except at the point of attachment of the worm."



Appearance of the eye with a living adult form of nematode attached to the iris

No parasites or ova were found in the stool on repeated examinations. No microfilariae were found in smears of the centrifuged blood taken during either the day or the night. Eosinophilia was not present.

On April 23 the worm was removed by Dr C Cook. As the worm was lost, it was impossible to tell its actual length, type, etc.

This patient had never been in the tropics. She had lived in Los Angeles from 1927 to 1933, in Sacramento ten years prior to that and in Portland for the last five years. She had a severe attack of herpes oculi in 1933, which lasted for two months. Two years before the present complaint she was bitten by a tick at Wind River, Wash. This bite was swollen and painful for six days. Six months later, while on a hunting trip at Wind River, she was severely bitten by mosquitoes and had a swollen face for several days.

COMMENT

For the present, at least, this parasite must be classed as one of the so-called Agamofilariae. We might assume that it was *F. bancrofti*. This is supported by several facts. Our worm seems to conform in diameter and gross morphologic structure with this type. The most

common vector of *F. bancrofti*, the *Culex fatigans* mosquito, is present in the vicinity in which our patient was living. It is possible that many persons with microfilarias of the *F. bancrofti* type in their blood have entered such a seaport city as Portland and have acted as sources of infestation. However, our patient showed no eosinophilia and no microfilarias in the blood and had no symptoms aside from those referred to the eye. The adult specimen of *F. bancrofti* is usually described as being at least 22 mm in length¹². The small size of our worm might be explained by its not being removed completely or by the fact that filarias removed from the eye are usually small and immature and represent a youthful migratory stage. The infestation may have occurred several months before, and the first attack of iritis may have marked the implantation of the small blood-borne larva in the ciliary body or iris.

DIFFERENTIAL DIAGNOSIS

Because of the smooth cuticle and the absence of segmentation, we can designate the worm removed from our patient as a nematode. In addition to *F. bancrofti*, several other possible species must be considered. *Filaria loa*, or the "eye worm," can probably be eliminated by the absence of its vector, *Chrysops dimidiatus* (mango fly), from the regions in which the patient was living. In addition, our patient had no Calabar swellings, which are characteristic of this condition. *Ascaris lumbricoides* must be considered, because at one stage of its development its larvae are present in the blood stream. However, the ascaris even in its early forms is a broad and heavy worm, while our worm had a thin, threadlike structure. *Oxyuris vermicularis* (pinworm) and *Trichuris trichiura* (whipworm) pass their entire life cycle in the gastrointestinal tract and never at any time enter the blood stream or lymphatics. They can therefore both be ruled out. The adult form of *Trichinella spiralis* (*Trichina*) is never longer than about 3 mm. The fact that the visible part of our worm was at least 9 mm long completely excludes this possibility. *Ancylostoma duodenale* and *Necator americanus* (hookworms) are rather stiff worms and do not show the rapid coiling movements manifested by our worm. We have been unable to find a report in the literature of an intraocular adult hookworm. This parasite can probably further be eliminated by the total absence of clinical findings and ova in the stool.

Outside of the nematode group, the tapeworms *Taenia solium* and *Taenia echinococcus* may be considered. However, when these worms occur in the eye they are in a larval cystic form with a short protruding

12 Wright, R. E., Iyer, P. V. S., and Pandit, C. G. Description of an Adult *Filaria* (Male) Removed from the Anterior Chamber of the Eye of Man, Indian J. M. Research **23** 199-203 (July) 1935.

neck This in no way resembles the worm under consideration Fly larvae have been found within the eyeball many times They always have a spindle-shaped segmented form, which in no way compares with a coiling nematode A *Schistosoma* blood fluke can be ruled out because it is a flat worm, while ours was a round one In addition, it has never been reported as found in the eye

That this worm was a parasite normally infesting animals in the northwestern states and appearing in man as an erratic is a possibility Filarias have been found in the ventricles of hearts of dogs in the Southern states Dr R Parker, of the Rocky Mountain Laboratory of the Public Health Service in Hamilton, Mont, in a personal communication, made the following statement

A number of wild and domestic animals in the United States are infested with *Filaria* This group includes deer, porcupines, moose, horses, cattle, and jack rabbits Worms have been found in the eyes of moose and horses Little is known of their insect vectors or of the ability of these filariae to infest man

Many other *Filariae* which infest human hosts have been described Most of these were found in the tropics However, none of them has been observed within the eye

OTHER CASE REPORTS IN THE LITERATURE

Bachelet,¹³ of Gabon, Africa, in 1880 reported the removal of a worm of the species *F. loa* from the eye of a native young man It was on the iris beneath the sclera In 1894 Coppez¹⁴ reported the removal of a dead worm of the same species, 15.2 mm in length, from the eye of an infant Negress who came from the Congo It was rather incompletely identified *Filaria equina* is often found in the abdominal cavity of the horse in India Drake¹⁵ reported removing a worm of this type from the eye of a young woman in Madras, India Since that time the identity of this particular specimen has been doubted

Calhoun,¹⁶ of Atlanta, Ga., in 1937 found an *Ascaris lumbricoides* larva in the anterior chamber of the eye of a boy No ova were found in the stools, and there was no eosinophilia In the same year, Tso-Chen-Te¹⁷ described a specimen of *Cysticercus cellulosae* attached

13 Bachelet, H. M. The Eye-Parasite, *Dracunculus Loa*, M. Rec. New York **17** 244, 1880

14 Coppez, H. Un cas de filaire dans la chambre anterieure d'un oeil humain, Arch. d'ophth. **14** 557-562, 1894

15 Drake, B. Ophth. Rev., London **13** 331, 1894

16 Calhoun, F. P. Infra-Ocular Invasion by the Larva of the *Ascaris*, Arch. Ophth. **18** 963-970 (Dec.) 1937

17 Tso, C. T. Intraocular Cysticercosis, Chinese M. J. **51** 545-548 (April) 1937

to the retina. There were no ova in the stools. In 1933 Wilson¹⁸ reported a small thread worm of the *Onchocerca volvulus* type attached to the macula.

SUMMARY

A case is reported in which a living adult form of nematode, measuring about 9 mm in length and 0.2 mm in diameter, was found attached to the iris of a patient who had never been in a tropical country. This parasite was classified as an *Agamofilaria* because it was lost after its removal and because there were no other physical or laboratory findings to assist in its identification. The differential diagnosis is presented, and cases of intraocular parasites reported in the literature are reviewed.

¹⁸ Wilson, R. P. Onchocerciasis of the Macula, in Eighth Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo, Schindler's Press, 1933, pp. 85-87.

SCLEROMALACIA PERFORANS

REPORT OF A CASE IN WHICH THE EYE WAS
EXAMINED MICROSCOPICALLY

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AND

MERRILL J KING, M D

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Van der Hoeve¹ in 1930 described before the Dutch Ophthalmological Society 2 cases of an ocular condition which he regarded as a clinical entity and designated scleromalacia perforans. In 1932 Rochat² described before the same society 2 similar cases under the designation scleritis necroticans. In 1934 van der Hoeve³ reported in somewhat more detail his own 2 cases and those of Rochat. Including these cases, we have been able to find in the literature references to only 14 cases of this ocular condition⁴. The inclusion of 1 of these cases was doubtful. The patient was aged only 34, there was no arthritis, and the only lesion consisted, even when the patient was first observed, of a filtering cicatrix at the corneal limbus⁵. Three cases, mentioned in the discussion on Rochat's communication, were described simply as being closely similar to his cases, with the exception that in 1 there was no arthritis. We have analyzed the data obtainable from the reports of these 14 cases.

From the Howe Laboratory of Ophthalmology, Harvard University, and the Massachusetts Eye and Ear Infirmary

Read before the Section on Ophthalmology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 15, 1938

1 van der Hoeve, J. Scleromalacia perforans, *Nederl tijdschr v geneesk* **2** 4733, 1931, abstracted, *Zentralbl f d ges Ophth* **26** 504, 1932

2 Rochat, G F. Scleritis necroticans, *Nederl tijdschr v geneesk* **77** 1935, 1933

3 van der Hoeve, J. Scleromalacia Perforans, *Arch Ophth* **11** 111 (Jan) 1934

4 These cases are referred to specifically in the text with the exception of that reported by C T Eber (Fistula at Limbus [Scleromalacia Perforans], *Am J Ophth* **17** 921, 1934)

5 Arkle, J S, and Ingram, H V. Scleromalacia Perforans, *Tr Ophth Soc U Kingdom* **55** 552, 1935

ANALYSIS OF FOURTEEN RECORDED CASES OF
SCLEROMALACIA PERFORANS

Age—Of 9 patients, the youngest was 54 and the oldest 76. The patient in the doubtful case was aged 34. The age of 2 of the 4 other patients was not given, and 2 were said to be "old."

Sex—Seven of the patients were females and 5 were males. The sex of 2 was not stated.

Eyes Affected—Both eyes were affected in 8 cases and 1 eye in 3 (including the doubtful case). It was not stated which eye was affected in 3 cases.

Situation of Scleral Lesions—The lesion was situated between the corneal limbus and the equator in 8 cases and at the limbus only in 3 (including the doubtful case). The location of the lesion in 3 cases was not stated.

Character of Scleral Lesions—The scleral lesion was not definitely described in 3 cases. There were only scleral cavities in 8 cases (including filtering cicatrices at the limbus in 3), nodules and cavities in 3 cases (nodules described as abscesses in 2) and "sequestrums" in cavities in 2 cases.

Period Between First and Last Observation—There was a period of one year between the first and last observation in 1 case, of eight months in 1 case and of four months in 1 case. The intervals between examinations in 11 cases were not definitely stated.

Recurrences—There were no definite data as to recurrence.

Ocular Complications—There were no central corneal opacities in any case. Descemetitis was present in 1 case, posterior synechia in 2 cases, atrophy of the iris with holes in the iris in 1 case, cataract in 3 cases (in only 1 was it secondary to the scleritis), marked opacities of the vitreous in 1 case, staphyloma in 1 (ruptured) and purulent infection in 1 (both eyes). The presence of glaucoma was not mentioned. The fundus was normal in 1 case, and its appearance in the remaining 13 cases was not mentioned.

Visual Acuity—The visual acuity was normal or was only slightly affected in 4 cases (including the doubtful case) and was markedly reduced in 6. The status of the visual acuity in 4 cases was not given.

Biopsy of Active Lesions—Biopsy was done in 2 cases, "granulation tissue" and "chronic inflammatory tissue" being reported.

Bacteriology—Cultures were made in 2 cases (negative), and animal inoculations were carried out in 1 case (negative).

Association with Rheumatoid Arthritis—Arthritis was present in 10 cases and absent in 4 (including the doubtful case). The duration of

the arthritis before the onset of scleritis was ten years in 1 case, twenty-one years in another case, "chronic" in 2 cases and not reported in 10 cases

In 1 case⁶ there were scleroderma and porphyria in addition to rheumatoid arthritis (It seems unlikely that either of these conditions were of etiologic significance in regard to the ocular disease)

From the reports of these recorded cases we have composed the following clinical description of the disease. In one or both eyes there develop simultaneously or at intervals slightly elevated nodules involving the sclera and overlying tissue and situated anywhere between the corneal limbus and the equator. Congestion is moderate and is limited to the nodules and their immediate vicinities. After a considerable period, in some cases six months or longer, one or more of the nodules may disappear. At the former site of a nodule there is seen in the sclera a shallow cavity, often of large size. Over a cavity the conjunctiva may be intact or absent. When, as rarely happens, a lesion is situated at the corneal limbus, it may perforate into the anterior chamber and produce a cystoid nodule such as occurs after trephining. Into this nodule the iris may or may not be herniated. When situated elsewhere, the lesions seldom if ever perforate the sclera, and the remaining tissue usually withstands the intraocular pressure, rupturing only in rare instances. Evidences of intraocular inflammation are relatively slight or entirely lacking, unless, as rarely happens, purulent infection occurs at the site of a cavity. Pain may be absent. Posterior synechiae, atrophy of the iris or cataract may occur as late complications in cases of marked involvement. Usually the ocular disease does not occur before the patient reaches the age of 50. In the great majority of cases definite evidences of rheumatoid arthritis exist at the outset of the scleritis.

We have had opportunity to study clinically, bacteriologically and histologically the following case of scleromalacia perforans, the first in which an entire eye affected with this disease has been obtained for microscopic examination.⁷

REPORT OF CASE

History—C. M., a married white man aged 52, was admitted to the Massachusetts Eye and Ear Infirmary on Dec. 9, 1936, complaining of a "sore tumor" on the left eye, poor vision in this eye and headaches.

He recalled that his left eye became red about one year before and was certain that the redness was localized in the outer portion of the eye. The eye became "sore," and the redness increased somewhat. He had noticed the "tumor" for at

⁶ Kiehle, F. A. Scleromalacia. Report of a Case, *Am J Ophth* 20:565, 1937.

⁷ The bacteriologic investigations were made by Dr. King. The histologic investigations and the deductions from them were made by Dr. Verhoeff.

least three months. The vision had been poor in the eye for several years. For a few weeks before admission to the infirmary he had had frequent attacks of severe headache, with pain radiating down the back of the neck. There was no history of trauma.

About fifteen years before, the patient first noticed that he had some soreness of the feet—they were tired and ached after he had been standing for only a moderate length of time. About fourteen years before, he had his appendix removed, and about eleven years before he was seriously ill for four months with "inflammation of the bowels." At about that time he began to have pain and swelling in the joints. These began in the ankles and gradually affected practically every joint in his body. He continued to work, however, until seven years before the present attack, at which time pleurisy developed. He was then hospitalized for a period of about three months and was discharged from the hospital with a final diagnosis of rheumatic fever and pleurisy with effusion. All examinations in that hospital as well as at a tuberculosis sanatorium had failed to show evidence of a tuberculous origin for the pleurisy. Since this acute rheumatic episode he had been compelled to use crutches or a wheel chair, and his arthritic condition had remained about stationary.



Fig 1—Appearance of the left eye when the patient was first seen on Dec 9, 1936. One lower nodule is hidden by the lower lid.

The family history was essentially unimportant.

Ocular Examination—The vision of the right eye was 20/20, and the eye was normal. The vision of the left eye was 6/200 with correction. The cornea was clear, the pupil was round and reacted normally. The media were clear. The tension was normal. Situated on the sclera, at the outer side of the eye, were five smooth, waxy-appearing nodules (fig 1). The largest of these, about 5 mm in diameter and elevated about 2.5 mm, was situated slightly below the horizontal meridian, with the anterior border about 2.5 mm from the corneal limbus. The other nodules, each about 3 or 4 mm in diameter, were situated close to the largest nodule, two being above and two below it. The nodules were covered by conjunctiva and were immobile, firm and tender to pressure. The conjunctival vessels around the nodules were considerably distended. Clinically, the picture somewhat suggested tuberculous nodular scleritis.

Examination of the Fundus—The optic disk was normal. In the macular region there were fine changes which suggested cystoid degeneration. The fovea was poorly defined. The retinal vessels showed no definite sclerosis. There were a few drusen. In the upper temporal region there were several small pigmented spots and one fairly large irregularly pigmented area of healed chorioretinitis.

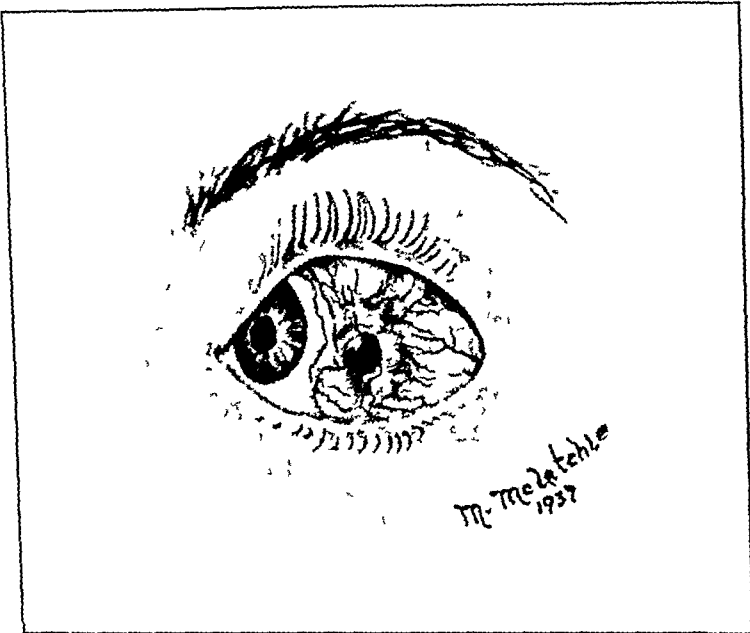


Fig 2 —Appearance of the left eye just before its removal four months later

Physical Examination—General physical examination made by members of the staff of the Massachusetts General Hospital revealed the presence of rheumatoid arthritis, with involvement of the hands, wrists, elbows, shoulders, feet, ankles, knees and hips and the cervical portion of the spine. Old fibrous pleurisy was the only other important finding.

Laboratory Examinations—The Wassermann reactions of the blood and spinal fluid were negative. The gonococcus fixation test gave negative results. The sputum did not show any tubercle bacilli. The urine was normal. The blood contained 90 per cent hemoglobin, 14,500 white blood cells and 80 per cent polymorphonuclears. Chemical examination of the blood showed 93 mg of sugar per hundred cubic centimeters, 23 mg of nonprotein nitrogen and 3.16 mg of uric acid (plasma). The blood pressure was 140 systolic and 92 diastolic. The reaction to the Mantoux test with 0.1 mg of tuberculin was negative, while that with 1 mg was positive. The prostatic secretion did not contain any white blood cells or cocci. The electrocardiograms were within normal limits. Roentgen examinations revealed a picture consistent with that of rheumatoid arthritis. There were no calcified nodes in the chest, neck or abdomen. The apical pleurae were thickened.

One week after the patient's admission to the infirmary it was decided to remove a bit of the largest nodule for histologic and bacteriologic studies. The conjunctiva was dissected from this nodule, and with a sharp knife a horizontal incision was made into the nodule just below its midpoint. Immediately on withdrawal of the knife about 3 drops of thick yellowish material escaped from the incision. This material was collected, and a small piece of the wall of the nodule was excised. The conjunctiva was then carefully sutured at the site of the incision. The conjunctiva healed quickly, and the patient was discharged to his home.

Laboratory Examinations of Biopsy Material—Smears made of the purulent material which was obtained at the time of performing the biopsy were stained with Giemsa's and Wright's stains. Only pus cells and occasional plasma cells were identified in these smears.

Other smears were stained by the Gram method and with methylene blue, dilute fuchsin and the Ziehl-Neelsen stain. In none of these smears were bacteria or fungi found.

The remainder of the purulent material together with a portion of the excised tissue was ground in a mortar. Some of this mixture was transferred to various kinds of bacteriologic mediums for cultural studies. The remainder was then injected beneath the conjunctiva of a rabbit's eye and into the groin of a guinea pig.

A portion of the excised tissue was fixed in Zenker's solution for histologic examination.

All attempts to cultivate bacteria and fungi aerobically, as well as under partially increased carbon dioxide tension, proved fruitless. The results of the animal inoculations were also negative. The guinea pig was found to be normal when it was killed two months after the injection. In the rabbit's eye, beneath the conjunctiva at the site of the injection, a slight reaction developed, but this permanently disappeared within four days.

Microscopic Examination of the Excised Tissue—The specimen consisted of episcleral and scleral tissue greatly infiltrated with lymphocytes and plasma cells but entirely free from purulent infiltration. Adherent to it was a small mass of pus cells, among which were a considerable number of eosinophils—about ten in a high power field. Presumably this mass of cells was part of the secretion from within the nodule.

Subsequent History—Eight weeks after the biopsy was done the patient returned to the clinic for admission to the arthritis service at the Massachusetts General Hospital. He stated that the "tumor" had since gradually decreased in size, but that the ocular pain had persisted. The right eye still remained normal, with vision of 20/20. The left eye was considerably injected in the temporal half. Its visual acuity was still 6/200. The largest nodule had entirely disappeared, and in its place there was an almost circular depressed area or cavity in the sclera about 4.5 mm in diameter. The area had a dark bluish appearance, and its anterior margin was 3.5 mm from the corneal limbus. Around it the other nodules remained. These nodules were still smooth and waxy and were covered by conjunctiva and elevated about 2 mm. The tension of the eye was normal, and the cornea was clear. The pupil reacted to light and in accommodation. The media were clear, and the fundus picture had not changed.

Progress in Arthritis Ward—The patient was placed on an extensive regimen in the hope that the advance of his arthritis might be stopped and some of his deformities corrected. A subcutaneous nodule on the right elbow was excised for microscopic examination. Laboratory examinations at this time revealed nothing new except that secondary anemia had developed (red blood cells, 3,300,000, hemoglobin, 70 per cent). The patient was therefore given several transfusions during the next few weeks. The arthritis improved considerably, but the ocular condition did not. One month after the patient's admission the eye was so painful that a hypodermic injection was necessary. The globe was extremely tender to touch. It was becoming more congested in the affected region, and the sclera in the depressed area was apparently becoming thinner. On April 12, 1937, two months after admission, the eye showed the appearance depicted in figure 2. The lesions appeared to be about the same as when previously seen, except that now a nodule situated at the posterior margin of the cavity was observed.

The patient continually begged to have the eye removed. Enucleation was finally agreed on because of the fact that the scleral process was progressing, because of the severe ocular pain and because of the low visual acuity of the eye. It was feared that perforation of the globe might occur should the process progress much further. The left eye was enucleated on April 17, at which time the scleritis had existed certainly seven months and possibly fifteen months. The patient made an uneventful recovery from the operation and was discharged from the hospital one month later.

Histologic Examination of Left Eye—The eye was fixed for forty-eight hours in a 4 per cent solution of formaldehyde and immersed for twenty-four hours in a solution of 2.5 per cent hydrochloric acid in 70 per cent alcohol. It was then opened (the retina was found to be in situ) and embedded in pyroxylin. Horizontal sections were made through the portion of the eye containing the lesions in the sclera and fundus. A part of the block containing the scleral lesions was later excised, embedded in paraffin and cut in serial sections. Most of the sections examined were stained with hematoxylin and eosin.

On microscopic examination the essential pathologic process was found to be confined to the anterior portion of the sclera and nowhere to extend farther back than 3 mm behind the ora serrata. Sections passing through the lower two scleral nodules showed in the center of each nodule a large abscess about 2.25 mm in length and 0.4 mm in thickness (fig 3). Each abscess consisted of a central mass of pus cells surrounded by a wall of epithelioid cells from four to six cells thick. The epithelioid cells were most often arranged radially with respect to the abscess, and among them there was an occasional giant cell of the Langhans' type. Most of the pus cells within the abscesses were necrotic, their nuclei showing chroma-

tolysis, pyknosis or fragmentation. Macrophages were not recognizable here or elsewhere in the tissues. In some sections remains of necrotic scleral tissue could be definitely recognized within the abscesses. The two abscesses were separated by a space of only about 0.5 mm.

Outside the walls of epithelioid cells many of the clefts in the sclera, especially those containing vessels, were distended with plasma cells and many fibroblasts. Some of the plasma cells showed colloid degeneration. Eosinophils were absent. The original sclera near the abscesses was being destroyed by the infiltrate and replaced by new fibrous tissue. There were few if any new vessels. Where the infiltrate was dense, the arteries showed marked obliterative endarteritis. The sclera was everywhere free from extravasated blood, blood pigment, edema and fibrin. Where the inflammatory process was marked, it also involved the underlying uvea, the stroma of which was here largely replaced by new fibrous tissue, chronic

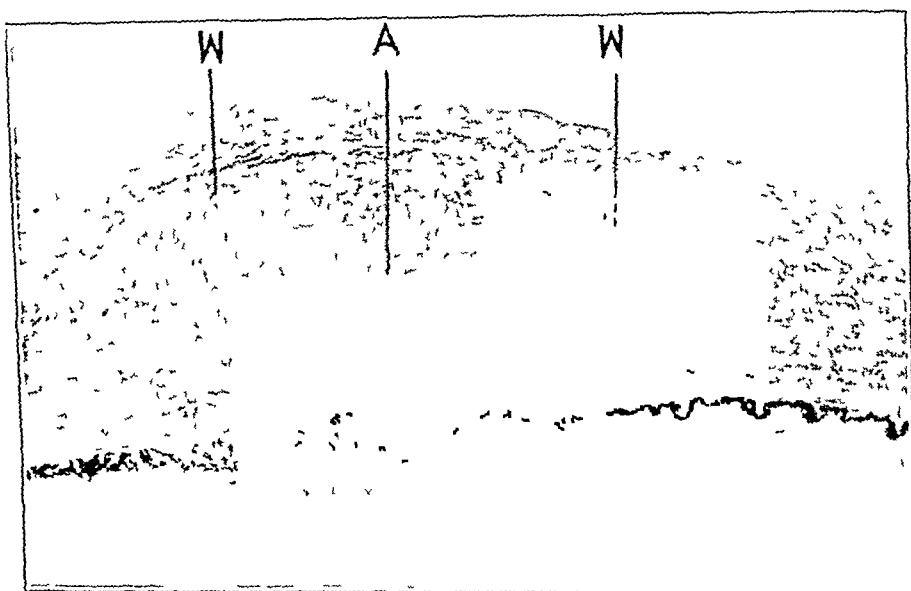


Fig 3—Photomicrograph showing a large abscess (*A*) in the sclera surrounded by a wall of epithelioid cells (*W*), $\times 27$. The conjunctiva is absent here as the result of the enucleation.

inflammatory cells and fibroblasts, down to the unpigmented layer of ciliary epithelium. This epithelium was unbroken but was altered by edema and separated from the overlying tissue in the form of numerous small vesicles containing mononuclear cells and a few pus cells. The pigmented layer of ciliary epithelium had almost completely disintegrated, and great numbers of pigment cells were disseminated through the tissue. In some places, but never farther back than 1 mm behind the ora serrata, the choroid had been disintegrated by the cellular infiltrate, which here had broken through the pigment epithelium and lifted up the retina. The latter, however, was only slightly altered. In spite of the marked chronic inflammatory reaction in the sclera and uvea, relatively few cells had exuded into the vitreous anywhere, and there was almost no tendency to the formation of cyclitic membranes.

In the affected region the whole thickness of the sclera proper was involved in the inflammatory process. The overlying episclera and conjunctiva showed slight edema, considerable congestion and infiltration with chronic inflammatory

cells, chiefly plasma cells. The entire ocular wall, even at the sites of the abscesses, was only about 1.5 mm thick in the sections.

Sections passing through the upper two nodules showed essentially the conditions just described. One of the abscesses here was longer and narrower than any of the other large abscesses. It was noteworthy that almost no pus cells could be seen approaching any of the abscesses from outside their walls. In none of the sections was an abscess found which was being invaded by blood vessels and fibroblasts, that is to say, which was undergoing organization.

Sections passing through the cavity in the sclera showed a different picture (fig 4). For a distance of about 3 mm the conjunctiva and episclera were absent, and the sclera and underlying uvea had been largely replaced by fibrous tissue. This was coated externally with new epithelium and was only slightly infiltrated with inflammatory cells. The ocular wall was reduced to a thickness of only 0.1 mm. The tissue replacing the uvea was markedly pervaded with pigment cells derived from the original pigmented epithelium. The unpigmented layer of ciliary epithelium had not been broken through. Anteriorly and posteriorly the original

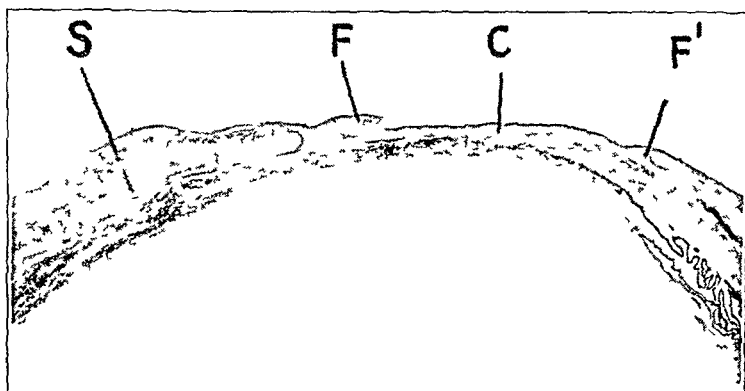


Fig 4—Photomicrograph showing a cavity (C) in the sclera coated with epithelium, flaps of conjunctiva (F and F') extending over the margins of the cavity, and a disintegrating new sequesterum (S) situated posterior to the cavity, $\times 10$. In some other sections the sclera is somewhat thinner at the site of the cavity.

conjunctival and episcleral tissue extended over the margin of the cavity in the form of a flap (fig 4), the under surface of which was coated with new epithelium. This new epithelium extended for a long distance deeply into the new fibrous tissue, just as the surface epithelium may do in cases of indolent ulcers elsewhere, and at one place formed a small cyst.

Just posterior to the thinned area described there was a nodule which showed a still different stage in the inflammatory process. Here at about the middle of the sclera was a stretch of necrotic tissue about 3 mm long and 0.25 mm in its greatest thickness. It was easily recognizable as a sequesterum of sclera (fig 4). It showed all stages in transformation from simple hyaline necrosis to almost complete granular disintegration. At the posterior end, where the sequesterum was hyaline and only slightly infiltrated, a wall of epithelioid cells was directly applied to it (fig 5). Elsewhere the sequesterum was surrounded by, and was being invaded from, an irregular zone of pus cells. Around this the wall of epithelioid cells continued, but in some places it had been so disintegrated by the pus cells as to be

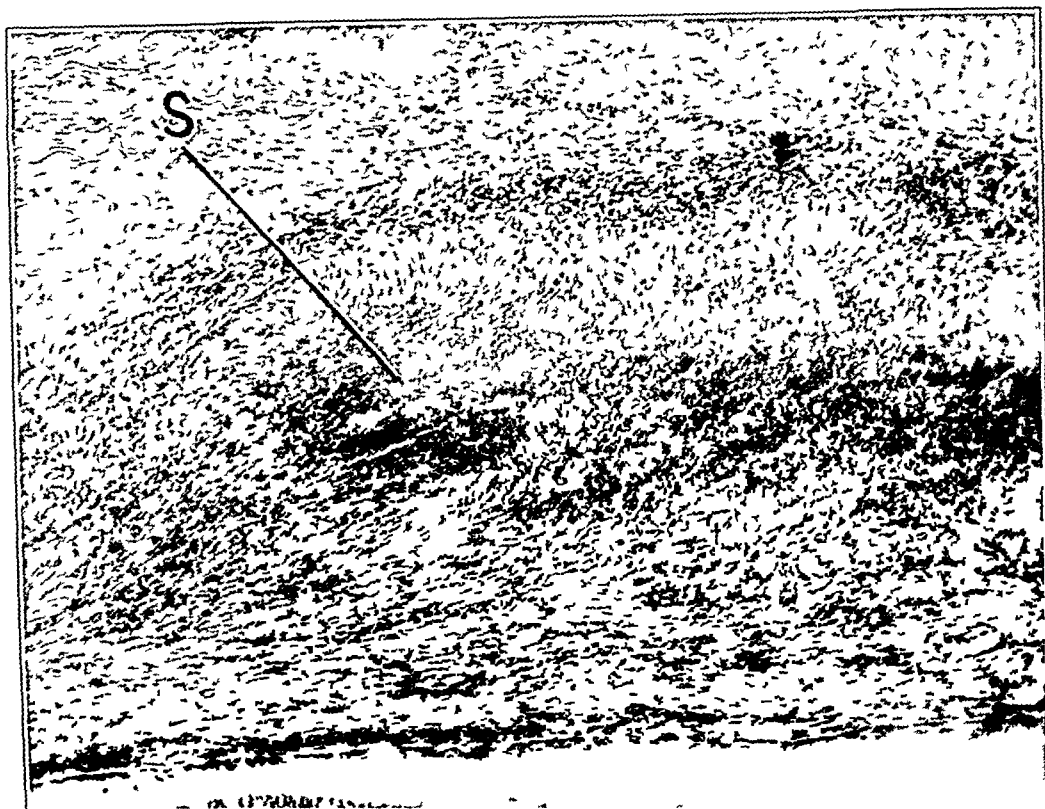


Fig 5—Photomicrograph showing the posterior end of the same sequestrum surrounded by a wall of epithelioid cells, $\times 85$. At *S* the sequestrum is only slightly infiltrated and is not disintegrated, proving that the formation of the wall of epithelioid cells precedes the formation of an abscess.

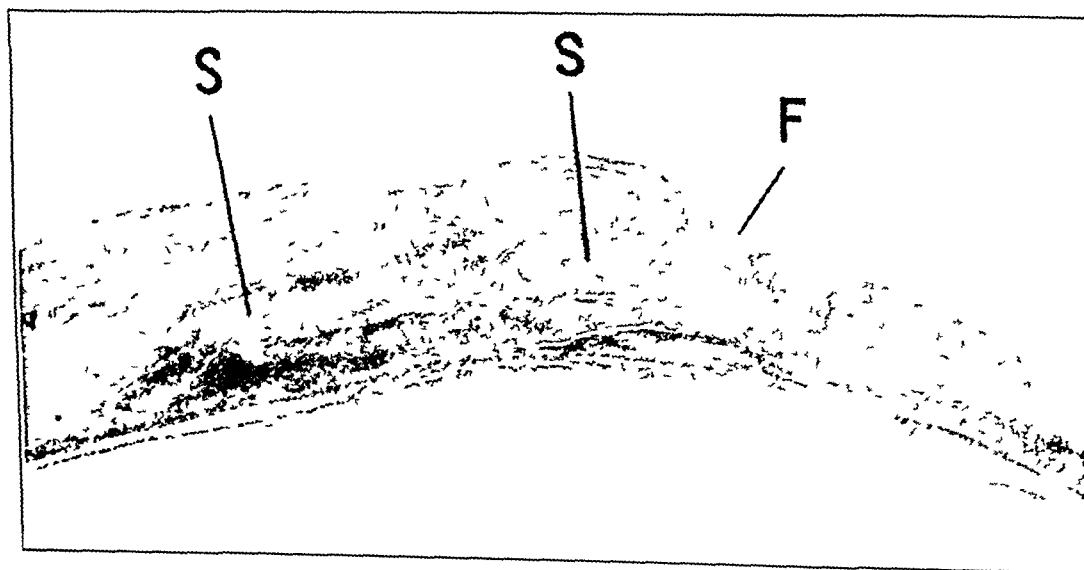


Fig 6—Photomicrograph showing a fistula (*F*) at the anterior end of same sequestrum (*S*), $\times 20$.

recognizable only with difficulty. At its anterior end the sequestrum was greatly disintegrated, and from the cavity it occupied there extended to the surface through a break in the conjunctiva a small fistula (fig 6). That this break was not an artefact was evidenced by the fact that the epithelium had proliferated downward and had begun to line the wall of the fistula. The entire fistula was included in a complete series of paraffin sections made through this region. In some sections the downgrowth of epithelium, extending from beneath the flap of conjunctiva, as previously described, reached the cavity of the sequestrum. A similar but much

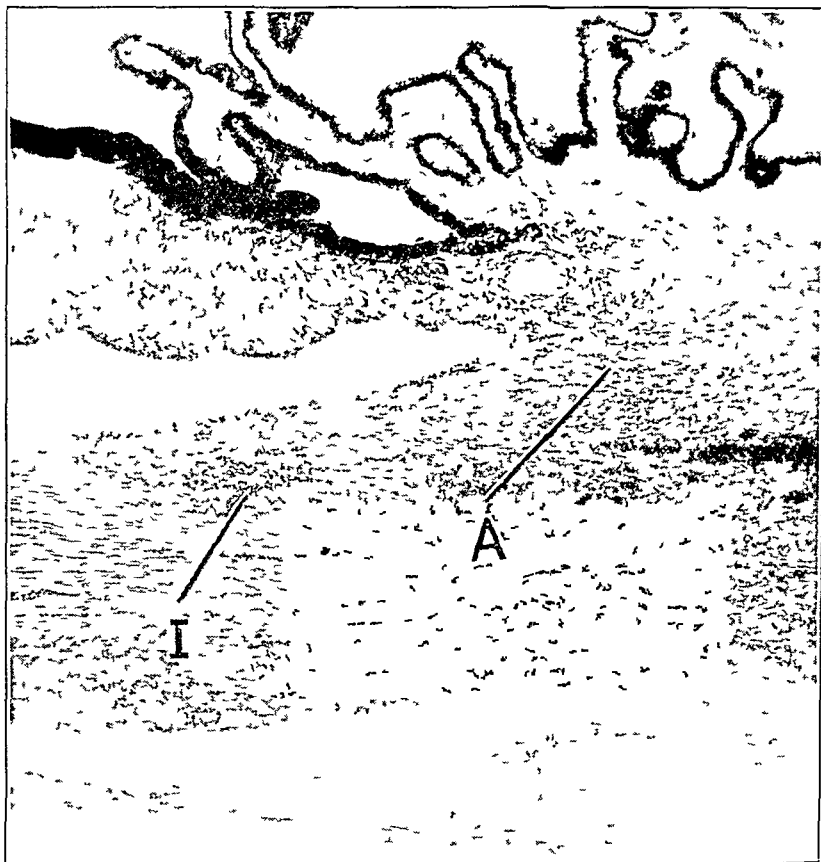


Fig 7—Section near the horizontal meridian of the eye, $\times 55$. *A* indicates a minute abscess near the filtration angle, surrounded by epithelioid cells, and *I*, an infiltrate in the region of Schlemm's canal. Note the absence of infiltration in the ciliary processes and the slight infiltration of the iris and ciliary body.

smaller sequestrum, not yet completely infiltrated, was found just beyond at the upper inner margin of the thinned area of the sclera.

In addition to the large abscesses, relatively minute foci, evidently of the same nature, were seen here and there within the affected region of the sclera. One such focus, situated near the canal of Schlemm, is shown in figure 7. This consisted of a small dense mass of necrotic pus cells, surrounded by a wall of epithelioid cells. It was evident that this focus had destroyed the scleral fibers here and not simply distended a scleral cleft. In some of these small foci remains of necrotic tissue could be seen. In several sections there was found near the abscesses a large intrascleral nerve loop surrounded, and to some extent invaded, by chronic inflam-

matory cells and fibroblasts. Several other large nerves, seen within the sclera and the anterior portion of the uvea in the affected region, were similarly involved.

In all sections the inflammatory process in the sclera ended posteriorly with notable abruptness, within a distance of 1.5 mm it was marked and completely disappeared. Anteriorly it ended less sharply but did not extend into the cornea. The tissue of the limbus showed relatively slight infiltration, chiefly with plasma cells. The filtration angle was blocked by the root of the iris only opposite the excavated portion of the sclera. Where the angle was open in the affected region, the ligamentum pectinatum was in general only slightly infiltrated, chiefly with plasma cells. In many sections, however, the site of the canal of Schlemm was occupied by a dense cellular infiltrate, which often continued into the sclera along vessels communicating with the canal (fig. 7).

Elsewhere than in the immediate vicinity of the affected portion of the sclera, the ocular tissues were almost free from inflammatory reaction. Posteriorly to the lesions, the infiltration of the choroid soon became perivascular and completely disappeared at the equator. The retina showed insignificant perivascular infiltration, which also disappeared at the equator. Even where closest to the scleral abscesses,

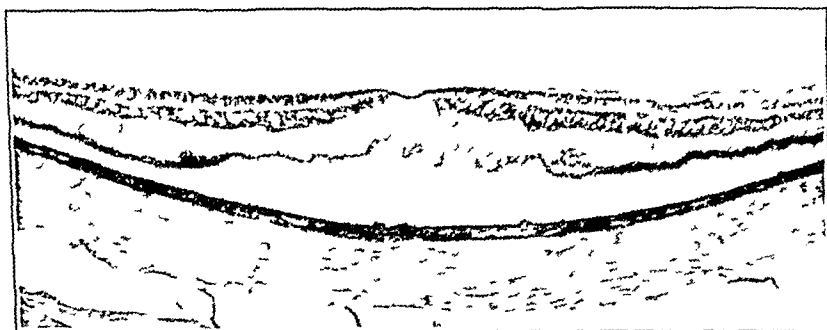


Fig. 8—Photomicrograph showing the marked cystoid condition of the macula, $\times 20$. The separation of the macula may be an artefact. There are two colloid excrescences almost directly behind the fovea.

the anterior part of the ciliary body and root of the iris showed only insignificant infiltration with plasma cells, and the ciliary processes were entirely free from infiltration (fig. 7). Elsewhere the iris was normal except for the presence within it of a few plasma cells. The ciliary body at a distance from the scleral foci showed an occasional small collection of lymphocytes. The retina showed unusually slight peripheral cystoid degeneration. There were many colloid excrescences scattered over the fundus. The optic disk and nerve stem were normal. The vitreous was free from cellular infiltration except in the immediate vicinity of the scleral lesions, and even here the cellular infiltration was slight, as already stated. The anterior chamber contained a delicate serous coagulum. Adherent to the posterior surface of the cornea were a few mononuclear cells, some of them in clumps of as many as six cells. These cells rarely contained pigment. The lens and cornea were normal. The central retinal vessels (examined in serial cross sections), the posterior ciliary vessels and the vessels of the choroid and retina showed slight if any endovasculitis.

In the fundus, at the site of the large pigmented area seen with the ophthalmoscope, the retina was atrophic and fused to the choroid. Neuroglia proliferated from the retina had here invaded the choroid. The histologic picture of this lesion was typical of an old completely healed focus of localized chorioretinitis of tuberculous origin.

In the macular region, involving an area about 6 mm in diameter, the retina showed a cystoid condition, which at the fovea was extremely marked (fig 8). Within the internal nuclear layer and Henley's layer there were numerous distended spaces. The largest were within Henley's layer, and these contained a delicate serous coagulum indicative of edema. There were also spaces within the ganglion cell layer, but these were small. The ganglion cells were apparently normal. There was no cellular infiltration or fibrin. The retina here was slightly separated from the choroid and considerably distorted, probably largely because of the fixation process.

Sections stained for tubercle bacilli, others stained by Verhoeff's modified Gram method, and still others by a variety of bacterial stains all failed to show any microorganisms within the abscesses or elsewhere in the eye.

COMMENT

The clinical appearances of the scleral lesions (figs 1 and 2), their prolonged activity and their association with rheumatoid arthritis leave no doubt that the ocular condition in this case was that designated by van der Hoeve as scleromalacia perforans. The old healed focus of localized chorioretinitis, seen ophthalmoscopically and microscopically, and the old pulmonary lesions, revealed by roentgen examination, were no doubt tuberculous in origin and evidently unrelated to the scleritis; tuberculous scleritis has an entirely different histologic picture. Histologically, the large abscesses within the sclera were the most conspicuous active ocular lesions. They corresponded in position to the nodules observed clinically. Except in the immediate vicinity of the affected portions of the sclera, the eye was almost free from cellular infiltration. From the histologic appearances of the lesions, and especially from the appearance presented by the large sequestrum seen in one nodule, the steps in the evolution of an abscess were readily ascertained to be as follows. An area in the sclera first undergoes simple necrosis. This area soon becomes surrounded by a wall of epithelioid cells. Pus cells then slowly penetrate the wall and surround and infiltrate the necrotic area, which has become more or less disintegrated into eosinophilic granules. Ultimately the sequestrum thus formed becomes completely disintegrated and densely infiltrated with pus cells, which also become necrotic. That the process of abscess formation is extremely slow is evidenced by the fact that almost no pus cells are to be found outside the wall.

The size ultimately attained by an abscess is only slightly larger than the original area of necrosis. Large abscesses do not form from small ones except when the latter break into each other. There is slight if any distention of the sclera by an abscess. The elevation observed clinically is due chiefly to the inflammatory reaction around the abscess.

Only in our case, in Oast's case,⁸ in van der Hoeve's first case and in Wojno's⁹ case were scleral lesions other than the characteristic cavities seen. In these 4 cases nodules were seen in addition and recognized as being the precursors of cavities. It was noted that after a nodule was incised a cavity later existed at the site of the nodule. But in no case have clinical observations been made with sufficient frequency to show how, without operative intervention, transformation of nodules into cavities takes place. The histologic changes in our case, however, clearly indicate that usually the cavities result from abscesses perforating the conjunctiva and discharging their contents externally. They also show that at the sites of the ulcers thus produced the underlying sclera and uvea are largely replaced by a thin layer of newly formed fibrous tissue pervaded in its deepest portions by pigment cells and coated externally by new epithelium. In our case the original conjunctiva, evidently owing to its having been undermined, projected for a considerable distance over the margin of the cavity, and the under surface of the projection was coated with new epithelium.¹⁰ No doubt in some cases the defect in the conjunctiva is so small that the edges later become approximated and the cavity completely covered with conjunctiva. Judging by the illustrations in Kiehle's case,⁶ islands of conjunctiva may remain on a large cavity. At present there is no evidence that the abscesses produce cavities by undergoing organization or absorption.

In accord with the foregoing explanation as to the origin of the cavities in the sclera is the fact that in our case a small fistula was found extending from a sequestrum to the surface. Also in accord with it is the fact that there was no histologic evidence of absorption or organization of the abscesses. The fact that the abscesses showed no tendency to break into the interior of the eye was no doubt due to the great vascularity of the uvea.

It was evident in our case that while the major destruction of the sclera resulted from abscesses, some destruction also resulted from the chronic inflammatory reaction in the adjacent scleral tissue. The cellular infiltration occurred here primarily within the scleral clefts, especially in those containing blood vessels, and was obviously destroying the scleral tissue, which at the same time was being partially replaced by new fibrous tissue.

8 Oast, S. P. Scleromalacia Perforans. Report of a Case, *Arch. Ophth.* **17**: 698 (April) 1937.

9 Wojno, Z. Ein Fall von fortschreitender Erweichung der Lederhaut, *Klin. oczna* **13**: 778, 1935, abstracted, *Zentralbl. f. d. ges. Ophth.* **35**: 692, 1936.

10 Dr. Trygve Gundersen has recently had a patient with scleromalacia perforans who clinically presented exactly this condition. At the margin of a large scleral cavity a spatula was freely passed under the overhanging conjunctiva for a distance of about 1 mm.

In 2 of the previously recorded cases nodules were clinically regarded as containing abscesses. It was stated, however, in the report of van der Hoeve's case that when a nodule was incised it was found not to be an abscess. Probably this statement was based on the fact that fluid pus was not obtained. When one of the nodules was incised in our case, pus was obtained, as ascertained by microscopic examination.

In 2 cases,¹¹ scleral sequestrums were reported to have been seen within scleral cavities. One "sequestrum" was removed and on microscopic examination was found to consist of hyalinized scleral tissue free from cells.^{11a} Judging by our microscopic observations, these "sequestrums," since they were entirely free from infiltration, could not have caused the cavities in which they were seen. Possibly they were simply degenerated scar tissue. As to whether or not a sequestrum can be spontaneously extruded before it has been completely infiltrated with pus cells, there is yet no evidence.

It seems possible that there may be cases of this condition in which some or all of the scleral lesions heal without producing the cavities supposed to be characteristic of the disease. In fact, one of us (F. H. V.) has recently seen what appeared to be undoubtedly such a case.¹² The patient, a woman aged 53, had well marked rheumatoid arthritis. In each eye the bulbar conjunctiva was everywhere intact, and the sclera showed many large bluish areas but no definite cavities. On the outer side of the right eye the bulbar conjunctiva was slightly congested and slightly elevated and showed beneath it a yellowish opaque substance occupying an area about 5 mm. in diameter. Presumably this was an area of scleral necrosis. Information was obtained that one year previously both eyes had been considerably congested for a long period and that there had been "episcleritis," descemetitis and mild iritis. This case strongly indicates that in the ocular disease under consideration, necrotic areas in the sclera, even when of large size, may in some cases be replaced by fibrous tissue without being infiltrated or extruded. Probably the case briefly described by Friedenwald¹³ in 1921 was of this nature.

In the reports of none of the cases in the literature was ocular pain mentioned as a prominent feature, and in some of them it was stated to be absent. In our case it was severe and persistent. It was explained

11 (a) Urrets Zavala, A., Maldonado Allende, I., and Obregon Oliva, R. Escleromalacia observada en el curso de una porfirinuria cronica, *Arch. de oftal. de Buenos Aires* **12** 115, 1937. (b) Soriano, F. J., and Riva, A. Escleromalacia perforante, *ibid.* **12** 139, 1937.

12 The fact that one of us has seen within one year 3 patients affected with scleromalacia perforans suggests that this condition is far more frequent than the scarcity of its reported cases would seem to indicate.

13 Friedenwald, H. Ocular Conditions Associated with Arthritis Deformans. *Am. J. Ophth.* **4** 431, 1921.

by the involvement of the anterior ciliary nerves in the affected tissues and particularly by the marked involvement of a large intraocular nerve loop

The marked cystoid condition of the macula in our case evidently resulted from edema due to the long-continued action of toxins arising from the inflammatory process in the sclera and diffusing through the vitreous. It was the great loss of central vision thereby produced and the continued pain that led us to acquiesce in the desire of the patient for removal of the eye. It seems probable that early curettage of the abscesses would prevent this edema of the macula and other possible intraocular complications.

At first glance, of the two designations for the disease under consideration, scleromalacia perforans, employed by van der Hoeve, and scleritis necroticans, employed by Rochat, the latter designation may seem to be preferable because it denotes the existence of scleritis. Since, however, it indicates that the scleritis causes the necrosis whereas the reverse is actually the case, it is really unsatisfactory. Unfortunately, scleromalacia perforans fails to indicate some of the important features of the disease. Thus it does not indicate that the primary process is necrosis, that it is focal, that there is inflammatory reaction with formation of nodules and that the sclera is excavated without necessarily being actually perforated. Necroscleritis nodosa excavans would describe the disease more completely and accurately. For cases in which there were no cavities, the term excavans could be omitted. Nevertheless, since van der Hoeve's designation has priority, has been generally accepted and is sufficiently distinctive, it seems best to retain it, at least until the exact cause of the disease has been ascertained.

A condition that bears some resemblance to scleromalacia perforans is brawny scleritis. Clinically, the two conditions are alike in their chronicity and in affecting the sclera predominantly. They differ in that brawny scleritis is more diffuse, does not cause definite nodules or cavities in the sclera and is not definitely associated with rheumatoid arthritis. Gilbert¹⁴ in 1914, after microscopically examining an eye affected with brawny scleritis, contended, chiefly because the patient had gout, that this ocular condition was a manifestation of gout. Since, however, he was unable to find urate crystals in the eye, his case affords only suggestive evidence as to the cause of brawny scleritis. One of us has examined microscopically sections of five eyes supposed to have been affected with brawny scleritis.¹⁵ The cases in which these eyes were removed were reported by Oatman, Parsons, Friedenwald, Schlodtman and Verhoeff. In Oatman's case there was no necrosis, the

14 Gilbert W. Zur Ätiologie und pathologischen Anatomie der eitrigen Skleritis, Arch f Augenh 76 111 1914

15 Verhoeff F H Brawny Scleritis Ophthalmoscope 11 2, 1913

infiltration, confined almost exclusively to the episcleral tissue and the uvea, was slight and not distinctive. In the other cases necrosis, although a conspicuous feature, undoubtedly was not the cause but a result of the inflammatory process. In Friedenwald's and Schlodtmann's cases the necrosis closely resembled the caseation of syphilis and tuberculosis. The possibility that brawny scleritis and scleromalacia perforans are due to the same cause, therefore, seems remote.

The problem as to the cause of scleromalacia perforans may now be considered. A possibility to be considered is that the scleral lesions are due to anemic necrosis, that is to say, to vascular obstruction. As to this possibility, we have at present no conclusive evidence. It is true that in our case obliterative endarteritis was marked in the vicinity of the abscesses, but it may well have been the result, not the cause, of the inflammatory process. On a priori grounds, it seems unlikely, although not impossible, that primary obliterative endarteritis, whether or not due to infection, would be confined to the particular sites and tissues in which the lesions of this disease occur.

In the present case, as in 2 previous cases, the bacteriologic investigations, including attempts at artificial cultivation and animal inoculations, yielded negative results. The histologic structure of the ocular lesions definitely excluded tuberculosis and syphilis. The abscesses in an advanced stage were consistent with a fungus infection, but, excluding from consideration vascular obstruction, in their formative stage did not suggest any process known to be infectious in origin.

A question of obvious importance is whether or not the nodules of scleromalacia perforans are similar to the subcutaneous nodules which not infrequently occur in cases of rheumatoid arthritis. In an attempt to answer this question one of us has examined sections of eight such nodules removed from different patients afflicted with rheumatoid arthritis.¹⁶ One of these nodules was removed from the elbow of our patient. One nodule (not the one from our patient) showed in the connective tissue a large sharply defined area of necrosis, about 3 by 1.5 mm. in size. The necrotic area was intensely eosinophilic and almost completely disintegrated into granules of various sizes, from extremely minute to a size larger than a red blood corpuscle. At the periphery, the surrounding connective tissue could be seen undergoing rather abrupt transition into this necrotic material. In the contiguous tissues there were a few collections of coarsely vacuolated cells, apparently macrophages, the origin of which from connective tissue cells seemed obvious. There was no infiltration with lymphocytes or plasma cells. The impression given was that the process was more recent but more severe than in the other nodules. The latter showed

¹⁶ Dr. T. B. Mallory and Dr. W. Bauer, of the Massachusetts General Hospital, gave us the opportunity of examining these sections.

similar large areas of necrosis, but the necrotic tissue was far less granular. Most of the areas were completely walled off by epithelioid cells, just as were the scleral lesions in our case. In only two nodules was there considerable infiltration of the surrounding tissue with lymphocytes and plasma cells. In none of the nodules was there obliterative endarteritis or formation of new vessels around the necrotic areas. Within the necrotic areas irregular reticulated structures could often be seen. Each showed various transitions in staining, from intensely with hematoxylin to intensely with eosin. The impression given was that it was the cell syncytium of the original tissue, which in undergoing necrosis had been to a greater or less extent perfused with chromatin. Most of the necrotic areas showed insignificant infiltration with pus cells, some of them contained no pus cells. However, a large necrotic area in the nodule from our patient contained the largest number of pus cells, about one hundred in a high power field, but even this number was small compared to the number in the scleral lesions.

It is evident from the foregoing observations that the subcutaneous nodules of rheumatoid arthritis and the nodules of scleromalacia perforans in their initial stages are essentially alike. Why, as indicated by our case, the scleral nodules in their later stages tend to become densely infiltrated with pus cells and cause marked chronic inflammatory reaction while the subcutaneous nodules tend to remain relatively free from infiltration can only be conjectured. Possibly it is because of differences in the sensitivity of the affected tissues to the exciting agent. Trauma is undoubtedly an important contributory cause of the subcutaneous, but evidently not of the scleral, nodules.

Since the nodules of scleromalacia perforans and the subcutaneous nodules of rheumatoid arthritis are thus essentially alike, and since this type of scleritis is almost invariably associated with rheumatoid arthritis, it seems reasonable to suppose that the arthritis, the subcutaneous nodules and the scleritis are due to the same cause. It is evident that the initial scleral and the subcutaneous necrotic areas histologically resemble the necrotic areas of gouty lesions and do not resemble any lesions known to contain micro-organisms. In rheumatoid arthritis, areas of necrosis and in gouty arthritis, urate deposits occur in the cartilage of the affected joints. These facts suggest that the lesions of rheumatoid arthritis, including the subcutaneous and scleral nodules, are dependent on the deposition of some chemical substance in the connective tissues concerned. The relative avascularity of these tissues may be an important factor in the process. Chemical investigations of the lesions, by revealing the existence and nature of this chemical substance, may establish the fact that rheumatoid arthritis is due to some metabolic disturbance and even indicate the nature of the disturbance. Although the subcutaneous nodules of rheumatoid

arthritis are easily obtainable, apparently they have never been subjected to such investigation

SUMMARY AND CONCLUSIONS

Fourteen cases of scleromalacia perforans described in the literature are reviewed, and a new typical case is reported, the first in which an eye affected with this disease has been obtained for microscopic examination. The patient was afflicted with rheumatoid arthritis.

From the histologic changes in this case it is evident that the primary ocular lesions each consists of a sharply defined area of necrotic scleral tissue. This area becomes surrounded by a wall of epithelioid cells and slowly infiltrated with pus cells. This process results in the formation of a sequestrum, which becomes completely disintegrated and densely infiltrated with necrotic pus cells.

The characteristic cavities in the sclera are generally due to abscesses so formed, perforating the sclera and discharging their contents externally. At the site of an abscess there remains a thin layer of newly formed fibrous tissue pervaded in its deepest portion by pigment cells. It is probable that there occur similar cases in which, without the formation of cavities, the necrotic tissue is replaced by fibrous tissue.

Through the long-continued action of toxins diffusing through the vitreous from the necrotic foci in the sclera, destructive edema of the macula may result. To avoid this and other possible intraocular complications, curettage of the scleral nodules is suggested.

Severe ocular pain of long duration may be a prominent symptom of the disease.

In the present case attempts to demonstrate micro-organisms in the abscesses by means of special stains, cultures and inoculation of animals failed.

The histologic structure of the lesions excludes tuberculosis and syphilis as possible causes of the condition.

Histologically, in their initial stages the scleral nodules are essentially similar to the subcutaneous nodules of rheumatoid arthritis. Their resemblance to the nodules of gout and their lack of resemblance to any lesions known to be due to direct infection suggest that the lesions of rheumatoid arthritis are dependent on the deposition of some chemical substance in the tissues concerned and hence that this disease is due to some metabolic disturbance.

ABSTRACT OF DISCUSSION

DR. FREDERICK A. KIEHLE, Portland, Ore. A review of this case in connection with the others reported elsewhere calls attention to three points: (1) the comparatively short period of time the condition had existed, (2) the severe pain which the patient suffered and (3) the

minute abscesses which appear in the section. One might infer that the two last-named conditions—the abscesses and the pain—may be related as cause and effect.

The patient in the case which I reported at the 1936 meeting of the Academy of Ophthalmology and Otolaryngology, now bedridden for ten years, is still living, she is blind save for perception of light and is helpless due to arthritis but has a healthy heart and kidneys and good assimilation. The eyes show no further changes. No site of impending or of probable future perforation can be identified. As these cases, however, are often of many years' duration, the inference is that perforation is a late manifestation of the process.

In a recent personal communication Professor van der Hoeve confessed that he is unable to throw any new light on the subject, while in a communication from Sir Stewart Duke-Elder a recent case which he has observed is described. I quote from his letter:

"The patient with scleromalacia whom I saw in March was a woman of 56 who had been suffering from polyarthritic rheumatism for about thirty years and is now a complete cripple with most of her joints ankylosed. The right eye showed two large areas over which the sclera had completely disappeared, exposing the uvea and in the left eye all around the ciliary region a similar condition existed, there being only one or two bands of sclera remaining. In the left eye there was a circular ulcer all around the cornea of an atrophic indolent type. Nothing in the picture suggested inflammation. I am proposing to do a mucous membrane graft from the lip to cover the exposed areas sometime in the near future."

It is only by calling attention to the existence of this disease, now attaining recognition as an entity, that these sporadic cases will be garnered into some sort of a whole from which definite conclusions can be drawn.

The evident association between scleromalacia and polyarthritis is a marked feature of the condition and one which continues to await explanation.

DR SAMUEL P. OAST, New York. In the April 1937 issue of the ARCHIVES (page 698) I reported a case in which the ocular picture was strikingly similar to that in the case so painstakingly investigated and so ably presented by Dr Verhoeff and Dr King but in which there was no history of any condition resembling arthritic involvement, my patient was a robust appearing man of 76. The ocular condition was unilateral, and the patient did not have arthritis.

I have two slides made from drawings which I believe give an accurate idea of the condition as it was observed.

The first slide shows one of the characteristic nodules at about the height of its active stage before discharging its necrotic center. This drawing was made late in the course of the disease, so that the edges of the older healed foci may be seen to either side.

The second slide shows the sites of the nodules after healing had taken place, the blue uvea being evident through the residual scleral and cicatricial tissue. The eye remains in this state today, with the exception that all evidence of redness has disappeared.

In the 14 cases dealt with here there are two outstanding features, the most striking and common of which is the scleral thinning. The other is the localized inflammatory nodule. This was observed in but 4 of the 14 patients. Now whether or not some of the other 10 patients came under observation after the nodules had been present but had disappeared is a thought which has to be considered, but surely Dr Kiehle's patient, who was under more or less continuous observation by him and other prominent ophthalmologists for over a period of years, did not have any localized inflammatory lesions. The fact stands out, therefore, that in some of these 14 patients inflammation was either absent or was not a prominent feature.

The different behavior of the condition in these two groups suggests to me that more than one etiologic factor may be responsible and that one may be classifying these possibly different conditions as one disease entity solely because of their similar end effects, as evidenced by absorption of the sclera.

I am therefore in accord with Dr Verhoeff and Dr King in objecting to the term "scleromalacia," particularly as applied to the type of lesion depicted in their report, for the principal reason that the term "scleromalacia" does not carry with it any thought of inflammation in the customary acceptance of that term.

I feel that I should not let this occasion pass without mentioning a circumstance in the treatment of my patient, the evaluation of which I shall leave to your judgment.

The usual symptomatic local measures had been followed for four months or more, and nothing that was done seemingly had the slightest effect on the unrelenting progress of the disease. I had all but despaired of saving the eye, when, skeptically, I began treatment with ultraviolet rays. Almost immediately the pain became less (pain had been an annoying feature from the onset), the old lesions began to disappear and no new nodules formed. I have since kept the patient under observation for nearly two years, and the eye has remained quiet clinically ever since.

I should like to ask Dr Verhoeff whether, in view of the roentgen evidence of a healed, possible tuberculous lesion of the chest, the microscopic evidence of a tuberculous choroidal lesion and the finding in the scleral nodules of a broken-down center with a surrounding zone of epithelioid cells and some Langhans' type of giant cells, he has dismissed tuberculosis categorically from further consideration as a causative agent in this particular type of lesion.

DR ARTHUR BEDELL, Albany, N. Y. The presentation of a case of scleromalacia is of considerable interest because of the rarity of the disease and the thoroughness with which the authors have reported the case.

About two years ago a 70 year old woman was sent to me by Dr Marshall of Rutland, Vt, with a history that her right eye had been red for six months, with what appeared to be scleritis and several marginal ulcers. The latter responded to treatment with trichloroacetic acid. Three weeks ago the scleral area became more purple, the overlying conjunctiva disappeared, and an ulcer developed.

The patient was otherwise in good health, never having suffered from any serious illness or local ocular disease.

When I examined her, vision in the right eye was 10/200. There was moderate conjunctival congestion with a few flat discrete follicles. The pupil was 3 mm in diameter and regular, with a sluggish reaction to light. There were several small, deep corneal ulcers, 0.5 mm in diameter, near the upper limbus, and the upper one fifth of the cornea was rough, gray and infiltrated. Four millimeters from the superior limbus was a large, oval, bluish black crescent, 10 by 5 mm, with depressed, rounded borders. The floor of the ulcer, a dark, almost black, protruding soft mass, like a choroidal prolapse, was divided into two parts by a gray band 2 mm wide. The nasal half was a little the larger.

The clinical course was protracted. At times the eye became almost white, the cornea became smooth, and the scleral ulcer lost some of its dark, bulging protrusions, but never flattened. The conjunctival overgrowth and the scar tissues reduced the surface loss, but the corneal infiltration became much greater and the vision was reduced to perception of light.

The eye was such a constant source of irritation that it was enucleated eight months later.

The sclera was thin, beneath an overhanging conjunctival shelf. The infiltration of the cornea was so great that the only portion which remained clear was a small central opening.

The left eye was normal.

General examination gave negative results. Smears and cultures gave no clue to the cause.

The patient had no evidence of rheumatoid arthritis.

(Several lantern slides of direct color photographs of the fundus were used to illustrate the discussion.)

DR. TRYGVE GUNDERSEN, Boston. I have some colored photographs of the eyes of a patient who has been under my care for the past two and a half years. The patient was first seen in November 1935 at a convalescent home, where she was being treated for severe rheumatoid arthritis. She complained of sore eyes. A diagnosis of chronic conjunctivitis was made.

She was seen again about a month later, when acute glaucoma was present in each eye, and the tension was 50 mm (Schiotz). The pupils were dilated. The anterior chambers were deep. There were no aqueous cells or keratitis punctata. The patient was immediately transferred to the Massachusetts Eye and Ear Infirmary. Paracentesis was performed on the right eye and iridectomy on the left eye. A complete separation of the choroid occurred in the left eye, which lasted for the ensuing three months. For three or four months the right eye continued to show a tension of between 40 and 50 mm (Schiotz), which gradually subsided.

Scleritis developed in both eyes about four months after the onset of the glaucoma. Although the patient was seen at frequent intervals in the convalescent home, at no time could it be said that there was a definite discharge from any of the scleritic nodules. The undermining of the conjunctiva was such that a Bowman probe could be

passed under the overhanging edge of conjunctiva for a distance of 2 mm. At no time was there any ectasia of the ciliary body or uvea. The intraocular tension is now 10 mm (Schiotz) in each eye.

DR LLOYD MILLS, Los Angeles. I wish to take exception to a statement made in most of the texts, that no local treatment is of avail in the various forms of scleritis. About twenty-four years ago, when working in Fuchs' clinic, I was struck by the constrictive effect of a 20 per cent solution of zinc sulfate when applied to lesions about the limbus. This application often led to the healing of phlyctenules. It was but a step to apply this, with considerable success, in cases of episcleritis, scleritis and brawny tenonitis, always in connection, of course, with the elimination of infective or other allergens wherever possible.

The element of chronicity appears to be the element of importance in these cases. The examination of sections from such eyes show them packed with lymphocytes, the tissue spaces being filled with edema, and it is only logical that continued increased tissue pressure of this sort may provide the basis for necrosis.

It has seemed to me, therefore, that if the time of healing could be hurried, there would be far less chance for these serious sequels. For over twenty years I have been using a 20 per cent solution of zinc sulfate in all cases of scleritis, regardless of the clinical background or severity of the condition. It definitely has hastened the arrest of the lesions and has lessened or prevented scarring.

The application is made as follows. A 4 per cent solution of cocaine hydrochloride and epinephrine in a dilution of 1:1,000 are instilled four times. In the first treatment there is little or no blanching. Then a swab, wet, but not dipping, is applied to the margin of the lesion and kept steadily there for twenty seconds. The entire circumference of the lesion and its center are similarly covered, with special attention to the blood vessels. The eye is finally washed with boric acid, and cold compresses are applied until the patient is comfortable. Treatment is repeated daily. Usually on the third or fourth day of treatment the lesions begin to blanch, become defined and gradually flatten, with scars, if of long duration, and without scars, if acute.

DR FREDERICK H. VERHOEFF, Boston. Dr. Kiehle brought up the question of the short duration of the condition in the case reported. Since in previous cases there is no evidence as to how long a time is required for a nodule to come and go, I do not think that his point is well taken.

As to the question of pain, according to the reports it is seldom mentioned, and I think that was an unusual feature in this case. It was explained well, however, by the fact that we found ciliary nerves much infiltrated in the region of the lesions.

Dr. Oast suggests that in some cases this scleral process may occur without its having been preceded by a nodule. I called attention to the possibility that the condition in all cases may not be like that observed by us, in fact, I recently saw a patient in whom the sclera showed these bluish areas of discoloration, with the conjunctiva intact over them and no depression whatever. I think that such instances might often be overlooked.

Dr Oast suggests that the condition is not an entity, it may be due to various causes. This seems to me unlikely, because the ocular condition is so much alike in all the cases and in the great majority is associated with rheumatoid arthritis.

Dr Oast also brings up the question of tuberculosis because I mentioned the occurrence of giant cells, epithelioid cells and necrosis. But these are found in a good many conditions. It is not their occurrence but their arrangement that is of chief diagnostic significance. No general pathologist, I am sure, would think for a moment that the lesions in this case were tuberculous. Moreover, if they were tuberculous, which I cannot conceive of, the material in the abscesses would have been just the sort of material that would infect a guinea pig. We injected the material into a guinea pig and did not produce tuberculosis. We also injected the material beneath the conjunctiva of a rabbit with negative results.

The condition in Dr Bedell's case may or may not be scleromalacia perforans. I think that it perfectly well could be. The corneal involvement is unusual, but it seems to me that it should be expected, judging from the pathologic changes, that the cornea should be involved in some cases in the way he describes.

Dr Gundersen's case is the only one in which glaucoma was noted and his case was interesting to me because it confirmed my observations regarding the formation of the excavations.

USE OF SORBITOL IN GLAUCOMA

JOHN BELLOWS, M D

IRVING PUNTENNEY, M D

AND

JACK COWEN, M D

CHICAGO

The lowering of the intraocular pressure by osmotic changes produced by the intravenous injection of hypertonic solutions is well known. Cantonnet¹ demonstrated that the ocular volume diminished after the intravenous injection of hypertonic salt solution. Hertel² showed in animals that the decreased tension produced by injections of hypertonic saline solution was accompanied by a decreased water content of the eyes. The clinical usefulness of the hydrophilic character of hypertonic solutions has found application in various fields of therapy, including that of ophthalmology. However, many ophthalmologists have not taken full advantage of osmotic therapy.

The possession of an additional means of lowering intraocular tension when methods ordinarily used either are contraindicated or are inadequate is a valuable asset. This is particularly apparent when intraocular tension cannot be controlled immediately after an operative procedure on the eye, in this case inflammation and tenderness are so great that neither additional operative intervention nor local ocular medication is tolerated. The further desirability of the use of a hypertonic solution intravenously can be demonstrated by the dangers and shortcomings inherent in the accepted surgical procedures for glaucoma.

Paracentesis, the simplest method for lowering intraocular tension, is fraught with the dangers of prolapse of the iris and injury to the lens and is followed, as Kronfeld³ has shown, by a secondary rise in tension to a point higher than the initial tension.

Posterior sclerotomy, in addition to the dangers listed under paracentesis, adds the hazards of injury to the retina and choroid with possible detachment of the retina, injury to the uvea with ensuing sympathetic ophthalmia, intraocular hemorrhage following the chance

From the Department of Ophthalmology, Northwestern University, and the Illinois Eye and Ear Infirmary

1 Cantonnet, A. Arch d'opht 24 193, 1904

2 Hertel, E. Arch f Ophth 88 197, 1914

3 Kronfeld, P. C. Tr Am Ophth Soc 22 115, 1928

perforation of a retinal or large choroidal vessel and the introduction of infection

The injection of procaine hydrochloride and epinephrine hydrochloride deep into the orbit adds to the danger of retrobulbar hemorrhage and proptosis the possibility of injecting material intravenously with serious consequences. The tension is often not affected by this means and may rise even higher.

Medical control of ocular hypertension also presents difficulties, for which intravenous hypertonic therapy is a solution. Miotics may be undesirable because of the possible formation of synechiae in those cases in which an exudative process is present. The surface application of epinephrine hydrochloride has its limitations. The patient must be watched for systemic reactions, since hypertensive persons may show an alarming rise in arterial tension, and, as previously mentioned, a paradoxical rise in ocular tension may occur. Purgatives and agents to produce sweating may be conceivably used, but both have undesirable side-effects in older and debilitated persons. In addition Duke-Elder⁴ has shown in a comparison of the effects of hypertonic solution given orally and intravenously that the osmotic effect produced by administration by the intravenous route is far greater than that produced by administration by the oral route. Osmotic therapy may be of great value as an emergency measure at a time when the services of an ophthalmologist are not available.

Hypertonic solutions in the control of ocular hypertension finally have their greatest usefulness in those cases in which the tension remains high in spite of the administration of the strongest miotics. Such therapy is a boon to the ophthalmologist who is faced with the necessity of operating on a stony-hard glaucomatous eye with all the attendant hazards, especially the hazard of intraocular hemorrhage.

Osmotic therapy, however, also has its disadvantages. There is danger of overburdening the heart with the increased blood volume, and injury to the median nerve may result. Specific objections can be raised as to the substances used for osmotic therapy. Sodium chloride is limited in its use because of its great diffusibility. The salt enters into the tissue spaces of the body, causing a general water-logged condition. This is most feared in those patients with cardiac and renal damage in whom chlorides are already retained and an edematous state is already present. In a like manner the salt enters the anterior chamber of the eye and causes imbibition of water with a secondary rise in tension to a point higher than the original tension. Furthermore, the injection of sodium chloride is in itself painful, and it produces a severe slough if extravasated into the tissue. However, it has the

⁴ Duke-Elder, W. S. *Brit J Ophth* **10** 1, 1926

advantage of producing the greatest osmotic effect of any of the substances compared for the quantity used. It owes its great osmotic action to the small molecular size and marked ionizing power.

Dextrose, like sodium chloride, because it is readily diffused into the tissues produces a secondary rise in tension after an initial drop. This diffusibility of dextrose and chloride into the anterior chamber has been demonstrated by Duke-Elder and others, who found in the aqueous humor increased quantities approximating those in the blood stream after intravenous injections of these substances. A further objection to dextrose is that its use is limited to nondiabetic patients.

Sucrose, a nondiffusible disaccharide, was introduced to supplant the difficulties found with the use of sodium chloride and dextrose. Because of its large molecular size, greater quantities of this substance are necessary than of a monosaccharide. Experiments on animals show that this sugar does not enter the ocular tissues.

TABLE 1—*Data on Sugar Content of Aqueous Humor*

	Before Injections, Aqueous Humor, Mg per 100 Cc of Sugar	After Injection of 30 Cc of 50% Sucrose Intravenously, Aqueous Humor, Mg per 100 Cc of Sugar
Dog 1	79	80
Dog 2	81	80

EXPERIMENTAL WORK ON SUGAR CONTENT OF AQUEOUS

The aqueous humor of the right eyes of two dogs weighing from 35 to 40 pounds (15.9 to 18.1 Kg.) was aspirated, and the amount of sugar was determined. Then 30 cc. of a 50 per cent solution of sucrose was injected intravenously. After one hour the aqueous humor of the left eye was aspirated. After hydrolysis the total amount of sugar was determined. Table 1 shows that there was no increase in the reducing sugar. This points to the nondiffusible character of sucrose.

For these reasons sucrose seemed an ideal choice for osmotic therapy, since it accomplished its purpose without a secondary undesirable effect. Masserman⁵ had already found that sucrose did not enter the cerebrospinal fluid and therefore did not occasion the secondary rise in intracranial pressure found with dextrose and saline solution. Dyar and Matthew⁶ obtained favorable results with sucrose in a series of glaucomatous patients. Sucrose had been used by us prior to this report with good results but had been abandoned because of the findings

5 Masserman, J. H. *Bull. Johns Hopkins Hosp.* **57**: 12, 1935.

6 Dyar, E. W., and Matthew, W. B. *Use of Sucrose Preparatory to Surgical Treatment of Glaucoma. Preliminary Report, Arch. Ophth.* **18**: 57 (July) 1937.

of H A Lindberg,⁷ of Northwestern University, who presented evidence of severe renal damage in dogs after its use. This damage consisted of swelling of the collecting tubule to apparent closure and of inflammation of the glomeruli, with later hyalinization in some, indicating permanent damage. After the intravenous injection of sucrose in human beings Lindberg found occasional blood cells and casts in the urine.

Because of the objectional features of the aforementioned agents which have been used up to now for osmotic therapy, it would be of great value if a substance could be found which would be relatively nondiffusible, nontoxic, effective in moderate quantities and usable in persons with diabetes. Of all the agents which are readily available, sorbitol⁸ most nearly satisfies these requirements.

Sorbitol is chemically a complex alcohol of approximately the same molecular size as dextrose. West and Carr,⁹ who have made extensive studies of its toxicity, find it less toxic than sucrose, while Lindberg has found that the kidneys are not altered in the slightest degree by it. Although data regarding the metabolism of sorbitol are yet incomplete, the evidence points to its inertness, and therefore it may be safely used in patients with diabetes. In Germany it is used as a sweetening agent by diabetic persons. For these reasons it occurred to us that this compound would be better than any substance heretofore used for osmotic therapy in glaucoma.

EXPERIMENTAL WORK WITH SORBITOL

In order to determine the amount of diffusibility into the anterior chamber of the eye, experiments were carried out in which sorbitol was injected intravenously into dogs in amounts slightly greater relatively to the amount used in human beings. It was found that only small amounts (from 5 to 10 mg per hundred cubic centimeters) entered the anterior chamber of the eye. (Further work along this line is now being carried out and will be reported later.) Therefore it appears that sorbitol is far less diffusible than dextrose and sodium chloride, and from this it follows that it would not produce as marked a secondary rise in tension as the latter substances.

The clinical investigation was undertaken by us to determine its value in patients with increased intraocular tension (including those with primary and with secondary glaucoma). One hundred cubic centimeters of the 50 per cent solution was administered intravenously. All liquid foods were restricted on the day of this study. Small sips of water were permitted to ease the thirst. Part of the series received no miotics from twelve hours prior to the injection until the end of the period of observation. The tension of the patients was determined at intervals of two hours day and night by the same observer and with the same

7 Lindberg, H A. Personal communications to the authors.

8 Abbott Laboratories supplied the material.

9 West, E S, and Carr, C J, cited by Potter, R D. *Science (supp)* **87** 8 (April 29) 1938.

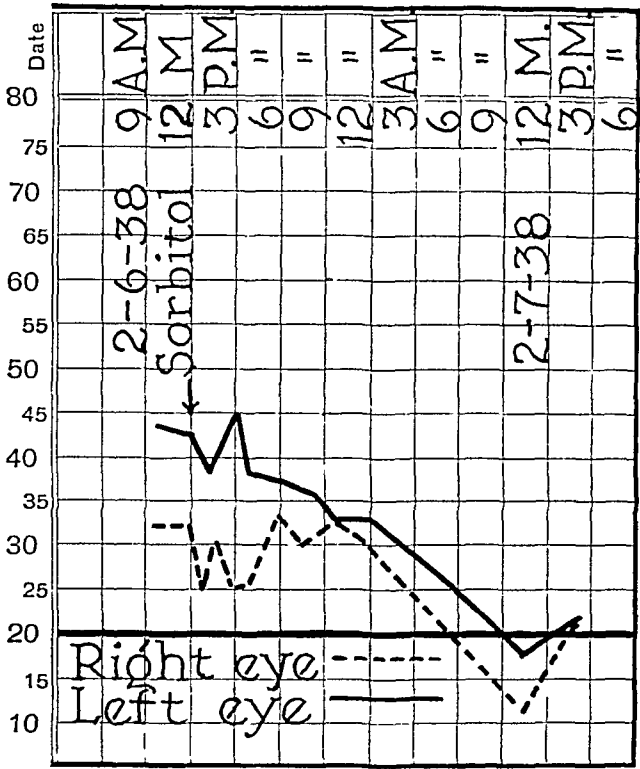


Chart 1—Tonometric chart of P S

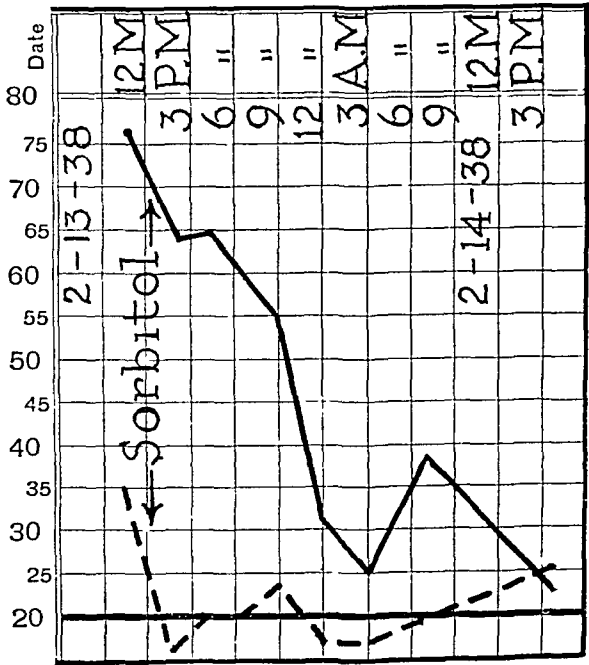


Chart 2—Tonometric chart of F L

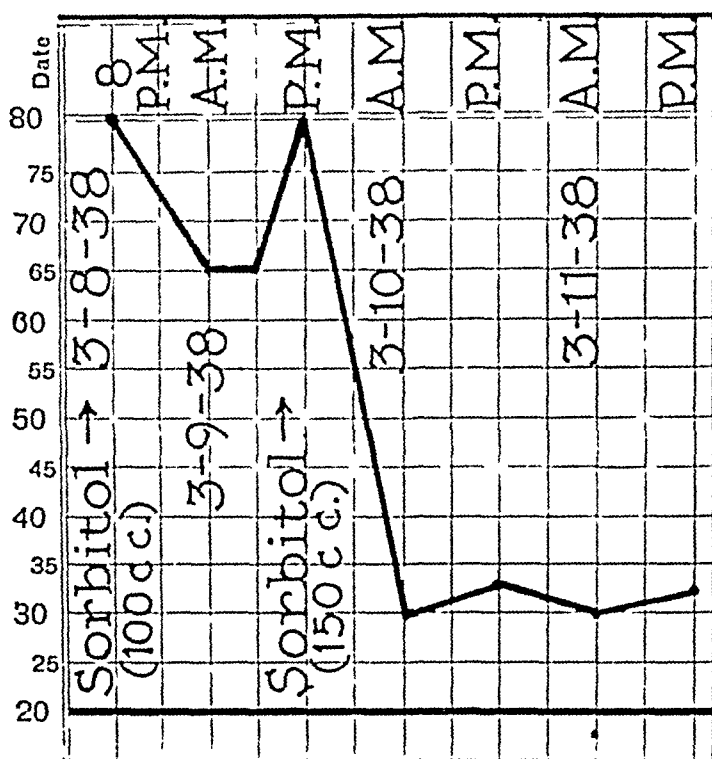


Chart 3—Tonometric chart of M H

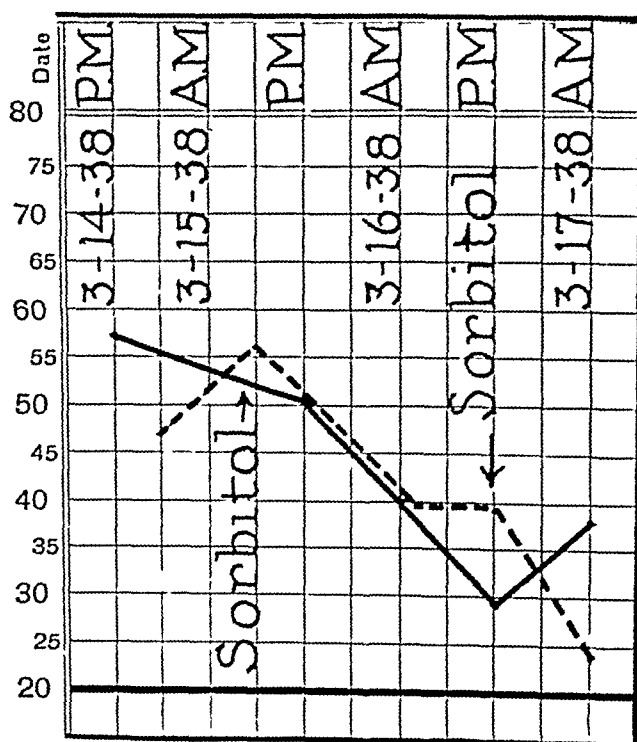


Chart 4—Tonometric chart of F M

TABLE 2—*Effect of the Intravenous Injection of Sorbitol on Intraocular Tension*

Patient	Diagnosis	Intraocular Tension, Mg of Hg (Schiotz)															
		Before Administration of Sorbitol		After Administration of Sorbitol, Hours													
				2	4	6	8	10	12	14	16	18	20	22	24	26	48
J K	Congestive glaucoma	40	38	38	33	33	31	34	34					20		35	
P S	Right eye, chronic glaucoma Left eye, hemorrhagic glaucoma	44 32	40 30	38 26	38 32	38 30	36 30	31 34	34 32			24 20		17 12		22 22	(chart 1)
F L	Glaucoma simplex Right eye Left eye	37 77	17 65	20 63	20 38	23 53	23 53	17 32	17 32		17 25	20 38				23 25	(chart 2)
N P	Glaucoma simplex	44	38	38	37	23		25			25	38				41	
T B	Chronic glaucoma	44	32	35	37	38			25			35					
O N	Bilateral glaucoma simplex Right eye Left eye	44 50	44 52	44 48	32 37	44 48	44 48	43 48		35 48		44 48		32 30			
C G	Left eye, glaucoma capsulare Right eye Left eye	17 32	20 28	20 28	16 33	16 32	18 30	18 30	16 31			12 24		12 17	20 30		
M H	Intumescent cataract, secondary glaucoma	80 83	(Sorbitol repeated)														(chart 3) 32 0 30
G J	Glaucoma simplex (diabetic)	51 5						38							38	32 5	
T M	Glaucoma simplex (chart 4) Right eye Left eye	55 0 51 5	50 5 51 5			51 5 51 5						41 5 41 5		(Sorbitol repeated)		23 5 38 0	
H S	Glaucoma after cataract	38	27	25				23	41					38	(Sorbitol repeated)		17
M P	Glaucoma simplex (normal) Right eye Left eye	64 20						44 12			55 12	(Sorbitol repeated)			41 13		23

Schiotz tonometer, with the exception of a few on whom this procedure could not be carried out

RESULTS

In all cases in which sorbitol was administered, lowering of the intraocular tension was observed (table 2 and charts 1-4). The greatest diastolic drop was obtained where it was most desirable, namely, in patients with high intraocular tension who do not respond to the administration of miotics alone. Although a gradual decrease in tension was noted as early as two hours after the injection, in most cases the maximal effect was reached from twelve to twenty-four hours after injection. Then the tension gradually rose and reached a level approaching the original value, but in no instance did the tension rise appreciably above that point. In several instances in which operative procedures were not undertaken the intraocular tension observed for a number of days remained at a fairly normal level. In some persons repeated injections of sorbitol achieved a pronounced effect when a single injection had produced little change.

SUMMARY AND CONCLUSIONS

Sorbitol was investigated with a view to replacement of less desirable agents in osmotic therapy. Laboratory experiments on dogs show sorbitol to be slightly diffusible into the anterior chamber as compared to dextrose and sodium chloride. It was found to be an effective agent in reducing abnormally high intraocular tension when injected intravenously into human beings. Because of its slight diffusibility, a secondary rise in tension above that of the initial value was not observed.

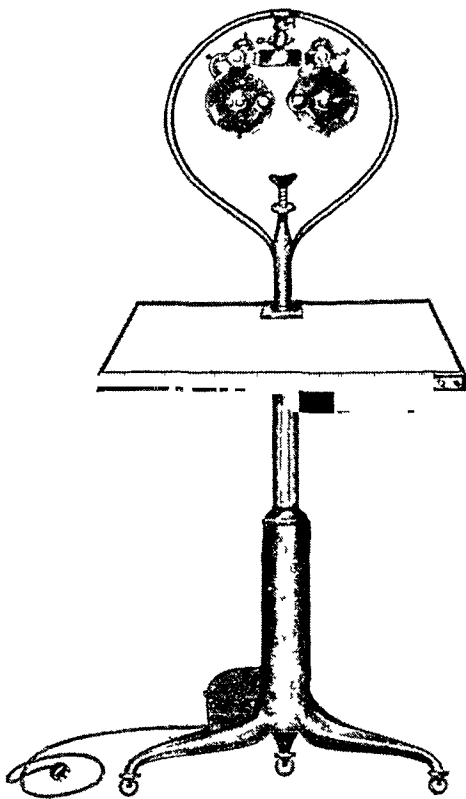
In conclusion, we recapitulate that 100 cc of a 50 per cent solution of sorbitol administered intravenously and repeated in twenty-four hours, if necessary, will nearly always reduce intraocular tension when it is greatly increased and is not controlled by the use of miotics alone. It thus relieves the attending pain and prepares the eye for operation.

Clinical Notes

A NEW REFRACTOR SUSPENSION

S MAISLER, M D , SAN FRANCISCO

The instrument here illustrated affords a new type of refractor suspension. It embodies new streamline designing, unity, ease of operation, facility of aim and chin rest for the patient and a writing table for the examiner.



A new refractor suspension

The black crackle-finished tubular suspension can be utilized for any type refractor, it is mounted on an electric lift table, which requires only the slightest pressure on the button switch for quick, smooth and easy vertical adjustments. Gross adjustments for the refractor in the lateral and anteroposterior directions are adequately controlled by the movement of the entire table, which is mounted on ball-bearing casters. The finer adjustments in the anteroposterior position of the refractor are made with an adjusting screw of the refractor itself.

The vitrolite-covered table provides spacious writing facilities for the examiner and affords a resting place for the patient's elbows and forearms. The chin rest also enhances the comfort of the patient. A retinoscope and ophthalmoscope with cord reels and the necessary rheostat are easily added accessories.

The table will also be available in offset pedestal design, to be used in conjunction with an ophthalmic chair.

This new suspension unit was developed with the assistance of Mr. A. Parsons, of Trainer and Parsons, San Francisco.

350 Post Street

A NEW CAPSULE-GRASPING FORCEPS

M. M. CULLOM, M.D., NASHVILLE, TENN.

A new capsule-grasping forceps for use in the intracapsular extraction of cataracts is here described. It is a double-headed forceps, designed to grasp the capsule at two points. The advantages are obvious.



Fig 1—A new capsule-grasping forceps

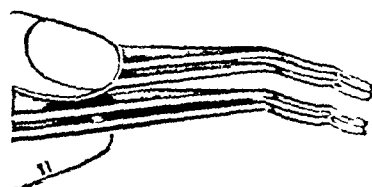


Fig 2—Head of the forceps, showing the prongs closed

Surgeons who use a capsule-grasping forceps say that in about 50 per cent of the cases the forceps tears out, necessitating extracapsular extraction. When the capsule is grasped at two separate points the chances of this occurrence are greatly lessened and there is much better leverage, so that it is easier to dislocate the lens and bring it out intact in the grasp of the forceps.

The forceps should, of course, be closed when it is inserted into the eye, and then be allowed to spread. The capsule can be grasped and the necessary rocking movements made to dislocate the lens. There is a natural spread of 4 mm between each pair of blades.

Ophthalmologic Review

EDITED BY DR FRANCIS HEED ADLER

ETIOLOGY OF RETINAL SEPARATION CONSIDERED FROM THE STANDPOINT OF SURGICAL CORRECTION

EDMUND B SPAETH, MD

PHILADELPHIA

In this review regarding the surgical correction of retinal separation certain factors are considered which must be of etiologic importance, judging from the successes and failures resulting from present day operations. They are presented without any attempt at correlation save their apparent pertinence. It is even impossible to estimate the relative importance of some of these various factors, one as compared with the other. The only certainty is that all are relevant.

HISTORY

The modern treatment of separation of the retina is perhaps the outstanding single advance in ophthalmology within the present generation. Even this period, relatively short as it is, is definitely narrowed, in that the earliest article which initiated the recent research (and the tremendous strides which have been achieved) is that by Verhoeff¹ in 1917. From this time until Larsson's² work in 1928 and in 1930, Vogt's³ in 1929, Sourdille's⁴ in 1929 and Gonnin's⁵ in 1930 the literature was rather sterile of satisfactory operative procedures. To be true, many discussions of the etiology, theories as to formation and considerations of the pathologic picture were presented in this interval, but for some reason the general ophthalmologic world seemed dormant to the marvels which were occurring, at least unfolding, before them. Even early articles connected with the surgical treatment passed unnoticed until attention was called to them by the tremendous amount of literature which suddenly appeared after 1930. Some of these early articles

From the Peter Clinic, the Graduate School of Medicine, the University of Pennsylvania

1 Verhoeff, F. H. *Ophth Rec* **26** 10, 1917

2 Larsson, S. *Acta ophth* **3** 319, 1926, **6** 344, 1928, **8** 172, 1930

3 Vogt, A. *Klin Monatsbl f Augenh* **82** 619, 1929

4 Sourdille, G. *Prat méd franç* **8** 122, 1929

5 Gonnin, J. *Ann d'ocul* **167** 361, 1930

included that already mentioned by Verhoeff,¹ Gonn's⁶ work of 1919 and of 1921, Lenz's⁷ of 1922, Sourdille's⁸ of 1923 and Jocqs's⁹ of 1920

It is interesting that the modern basis for the surgical treatment of retinal detachment had been considered long before, Anderson¹⁰ in his excellent bibliography on the subject called attention to the "formation of adhesions" with such treatment by citing an article by James Ware¹¹ on electrolysis and articles by Clavelier and Maravel¹² and Terson,¹³ the provision for permanent drainage was emphasized as early as 1872 by de Wecker¹⁴

Vogt,¹⁵ with the help of his assistant Latte, was able to present the following definite dates

Who was the first to see a retinal tear? Coccius,^[16] in 1893 The next was A von Graefe,^[17] who, however, regarded the tear as a part of the healing process, until Hansen^[18] in 1871 and Schweigger^[19] in 1873 advanced the opposite view

Who recognized for the first time that the tear was the cause for the detachment? De Wecker^[20] of Paris, in 1870 In his book he described three types of retinal detachment "Décollement par distension, par attraction et par soulèvement" (detachment by distention, by attraction and by swelling) In the first two, according to him (1870) the retinal tear is the direct cause for the detachment In 1882, Leber^[21] popularized this mode of development According to him (1882), every acute detachment which can be recognized with the ophthalmoscope is caused by a retinal tear

6 Gonn, J Cor-Bl f Schweiz Aerzte **49** 1675, 1919, Ann d'ocul **158** 175, 1921

7 Lenz Augenartzliche Operationslehre, in von Graefe, A, and Saemisch, E T Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1922, p 1289

8 Sourdille, G Arch d'opht **40** 419, 1923

9 Jocqs, R Clin opht **24** 357, 1920

10 Anderson, J R Detachment of the Retina A Contribution to the Study of Its Causation and Treatment, New York, The Macmillan Company, 1931

11 Ware, J Chirurg **2** 238, 1805

12 Clavelier and Maravel Ann d'ocul **115** 37, 1895

13 Terson, A Ann d'ocul **114** 22, 1895

14 de Wecker, L Ann d'ocul **67** 137, 1872

15 Vogt, A Operative Treatment of Detachment of the Retina Abstract of the Official Review Presented Before the International Ophthalmological Congress, Madrid, Spain, April 18, 1933, Arch Ophth **10** 293 (Sept) 1933

16 Coccus, A Ueber die Anwendung des Augenspiegels, Leipzig, I Muller, 1853, p 131

17 von Graefe, A (a) Arch f Ophth (pt 1) **1** 358, 1854, (b) (pt 2) **4** 235, 1858

18 Hansen, E Hospitalstid **14** 1, 1871

19 Schweigger, C Handbuch der speciellen Augenheilkunde, ed 2, Berlin, A Hirschwald, 1873, p 456

20 de Wecker, L, and de Jaeger, E Traite des maladies du fond de l'oeil et atlas d'ophtalmoscopie, Paris, A Delahaye, 1870, p 151

21 Leber T Ber u d Versamml d ophth Gesellsch **14** 18 and 165, 1882, Klin Monatsbl f Augenh **20** 18, 1882

Who introduced ignipuncture in the treatment of retinal detachment? Martin,^[22] de Wecker^[23] and de Luca,^[24] in 1881 After that time ignipuncture became generally accepted, particularly by French and English oculists, and was frequently employed

Who was the first to select the region of the retinal tear and the place of the beginning of the detachment as the site for operation? Schoeler^[25] of Berlin, in 1889 He injected tincture of iodine between the detached vitreous and the retina, in the neighborhood of the tear, and in the place of the beginning of the detachment, in other words, in the preretinal space In his book he published an illustration from a case in which the retina became reattached and the tear closed

Who was the first to use ignipuncture at the site of the tear? Deutschmann, our senior master, in a case in 1896, which, however, he seems to have forgotten The case was fully reported^[26] in 1899

Who was the first to use ignipuncture systematically for a retinal tear and with success? Xavier Galezowski^[27] of Paris, in 1902 and 1903, a few years before his death He not only performed ignipuncture on the tear, but aspirated the retroretinal fluid before this procedure He aspirated the fluid just as Gonin^[28] did later, so that the retina could apply itself before the cauterization took place "Je pratique l'aspiration du liquide et la galvanocauterisation de la partie déchirée de la rétine et de la choroïde J'ai obtenu ainsi de bons résultats" I practice aspiration of the fluid and galvanocauterization of the ruptured portion of the retina and of the choroid In this manner I have obtained good results^[27b]

Larsson in 1932,²⁹ in discussing electroendothermy in the treatment of detachment of the retina, expressed himself so well that he is quoted here, in part

The hopelessness that used to characterize the ophthalmologist's attitude toward the question of treatment for retinal detachment has to some extent begun to lessen Prospects have become brighter with the operative method of treatment suggested by Gonin There is no doubt that ophthalmologists stand in great debt to Gonin for having so successfully and energetically taken up this question A task of this nature becomes the more exacting as it involves, and will always involve, many great disappointments for the operator Thanks to Gonin's work, the operative treatment for retinal detachment has become one of the vital issues in ophthalmology

Gonin's view of the pathogenesis of and operative treatment for detachment of the retina are already well known to every ophthalmologist He maintains consistently de Wecker's old theory [published in 1870³⁰] of the importance of a rupture (hole) in the retina, as the primary factor in the origin of the detachment

22 Martin, G Tr Internat M Cong 3 110, 1881

23 de Wecker, L Ann d'ocul 87 41, 1882

24 de Luca, D Ann di ottal 12 330, 1881

25 Schoeler, H L Zur operativen Behandlung und Heilung der Netzhautablosung, Berlin, Peters Verlag, 1889, pp 28, 41, 86, 90 and 93, plate 1

26 Deutschmann, R Beitr z Augenh 40 46, 1899

27 Galezowski, X (a) Cong de chir, Paris, 1902, p 417, (b) Bull et mem Soc franç d'opht 20 214, 1903

28 Gonin, J Rev gen d'opht 37 337, 1923

29 Larsson, S Electro-Endothermy in Detachment of the Retina, Arch Ophth 7 661 (May) 1932

30 de Wecker and de Jaeger,²⁰ p 153

Gonin expressed the belief that if the hole can be made to close, the subretinal contents are absorbed and the detached retina resumes its normal position. He tried to achieve this closure of the hole by direct cauterization of its margins. Gonin³¹ stated

These unexpected successes were first referred to before the Swiss oculists at Bâle in 1919, with, of course, great reserve, then in some Swiss or French reviews, such as *Annales d'oculistique*, 1921, and *Revue générale d'ophtalmologie*, 1923, later, with a short description of some typical cases presented before the French, Swiss and German Societies in Brussels, Zurich and Heidelberg in 1925 and 1926. In 1927, there were shown at Berne several patients, the complete disappearance of whose detachments lasted for several months, and in the following year, at Lucerne, my colleagues, Siegrist of Bern and Vogt of Zurich, referred to their own confirmatory experiences, this was the first occasion on which credit was given to me by ophthalmologists other than my own fellow-workers and assistants. Later descriptions of cases or of the method were published in Italian, French or German in the reports of the ophthalmologic meetings held at Palermo, Paris and Heidelberg in 1928, and a more detailed account was published in the *Annales d'oculistique* (May, 1930).

My actual experience on about 250 patients and in more than 300 operations can be summed up as follows

1 In more than 95 per cent of the cases, whenever ophthalmoscopic examination is possible, one or several holes may be detected in the retina if looked for with sufficient care

2 In about 10 per cent of these cases, the hole is not in the retinal tissue, but consists of a rupture or tearing away from its insertion at the ora serrata ("desinsertion of the retina")

3 In all recent cases, when the hole or tear has been closed, cure is immediate, complete and permanent

4 In older cases (several weeks or months), closing the tear stops the detachment and may produce a more or less complete reposition of the retina, but restoration of the vision generally remains incomplete

5 If the detachment relapses, it is found that the tear has not been completely closed or that there was another tear which had not been previously seen

6 A recurrence of detachment in a different region of the eye is due to formation of a new hole in the retina

It may appear curious that all my communications on that matter, numbering more than twenty, remained unknown to the English-speaking ophthalmologic world until the International Congress in Holland, no mention of them even being made either in an article in the *Lancet*^[32] or in an article by Stallard^[33] in the *British Journal of Ophthalmology*. Only following a recent visit to me at Lausanne by five members of the staff of the great eye hospitals in London were my ideas set forth in two papers published by Juler^[34] and by Ormond^[35]. The

31 Gonin, J. The Treatment of Detached Retina by Sealing the Retinal Tears, *Arch Ophth* 4 621 (Nov.) 1930

32 Operation for Detachment of the Retina, editorial, *Lancet* 2 1202, 1929

33 Stallard, H. B. *Brit J Ophth* 14 1, 1930

34 Juler, F. *Brit J Ophth*. 14 73, 1930

35 Ormond, A. W. *Brit M J* 1 940, 1930

former described accurately the preliminary procedures^[36] and the operation of thermopuncture, the latter dealt adequately with the anatomic side of the question^[37]

There is no doubt that a hole in the retina, a disinsertion of the retina at the ora serrata or a traumatic rupture of the retina play a tremendous part in (1) the causation of retinal separation, (2) the continuation of the separation and (3) the results of the operative closure, i e, is wholly responsible for the success or failure of the operative treatment. At least one, and probably more, of these factors are involved in every case of retinal detachment observed for treatment (These factors will be considered later in a more logical sequence). They are mentioned at this time only to give further credit and honor to Gonnin for his brilliant pioneer work. Since the advent of diathermy, chemical cauterization, superficial scleral electrocoagulation and catholysis, Gonnin's thermocoagulation has been utilized in a steadily decreasing number of cases. It is to be remembered always, however, that his procedure was the first which could give the patient and the operator any assurance of a favorable result.

ETIOLOGY

In considering the etiology of retinal separation one must of necessity consider the mechanism of the separation. The two cannot be separated, together they comprise the pathogenesis. Further, it is impossible to divide retinal separations into a primary and a secondary classification, and even the recent subdivision of Troncoso,³⁸ who divided detachments into the idiopathic and the symptomatic type, and of Arruga,³⁹ who considered symptomatic detachment of the retina as

36 [Footnote quoted from the original article] The whole procedure of locating the tear and preparing for the operation, as seen by Dr. de Jaeger, of Bruges, at Lausanne, has been again described by his partner, Dr. Rubbrecht, whose paper is summarized with full particulars and sketches by J. B. Lawford in the *British Journal of Ophthalmology* (**14** 359 [July] 1930). The only difference in Dr. Rubbrecht's method is that he makes a long scleral wound for the cauterization.

37 [Footnote quoted from the original article] On a single point of importance, Ormond seems not to have realized my own conception of the changes in the vitreous. He stated that "liquefaction of the vitreous takes place." I do not think it does. I noticed only a separation of the vitreous fluid from its fibrillar mass, the latter being penetrated with epithelial cells and keeping thus a retractile strength sufficient to drag on the retina. I generally compare the retracted vitreous with a squeezed lemon, the cellular part of which has been not liquefied but simply deprived of its juice.

38 Troncoso, M. U. *Internal Diseases of the Eye and Atlas of Ophthalmoscopy*, Philadelphia, F. A. Davis Company, 1937.

39 Arruga, H. *Detachment of the Retina*, translated by R. Castroviejo, New York, B. Westermann, 1936.

belonging to a general group, with detachments caused by myopia, traumatism and chorioretinal conditions as other equally important subdivisions, are unsatisfactory and not correct. For this reason, such a classification will not be considered here.

When pathologically involved, the retina splits within itself, separating into two layers, the outer layer of pigment epithelium remains adherent to the choroid, while all the other layers are lifted from the pigment epithelium by serous exudates or hemorrhage or because of edema, cystic degeneration, neoplasms or cysts. Zeeman and Oltmanns⁴⁰ discussed in great detail this matter of trauma to the tissue and retinal separation. Their arguments and conclusions as to the mechanics of separation, based on cases of post-traumatic separation, are quoted in part.

Structural cohesion is a function of the supporting tissue of the retina and also of the surrounding tissues so far as they are connected with the supporting tissue, that is to say, of the outer layers of the vitreous in its foremost part.

The actual supporting tissue consists of Muller's fibers and *Spinnenzellen* (neuroglia cells). Muller's supporting fibers stand between the *membrana limitans interna* and the *membrana limitans externa* like so many columns which carry and hold together the retinal elements, together with numerous smaller glia cells, the thin fibrils of which encircle nerve fibers, ganglion cells and granular cells and dissolve into a kind of limiting layer (*membrana limitans perivascularis*) against the walls of the blood vessels (Kruckmann). The broad ends of Muller's fibers, their cuticular thickened *Basalplatten* (basal plates), unite into a continuous limiting membrane, which extends into the cuticle of the ciliary epithelium. This layer, which by reason of its continuity resembles a boundary, a frontier between territories of different structure, can derive some strength from the solidity of its cellular construction. Yet we agree with Leber in considering the connection of the retinal elements translaterally looser than between its layers.

Denig, with his experiments, not only refuted Berlin's idea that the picture of *commotio retinae* is connected with a subretinal hemorrhage but observed strange bumps reminiscent of a chain of pearls on the inner surface of the retina which made him presume that there were distention of Muller's fibers and small tears in the *membrana limitans*. Beginning detachment of this layer must, according to Lohmann, too, be the cause of the picture in cases of *commotio retinae*. Leber further pointed out that it has always been designated incorrectly as edema, that it is of no use to indicate the source which should produce the edema (in older theories, the vessels, in Denig's theory, the vitreous) and that in any case a swelling opacity is a much more suitable term. Microscopic tearing of the tissue elements, secretion of myelin, with its well known swelling capacity, a granular, intensely white opacity and rapid resorption are the bases of the picture.

Differences in degree, greater distention of the retinal elements, wider tearing of the *membrana limitans* and tearing of the thickened outer layer of the vitreous create the conditions which, according to clinical observation, may later lead to the formation of a hole or to detachment.

Zeeman and Oltmanns expressed the belief that there are three definite facts of importance, i. e., (1) that the time which elapses between the onset of the lesion and the separation, which is its indirect conse-

⁴⁰ Zeeman, W. P. C., and Oltmanns, H. J. Trauma and Retinal Detachment, Arch Ophth 13 971 (June) 1935.

quence, is variable and may be long, (2) that the retinal alterations which are first seen by the ophthalmoscope may be rather diffuse, spotted or cystic but hardly characteristic of changes universally considered as having been produced by trauma, and (3) that an eventual tear or separation at the ora serrata is not always the direct consequence of trauma. If the trauma is too light to cause commotio retinae, i. e., too slight to give rise to a beginning separation and thereby predispose to complete separation, it is impossible to assume that the separation which presents itself weeks or months later is due to this trauma.

As Zeeman and Oltmanns further stated, the relation of various traumatic and spontaneous forms of detachment makes it probable that the point of attack and the transference of the active forces (pathologic or physiologic) differ little if at all. The point of attack must lie in the supporting tissue of the retina. Only structures which are normally attached to the retina transfer forces to it and these are the anterior parts of the vitreous and the zonular fibers, that is, the very elements which play an important mechanical part in the physiologic accommodative mechanism. It is probable that the zonular fibers and the structures in the anterior parts of the vitreous show the lines of force and direction along which the active forces and the finer tissue shiftings connected with these active forces travel when the accommodative mechanism is brought into play, but these lines and forces must also be recognized as a menace to the retina, constructed as it is for some other function. There is an incongruity in the requirements seemingly made on the retina. The retina in a more limited sense, the sensitive derivative of the optic vesicle and part of the central nervous system is in this instance passive, it does not bear but is borne, it scarcely resists but does not succumb. The skeleton of supporting tissue against and in the retina and along its vessels is in casu the important part in question here. This bears, resists, gives way and tears. It is not proved and is at least doubtful whether the Blessig grooves in the retina near the ora serrata may be called predisposing elements for tearing and detachment. This may be conjectured only so far as they are the consequence of, or are accompanied by, congenital or acquired weakness of the supporting elements, i. e., Muller's fibers and the membrana limitans interna, the glia coat and connective tissue along the vessels, the fibrous, thickened outer layer of the vitreous and the membrana hyaloidea posterior, and of the connections between these various elements. Coarser connections may be found among the innumerable more delicate elements. Tearing of the membrana hyaloidea and of the membrana limitans interna means tearing of the intraretinal supporting tissue and hence the first step in detachment of the retina. No wonder that the edges of the retinal tear often seem to lie in the normal retina even if there is a serious decrease in thickness in the immediate vicinity. Therefore, predisposing factors for complete separation appear to be all those factors

which cause in the delicate structure distention and fragility and thus promote the development of a tear in the membrana hyaloidea posterior. The reactive phenomena along the walls of the torn vitreous appear to confirm the great significance of this tear as only rarely do they prevent such complications as retinitis proliferans or retinal and preretinal hemorrhages and never do they restore the retina.

Speciale-Cirincione⁴¹ studied a series of enucleated eyes histologically to determine if possible the sequence of events which occurs in the eye with retinal separation. He described four stages. In the first stage the rods and cones are only slightly separated from the pigment epithelium, a number of processes from the rods and cones still being connected with the pigment epithelium but many being broken. No changes in the structure of the rods and cones are noticed. The vision in such an area may be as high as 1/10 and perception of color is present. In the second stage all the protoplasmic connections between the rods and cones and the layer of pigment epithelium are broken and massed together in a sort of membrane. This layer seems to pull the rods and cones in one direction at their external end. The vision in such an area is limited to perception of hand movements, and blue is the only color which can be distinguished. In the third stage swelling and degeneration of the rods and cones themselves become evident. The cones show more degeneration than the rods. The vision is limited to uncertain perception of light. In the fourth stage observed in eyes with long-standing retinal detachment no trace is left of the rods and cones, and marked degenerative changes are seen in the ganglion cells and other cells of the retina. The pigment epithelium in all these stages show two types of change, an irregularity in the arrangement of the cells and a tendency to proliferation through the subretinal space. Speciale-Cirincione expressed the belief that in the first and second stages before changes in the rods and cones have occurred, a return of useful vision is possible if reattachment can be obtained.

Recently it was my privilege to study an eye microscopically⁴² after a fairly successful reattachment had been obtained by means of the classic Guist-Lindner technic for trephining the sclera, with the subsequent application of potassium hydroxide. (The patient died in a state of diabetic coma three years after operation. She had been in a state of coma repeatedly for three months before her death at which time she had preretinal, retinal and subretinal hemorrhages and complete hemiplegia on the left side.) The following quotations appeared in my previous report:

41 Speciale-Cirincione. *Ann di ottal e clin ocul* 53:641, 1925

42 Spaeth, E. B. Reattached Retina. *Physiologic, Ophthalmoscopic and Microscopic Observations and Comparisons*, *Arch Ophth* 14:715 (Nov) 1935

43 Footnote deleted by author

Slow progressive retinal as well as choroidal degeneration was seen. Subretinal exudates and hemorrhages were present to outline the limits of the detachment—in some places to a marked degree, and in others in such slight amounts that one could just see a thin line of exudate, in some areas with, and in others without, early changes in the layer of rods and cones. A few less definite changes were seen in the epithelial cells. The layer of rods and cones seems to be the index of retinal viability. When these were not present as healthy looking elements, the other layers of the retina were also markedly impaired or even wholly wanting. The outer and inner nuclear layers when normal were rather certain to be accompanied by normal-appearing rods and cones.

In the light of the observations just mentioned it is likely that the degree of recovery, in all its details, depends largely on the presence of healthy rods and cones and the absence of certain irregular subretinal cells, which probably are proliferated, epithelial cells. The observation of cholesterol crystals microscopically is almost certain proof that degeneration is so extensive that failure is certain.

Anatomically, it is known that detachment develops between the outermost limits of the layer of rods and cones and the epithelial layer of the retina, i. e., in the layer of pigment epithelium. This region of separation is the space (if one can call it such) through which nourishment, by osmosis perhaps, normally reaches the outermost layers of the retina. It is possible that even in the presence of detachment these rods and cones continue to receive nourishment through this subretinal fluid, for in certain cases this fluid may be nothing but a transudate and pathologic only mechanically by reason of its presence. If it were pathologic in the inflammatory sense in all cases, the reason that recovery in some instances is so nearly complete, as observed grossly, is difficult to understand. At the same time, one can understand more readily the cause of the failures. Microscopic sections of detached retinas seem to show that as long as a certain number of filaments lying between the cells [see Zeeman, Oltmanns and Deng] of the pigment epithelium remain unbroken degenerative changes do not develop in the epithelium and in the layer of rods and cones. Only after loss of continuity has occurred do the multiple changes appear that are so commonly observed in detached retinas of long standing. It is perhaps through this maintained integrity of structure that recovery of function can occur after reattachment, even when detachment has existed for an indefinite length of time.

Arruga's and Troncoso's group of cases of primary, or idiopathic, detachment included cases of traumatism, myopia and chorioretinal diseases. They stated that the detachment in these cases should be called primary in that the retinal process was only distantly related to its original cause. Their group of cases of symptomatic, or secondary, detachment included cases of nephritic retinitis, acute uveitis, acute scleritis and orbital cellulitis, intraocular tumors and cysticercosis, and cardiovascular-renal conditions and diabetes. The detachment in these cases was considered secondary in that the development, course and prognosis of the separation was closely allied to an original and evident underlying basic disease. The manifest fault with this classification is that it considers these various types of detachment not as a symptom of the underlying disease (for this is what they are) but as the disease itself and pays only indirect attention to pathologic anatomy, that is, to

the underlying histopathologic factors. These include pathologic changes in the vitreous, disease of the sclera itself, disturbances in the equilibrium and the state of health of the intraocular fluids, disturbances in the relationship between intraocular tension and intravascular pressure, pathologic conditions of the choroid, retinal degeneration (retinosis), retinal exudates, retinal edema and retinal hemorrhages, and finally traumatic rupture and perforation in the choroid or in the retina. A consideration of retinal separation on the basis of idiopathic and symptomatic grounds may consistently include certain of these factors in any one type of separation, but it is equally certain that a histopathologic consideration, and that it is simply mechanistic, must be the basis for research and the study of the causes of retinal detachment. Success in the surgical treatment rests wholly within the realm, both in the possibilities and in the probabilities, of these anatomic principles. Gonnin in 1919 stated that the large percentage of failures which occur in the treatment of this condition are due to the fact that the mechanism producing it is so poorly understood.

Anderson¹⁰ considered four possible basic mechanisms, and into these four possibilities one may fit the various histopathologic probabilities. They are the theories of distention, of depression, of attraction and of exudation. Anderson's bibliography pertaining to these mechanisms is complete up to 1930.

According to the theory of distention, stretching of the choroid and the sclera is the actual cause of the detachment. Myopia, degeneration of the retina and trauma are all to be included in a consideration of this theory.

The theory of depression is the reverse of that of distention. A sudden loss of vitreous, atrophy of the eyeball, hydrostatic changes, ocular hypotension, detachment of the choroid, the hypotony of diabetic coma and the transudates and exudates of inflammatory processes are included in a consideration of this theory. Apparently these conditions modify or permit retinal separation by the withdrawal of a force which normally should assist in keeping the retina in position. It is doubtful whether hypotony of itself is the cause of separation. Certainly in most cases seen early in their course hypotony is not present, nor as Gonnin⁴⁴ stated is it pronounced until many days or weeks from the time when the subretinal diffusion might be expected to stop. If the effusion plays the role of a compensatory exudation, the hypotony does not explain the limited character or the limited form of retinal tears. Moderate hypotony of itself is no contraindication to surgical intervention, and its disappearance after thermopuncture, electrocoagulation or catholysis is outstanding. This matter of effusion and of hypotony has been rather

44 Gonnin, J. Arch. d'opht. 51 426, 1934

well answered by a recent clinical case. After an uneventful corneoscleral trephining for simple noninflammatory glaucoma, a high degree of "choroidal" detachment developed. Sclerotic changes in the lens prevented any detailed observation of this, but visibility was adequate for diagnosis and for subsequent observation until complete recovery occurred. Some time later an equally uneventful intracapsular extraction was done. The ocular tension was constantly less than 12 mm of mercury, both before and after extraction of the lens. After extraction one could see in the fundus at the site of the former "choroidal" detachment parallel curved lines of retinal striae outlining indelibly the position of former effusion in the presence of extreme hypotony. This case suggests an answer to the question.

The theory of attraction considers largely the possibilities of a pathologic process of the vitreous being the principal cause for the retinal separation. This theory, first considered by Hanssen⁴⁵ as an "attraction theory," is probably most intimately connected with the entire question of retinal tears, though the theory of distention has this same important relationship. The theory of distention undoubtedly explains retinal separation in myopic eyes and in those affected by trauma, with their frequent retinal tears, but it cannot explain the same condition in nonmyopic eyes and in eyes not affected by trauma. Muller,⁴⁶ Leber,⁴⁷ Lauber,⁴⁸ Gonin,⁴⁹ de Wecker,⁵⁰ Nordenson,⁵⁰ Heine,⁵¹ Pillat,⁵² Iwanoff,⁵³ and Lindner⁵⁴ are among the men who have felt that strands of vitreous, epithelial cell proliferation, the preretinitis of Leber, equatorial choroiditis, fibrillary degeneration, detachment of the vitreous, contraction of the vitreous and retinitis proliferans are likely causes.

The theory of exudation existed long before the present era of surgical correction for retinal separation. Leber stated that in the days before the invention of the ophthalmoscope "detachment" of the retina was recognized only after enucleation and was known⁵⁵ as subchoroidal

45 Hanssen, R. *Klin Monatsbl f Augenh* **63** 295, 1919

46 Muller, H. *Anat Beitr z Ophth* **41** 363, 1858

47 Leber, T. *Tr Internat M Cong* **3** 15, 1881

48 Lauber, H. *Klin Monatsbl f Augenh* **72** 547, 1924

49 Gonin, J. *Klin Monatsbl f Augenh* **71** 232, 1923, **83** 667, 1929

50 Nordenson, E. *Die Netzhautablosung*, Wiesbaden, J F Bergmann, 1887, p 4

51 Heine, L. *Klin Monatsbl f Augenh* **72** 305, 1924

52 Pillat, A. *Klin Monatsbl f Augenh* **69** 429, 1922, *Ztschr f Augenh* **57** 347, 1925

53 Iwanoff, A. *Arch f Ophth* **15** 1, 1869

54 Lindner. Personal communication to the author, 1934

55 Leber, in von Graefe, A, and Saemisch, E T. *Handbuch der gesamten Augenheilkunde*, ed 2, Leipzig, Wilhelm Engelmann, 1901-1911, vol 7, pt 2, chap 10

hydrops The theory of exudation was originated by von Graefe,⁵⁶ who felt that serous chorioiditis was the primary lesion in the majority of cases of spontaneous retinal separation Detachment associated with intraretinal fluids, especially in those instances in which a fluid vitreous is present, with intraretinal hemorrhage, with nephritic chorioretinitis and nephritis; with the toxemia of pregnancy, and with inflammatory processes, such as iridocyclitis, tenonitis, tuberculous chorioretinitis and sympathetic ophthalmia, and the detachment which follows actinic retinitis are types which can be fitted within this classification Experimental retinal separation is developed wholly on this basis It is likely that the primary pathologic process in these instances reacts subsequently and unfavorably on the vitreous, causing degeneration of the vitreous and subsequently retinal separation through attraction or traction

These four possibilities do not include the possibly mechanical separations due to cysts of the vitreous, choroidal and retinal neoplasms and retained intraocular foreign bodies It is possible that any one, or perhaps all four, are relevant in these instances

RETINAL TEARS

The presence of tears (holes) in the retina is a finding which is associated with practically all types of retinal separation, even those connected with inflammatory and exudative states They may be subdivided into two general groups, disinsertions and all other tears This subdivision is superficial and not especially satisfactory but is to be considered in that different types of etiologic factors are likely responsible for these two different types of tears

A disinsertion is an oval or slit-shaped rent in the retina, occurring at the ora serrata and probably connected with cystic degeneration in the neighborhood of the ora serrata At this point the retina separates from its pigment epithelium, i. e., there are no retinal elements, except the pigment epithelium lying peripheral to the rent Traumatism and traumatic rents in the retina often simulate these disinsertions Disinsertions may be multiple, with the separated lobes lying close to each other Two or more of these may unite as a larger rent, and in such instances a small island or flap of the peripheral portion of the retina can be found intact and still adherent to the ora serrata These findings do not deny the statement just made, they simply indicate the process through which that disinsertion passed in its formation and in the formation of the peripheral flap still present

This peripheral rent is often difficult to find ophthalmoscopically The folds in the retina prevent a view of the peripheral portion of the retina, so that with even extreme dilatation of the pupil it may be impossible to view the neighborhood of the ora serrata In every case

⁵⁶ von Graefe, A Arch f Ophth (pt 2) 2 202, 1855, footnote 17a

in which the greatest elevation lies closest to the periphery one must suspect this type of tear. Further, when a most careful search of the fundus, frequently repeated, fails to show a hole in the retina at any place, then one should also consider the likelihood of a disinsertion being present. Changing the position of the patient's head and his body in bed or in a chair so that the retinal folds can fall away by gravity from the periphery will at times permit the operator to see these disinsertions. Preliminary subretinal drainage with a hypodermic syringe and needle is permissible in the case of larger bullous detachments and may also uncover a probable disinsertion. Pressure on the sclera while viewing the fundus as far peripherally as is possible has been suggested by Arruga as being of great assistance. Transillumination along the circle of the ora serrata has been definitely valuable in 3 cases which came under my observation, for it was the means of locating the disinsertion. Finding a disinsertion makes possible a much lessened amount of diathermic coagulation in that the rent can be completely sealed from the remaining portion of the retina, making unnecessary extensive treatment of the entire sclera overlying all of the separated retina, as would be almost obligatory in cases in which no disinsertion or other hole could be found.

The consideration of the remaining types of retinal tears is an interesting subject. The percentage of cases in which tears are found varies a great deal with the individual ophthalmologist. Arruga stated that in 1925 he found rents in 40 per cent of his cases of retinal detachment, in 1928, in from 60 to 65 per cent, and in 1932, in 80 per cent, at the present time he is finding them in from 85 to 95 per cent, depending on whether they are cases of old or of recent separation. Tears are most readily found in cases of recent separation in which the media are still transparent, but as the weeks pass after the onset of a retinal separation it becomes increasingly difficult to find them. Rest in bed, dehydration of the patient and subretinal drainage through scleral puncture are not infrequently necessary before the hole can be found. Multiple rents appear in from 25 to 30 per cent of cases. Because of this, the finding of a single hole should not halt further detailed search for additional tears. In the majority of instances the tears are located in the superior half of the retina, the temporal quadrant first in frequency, and the nasal quadrant second. The inferior temporal quadrant is third and the inferior nasal quadrant last in this order of incidence. This is diametrically different from the disinsertions, for the majority appear in the inferior periphery of the retina rather than in the superior periphery. The order of frequency from the periphery toward the macula is, first, between the ora serrata and the equator, second, in the macula itself, and, third, in that portion of the retina which lies between the macula and the equator. The form or the shape of these

retinal holes is as variable as are the innumerable potential probabilities. They may be many cornered, round or oval and arranged in groups or in rows or after the manner of perforations in coarse lace, they may be subdivided by an overlying retinal vessel or lying between the branches of a retinal vessel, they may be horseshoe shaped or crescent shaped, with the calks of the horseshoe or the horns of the crescent toward the periphery, and with or without hinged flaps, they may appear as large continent-like lacerations from trauma, involving sometimes an entire quadrant of the retina, or as slitlike holes, narrow and elongated, resembling hemorrhages and at times confused with hemorrhages, they may also occur as a concentrated group of tiny sieve-like perforations. The macular holes are most commonly symmetric, while those at the equator are usually of a crescentic or a horseshoe shape. The region between the equator and the periphery presents the greatest variety of holes, while at the extreme periphery holes and disinsertions again assume a more regular outline, being large and spindle shaped, with their long axes parallel to the curve of the ora serrata. Traumatic

TABLE 1—*Incidence of Various Types of Holes in Relation to Refractive Error*

	High Myopes	Low Myopes	Emmetropes	Aphakics
Disinsertion	3	6	27	1
Round	15	8	6	1
Arrow head and horse shoe	12	7	4	
Slit	2	2	1	
Irregular		1	3	1

lacerations are of all varieties and shapes and usually are the largest in size. Not commonly, but still not rarely, one may find both disinsertions and extraperipheral tears present in the same case. Extramacular and macular tears may also be present simultaneously, this is more rare but has been observed.

Shapland's⁵⁷ classification of the holes in 100 cases is quite a satisfactory one.

(1) Festoon-shaped, situated at the extreme periphery, and corresponding to Prof. Gonn's "disinsertion" at the ora serrata.

(2) Rounded, punched-out holes, suggesting the possibility of a pre-existing cystic degeneration of the affected portion of the retina, or an origin in a previous focus of choroiditis.

(3) Arrow-head and horse-shoe-shaped rents, in which the point of the arrow or the convexity of the horse-shoe are always on the disc or central side of the base of the arrow-head or concavity of the horse-shoe.

(4) Radial slit-like tears. It is worth pointing out that the limbs of a horse-shoe rent may be mistaken for two radial tears, the convexity of the horse-shoe being hidden behind a secondary fold of detachment.

(5) Irregular rents.

⁵⁷ Shapland, C. Tr. Ophth. Soc. U. Kingdom **51** 152, 1931.

In Shapland's series there were 37 cases of the first type, 30 of the second type, 23 of the third type and 5 each of the fourth and fifth types. Table 1 (Shapland's) shows the incidence of these various holes as connected with the refractive error.

Gifford,⁵⁸ before Gonin's death (which occurred June 10, 1935), questioned him (Gonin) as to the success he had had in cases in which the holes were large. Gonin's picturesque words follow verbatim:

C'est plus facile de tirer sur un elephant que sur un lapin, mais pas si facile de le tuer (It is easier to shoot an elephant than a rabbit, but not as easy to kill him).

THE MECHANICS OF THE FORMATION OF RETINAL TEARS

A consideration of the mechanics of the formation of retinal tears includes a consideration of two basic factors, the architecture of the eyeball and the various structures related to it and the strong probability of different development of macular and extramacular tears. The explanation of the influence of the first of these factors is based on the relationship which exists between the rigid sclera, the inelasticity of the choroid and Salzmann's observation that spontaneous tears occur in the lamina vitrea, not involving the choriocapillaris. Anderson stated that Parsons concurs in this. The pressure of the various rectus and oblique muscles on the eyeballs may be a factor in changing intraocular pressure because of occlusion of the vortex veins. This would be an example of an increase of intraocular pressure due to physiologic pressure as outlined by Edridge-Green and again referred to by Anderson. Next is the consideration of the vitreous itself so far as retinal separation is concerned, the vitreous must be classed as a tissue subject to the changes that all tissues are subject to, and by its intrinsic attachments to the retina itself it will affect the retina secondarily when undergoing edema, shrinkage and inflammation or when lost, though only in part, through operation or by trauma. The last two factors are also considered by Comberg⁵⁹ and by Levinsohn⁶⁰. Comberg has shown that there is a vibration of the posterior portion of the eye when one is reading and that the eyeballs are pushed backward when the lids are closed for an amount equal to from 15 to 20 Gm. in weight. Levinsohn considered that the position of the head and the effect of gravity are most important. He developed myopia experimentally in monkey's eyes by keeping their heads downward and concluded thereby that the abuse of the eyes in ordinary life is a main factor in the development of this, rather than heredity, this being only one of several other determining factors.

⁵⁸ Gifford, S. R. Surgical Treatment of Retinal Detachment, Arch. Ophth. **16** 405 (Sept.) 1936.

⁵⁹ Comberg, W. Ber. u. d. Versamml. d. deutsch. ophth. Gesellsch. (1928) **47** 126, 1929.

⁶⁰ Levinsohn, G. Arch. f. Augenh. **102** 308, 1929.

In addition to these factors, further conditions may be relevant, so far as macular holes are concerned. The effects of direct trauma, especially when applied to the anterior segment of the eye, is significant. Tears produced in this manner are almost always round or oval, while the more frequent peripheral tears are angular, crescent shaped and slitlike, being ragged and usually large. It seems as if the macular holes due to trauma suggest an explosive effect, as if increased pressure in the fluids within the retina was released forward, toward and at the macula. Also, a more intimate anatomic relationship which exists at the macula between the retina and the choroid is undoubtedly a further factor.

There is no doubt that a definite relationship exists between retinal holes and retinal separation. The latter is dependent wholly on the first, and without the development of the first, forces which would otherwise cause retinal separation will be expended and their effects come to naught, i.e., while a potential separation is in process, spontaneous recovery occurs from these potentialities because a tear does not develop. In supporting Leber's statement that sudden retinal detachment could not occur without a hole, Lister⁶¹ stated

With an intact eyeball and an intact retina, no detachment can take place unless there is a simultaneous and practically corresponding outpouring of fluid into the inter-retinal space on the one hand and contraction of the vitreous on the other. Such pouring out of inter-retinal fluid and absorption of the vitreous cannot, of course, take place suddenly. The onset of any separation of the retina without a hole must, therefore, of necessity be gradual. It would, therefore, appear that if it can be substantiated, in any given case, that the loss of sight from separation was not merely suddenly detected but actually occurred suddenly, we can definitely predict the presence of a hole, whether or not it can be seen by the ophthalmoscope.

According to Anderson, one can conclude that the majority of detachments which arise suddenly are associated with the formation of a hole but not that the relationship is essential and absolute. In the light of more recent surgical methods and the more recent statistics, I believe that this relationship is "essential and absolute."

A retinal tear from an adherent contracting vitreous, with the tear and separation starting above, will permit subretinal fluid to infiltrate from above with but a minimal separation there (above), however, there will be a maximum of separation below. This is a finding seen not uncommonly. In these instances infiltration of vitreous through the tear is not as important as is that of the subretinal fluid. An area of preretinitis will cause adherent bands in the vitreous to the vitreous surface of the retina, resulting in a hole with an initial minimal separation. The separation then will continue and will grow owing to the passage of vitreous through this hole as a result of constant ocular

61 Lister, W. Brit J Ophth 8 1 and 305, 1924

rotations and unceasing rotary shocks to the vitreous body, on account of its inertia, as first outlined by Lindner, and not because of any continued traction on the retina. This force was expended when the retinal tear occurred, and the damage is continued by subretinal prolapse of the vitreous. In the formation of the common crescent-shaped and horseshoe-shaped holes, the hole always lies on the distal side of a fold with the aims of the tear toward the ora serrata because the pedicle of the fold or flap remains attached to the retina near to the ora serrata.

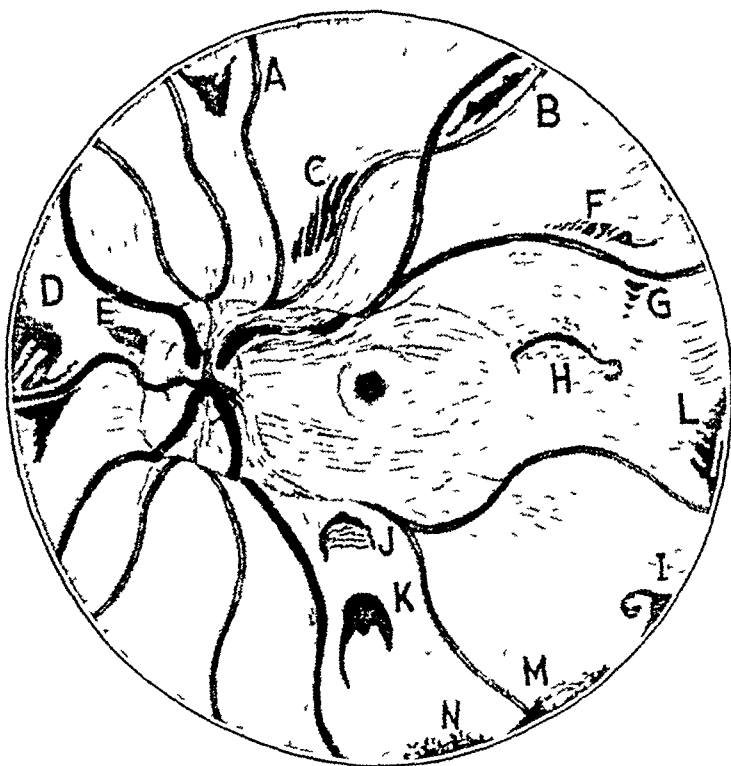
The accompanying illustration is a composite drawing of an actual instance of retinal separation with a small single hole at the macula. There were no other tears, rents or disinsertions present. The other holes illustrated have been sketched in at the approximate positions where they have actually appeared in other patients, as closely as this sketch would permit. The drawing thus shows some of the various common rents found.

A and *K* are the ordinary horseshoe-shaped tears mentioned previously. *B* is a small peripheral traumatic (direct blow to the eyeball) rent. *D* is a peculiar shaped peripheral tear with two flaps, the larger of the two flaps being folded in part on itself and the smaller lying free, a bridge passed over this tear continuing along a large retinal vein. *E* and *H* are two linear tears which appeared on the dome of bullous separations. *H* was undoubtedly a rent at an area of choroiditis. *E*, following operation and the reduction of the separation, turned out to be a considerably larger tear, bilobed with the larger lobe hidden underneath the fold. *I* and *L* were the two extremes of a tear which lay at the extreme ora serrata, which certainly resulted from peripheral cystic degeneration of the retina. *J* is a tear with a wrinkled flap, similar to one which Arruga demonstrated in his case. *M* and *N* show the appearance of the usual retinal disinsertion.

When one considers, therefore, the scleral elongation produced by myopia and the pathologic changes in the vitreous in myopic eyes, the many possible structural changes due to chorioretinitis, Arruga's pertinent observation that the retina does not detach, when it is adherent to both the vitreous and the choroid, as in disseminated choroiditis, while equatorial and peripheral chorioretinitis frequently accompanies separation of the retina, the various atrophic and cystic degenerations which occur in the retina, the possible structural changes in the retina, which are of a developmental nature (considered highly important by Vogt), adhesions between the vitreous and the retina, the multiple minimal traumatism to which the eyes are constantly subjected, and the elasticity of a ruptured pathologic retina, then only is one aware of the factors concerned in the development of retinal rents. When one adds to these certain conditions practically unknown, such as disturbances

of intraocular tension, intravascular pressure or fluid equilibrium and the effects which the albumins of the vitreous and the water of semi-fluid vitreous may have on the retina and choroid, one should not be astonished at the number of retinal separations which occur. Conversely, one must agree to the absolute relationship which the tear plays to retinal separation and, even more, express sincere astonishment that such separations are relatively infrequent.

The relationship which retinitis plays to retinal separation, either from direct damage or because of a response to the toxic effects of tissue damage, was shown rather well by Harman and MacDonald⁶² in their paper on detachment of the retina due to exposure of light dur-



Composite drawing of a retinal separation with a small hole at the macula (The letters are explained in the text)

ing an eclipse. The eye showed an extensive separation of the retina involving the whole of the upper half of the eye, the separation being ballooned downward so as to obscure the macula and the disk. The sequence of events in their case seemed to be as follows: (1) severe damage to the macula, consequent to exposure to sunlight, (2) a succeeding infiltration of the macular region, (3) extension of the exudate in such a quantity as to cause a gross separation of the retina, (4) absorption of the greater quantity of the fluid during the weeks of rest, (5) exposure (uncovering) of the initial area of damage in the macula, and (6) onset of scarring in the macular region.

⁶² Harman, N. B., and MacDonald, P. *Brit. M. J.* 1: 637, 1922.

Knapp⁶³ reviewed Van Manen's analysis of 124 cases in which operation was performed during 1935 at the Weve clinic in Utrecht. There were 31 failures in the series. Ten of these must be deducted for sufficient reasons.

The author arranged the cases into groups for a survey of the results. Group 1 consisted of cases of retinal separation in aphakic eyes. This group was further subdivided into two groups: cases in which detachment occurred in an aphakic eye and those in which the detachment preceded the extraction, requiring that the cataract first be extracted. In the first group 6 operations were performed with success in 4, in the second, 7 operations with success in 1. Group 2 was composed of 4 cases of retinal detachment which followed a perforating trauma, cure was effected in 2. Group 3 consisted of 3 cases of detachment with a tear in the fovea, cure was obtained in all. (Though improvement in vision occurs, coagulation in the macular region prevents the return of good vision, and a central scotoma is always present.) Group 4 consisted of cases of retinal detachment in myopic eyes with giant tears, 4 operations were performed, 3 of which were successful. Group 5 was composed of 20 cases of retinal detachment with a tear at the ora serrata, recovery occurred in all. Group 6 consisted of 32 cases of detachment with the larger part of the separation situated above the horizontal meridian, successful results were obtained in 26. (The treatment in these cases was the same as that usually given, except that the degenerative areas surrounding the tears or suspicious areas in any other meridian were, in addition, treated by surface coagulation.) In group 7, cases of retinal detachment in which the larger part of the detachment was situated below the horizontal meridian, 25 operations were performed with success in 20. Group 8 consisted of cases of detachment with centrally situated folds in the retina, 8 operations were performed with success in 2. In group 9, cases of nonidiopathic separation and retinitis proliferans, 4 operations were performed with failure in 3.

The summarization of two series of cases, that of Baer and Shipman,⁶⁴ and that of Dunnington and Macnie,⁶⁵ is presented in table 2. This table illustrates certain salient and interesting relationships. (It is remarkable to note the close similarity in percentages in many of the comparisons.) These data are presented not from a therapeutic standpoint but to show and emphasize common etiologic factors and the factual relationships such as sex, age, retinal tears and myopia.

63 Knapp, A. Die diathermische Behandlung der Netzhautablösung in der Universitätsaugenklinik Utrecht und ihre Ergebnisse im Jahre 1935, by J. G. Van Manen, *Book Reviews, Arch. Ophth.* **17**: 387 (Feb.) 1937.

64 Baer, B. F., Jr., and Shipman, J. S. *J. M. Soc. New Jersey* **34**: 494, 1937.

65 Dunnington, J. H., and Macnie, J. P. Detachment of the Retina. Operative Results in One Hundred Fifty Cases, *Arch. Ophth.* **13**: 191 (Feb.) 1935.

RETINAL SEPARATIONS FROM DIRECT TRAUMATISM

The force of a traumatism need not be one, necessarily, of great degree, in fact, injuries from objects such as soft balls, from a bump on the back of the head or from a sudden jar in missing a step are as frequent an etiologic factor as are injuries from a blow with a fist, from sticks, elbows or stones, from a sharp edge of a door or from the back of a chair. These are actual injuries which occurred in various cases. Penetrating injuries are somewhat different in effect. In such cases the choroid is also ruptured, perhaps even more extensively, so that the hole develops suddenly and abruptly. Sub-choroidal and subretinal hemorrhages appear as precipitously and the

TABLE 2—Summary of the Data on Two Series of Cases of Retinal Separation

	Baer and Shipman 44	Dunnington and Macnie 150 (155 eyes)
Total number of cases reported		
Females	31.8%	36.0%
Males	68.2%	64.0%
Cases in which myopia of varying degree was the refractive error	50.0%	61.9%
Cases in which 2 operations were necessary and done	27.2%	16.4%
Cases in which 3 operations were necessary and done	4.5%	4.5%
Cases in which retinal tears were seen	40.9%	56.0%
Successful results in cases in which tears were seen	55.5%	41.8%
Successful results in cases in which tears were not seen	38.4%	34.7%
Cases with history of injury	29.6%*	41.3%
Successful results in cases following injury	46.1%†	36.6%‡
Total cases with no improvement in the field of vision and continued detachment of the retina	38.6%	51.7%
Total cases with slight improvement (partial detachment of retina, slight improvement in visual field and central vision)	15.9%	13.7%
Total of successful results (retina in place full field, improvement in central vision)	45.5%	34.3%
Age of youngest patient operated on	16 yr	5 yr
Age of oldest patient operated on	79 yr	75 yr
Average age	45.5 yr	39.8 yr

* This group included 5 cases of aphakia

† This group included 5 cases of aphakia, in only 1 of which operation was performed

‡ This group included 12 cases of aphakia, with cure in 3, 41.6 per cent of the patients had hyperopia and 27.4 per cent myopia

vitreous is injured simultaneously. Because of these three factors the separations may occur immediately, or several days or weeks later or even after a delay of several months. Further, in all traumatic separations one must not forget that while the trauma may be the real cause of the separation, some of these eyes may have been affected prior to the injury by undiagnosed forms of chorioretinitis or iridocyclitis. One such instance is especially outstanding. A young man had complained of impaired vision several weeks before a fist fight. His attending physician, not an ophthalmologist, had examined the fundus himself and had informed the boy that the lower half of his retina was studded with signet-shaped rings of pigment. Twenty-four hours after a fight in which he was struck in the eye the entire inferior portion of the retina separated, with a massive rent running from 9 o'clock around to 4, gaping so widely in the inferior nasal quadrant that

the choroid was entirely visible in this region. The peripheral lip of this rent was invisible because of a dense crescentic-shaped hemorrhage which extended from the lip of the rent to the ora serrata. (Surgical treatment was unsuccessful in this case.)

The mechanism of traumatic separations is perhaps the most readily understood of all etiologic factors, even though possibilities are based wholly on theory. The eyeball as it rests in the orbit is subject to posterior impact from the bony walls of the orbit, because of the resistance of the orbit to transient flattening of the globe from a blow, because of its position, the majority of traumatisms will be applied from the front and perhaps slightly downward. Repeatedly, retinal separation seems to be precipitated by minor traumatisms of such inconsequential force and so distantly applied that their significance is often lost and not detailed by the patient when his history is taken. Because of the resistant bony orbital wall, retinal and choroidal rupture are not uncommon by contrecoup. In these instances the tear is usually at the posterior pole about the papilla or in the macula, and the choroid alone may be lacerated, the overlying retina remaining intact. The common findings in the macula in cases of *contusio bulbi* and of *commotio retinae* seem to indicate the delicacy of the retina at this point and the effect of countercoup. The temporary deformity or flattening of the eyeball due to a blow probably accounts for the large irregular giant ruptures which occur in the region of the equator. The edges of the laceration are turned inward, i. e., away from the sclera, and with the ophthalmoscope one can see that the enormous force, suddenly applied, plus the transient momentary deformity of the scleral shell, has resulted in an explosive rupture of the choroid and the retina together or singly. Giant traumatic retinal lacerations may be due not to the abrupt infolding but to a sudden stretching from the impact to, and the subsequent deformity of, the scleral shell. While disinsertions are probably due to peripheral cystic degeneration of the retina or to peripheral degenerative chorioretinitis, traumatic tears which appear in the preequatorial zone may simulate disinsertions. They probably are not true disinsertions but actual rents through a portion of the retina which was already thin and atrophic by reason of this peripheral retinal pathologic process. Choroidal and retinal ruptures, which occurred so commonly during the World War as a result of bullet wounds in the neighborhood of the eyeball (without actual perforation of the eyeball), were undoubtedly of this same explosive nature. These showed the ruptures of contrecoup, ruptures through the posterior pole and irregular and often huge ruptures close to the path of the missile through the bony orbit. Perforations of the sclera may result in destruction of the globe, especially if the force of impact has been great or the missile large. Stab wounds, with the immediate withdrawal of the penetrating weapon, and smaller perforating wounds, with probably retained

foreign bodies, cause retinal tears, first, by perforation, second, by the loss of some vitreous, third, by the retraction of the lips of the wounds by contraction, fourth, by the continued gaping of these wounds by the deposition of scar tissue in the vitreous and in the path of the missile, with consequent drag on the retina, and, fifth, because of the hemorrhage connected with the original wound and the slow absorption so common of hemorrhages into the vitreous. The frequency of retinal separation following extraction of magnetic foreign bodies by the posterior route is significant.

It has been a frequent observation that retinal separation follows late after retinitis proliferans. The injuries received during the World War seemed to show on analysis that choroidal rupture, even though of necessity complicated by a lacerated overlying retina, was followed in relatively few instances by retinal separation, either immediate or late. This observation is not consistent with that just mentioned relative to the choroidal and retinal damage connected with extraction of a foreign body by the posterior route. Massive retinal, subretinal and preretinal hemorrhages alone, as seen in pregnancy, Hodgkin's disease and diabetes, do not of themselves cause retinal separation. A tear from a contusion, however, plus a lesser degree of hemorrhage, will do this, and more frequently than when the choroid has also been ruptured by the concussion of the contusion. It seems as if the accompanying choroidal damage protects that patient from a later retinal separation.

MYOPIA

In from 50 to 75 per cent of cases of retinal separation myopia seems to be a major predisposing factor. Racial factors peculiar to an environment may change this, but the probable average is in the neighborhood of 60 per cent of all cases. The mechanism of the retinal separation in cases of myopia, according to Majewski⁶⁶ in a consideration of von Graefe's original opinion, demands that the myopia itself be due to posterior sclerochoroiditis and that the subsequent retinal separation (as described by Arlt, by Leber and by Nordenson) presupposes the development of an inflammatory process in the choroid consequent to the sclerochoroiditis, with a resulting serous exudation. This pathologic subretinal condition, added to an increase in the size of the globe (which can be considered approximately spherical), should be the anatomic basis for separation in cases of myopia. The sequence, however, which exists between the tear and the separation, or inversely, between the separation and the tear, is still unsolved. Certainly separation and choroiditis is an established entity, in fact, Elschnig⁶⁷ in 1931 presented a

66 Majewski, K. W. *Arch. d'opht.* **47** 273, 1930.

67 Elschnig, H. H. *Klin. Monatsbl. f. Augenh.* **86** 595, 1931.

case of primary choroidal detachment of unknown etiology in which there was a humplike, rigid and sharply demarcated mass of the usual fundus color of choroidal detachment and in conjunction with this, a retinal separation which included more than the lower half of the retina. Recovery was uneventful, with vision of 6/8 following subretinal drainage through a scleral window. Anderson stated

as the sclera and the choroid stretch, the normal fine relations between the retinal pigment epithelium and rods and cones are broken down. The vitreous becomes degenerated and the retina atrophic. This eye is now susceptible to toxic influences or circulatory disturbances make for effusion hemorrhage, and to trauma or exertion which can easily cause rents or apertures in the degenerate retina.

Both de Long and Samuels (as expressed at various times in their work on peripheral cystic degeneration of the retina) expressed the belief that while this peripheral cystic degeneration may be peculiar to myopia, it is not the cause, but the result, of the myopia, further, retinal separation is due to this cystic degeneration and not to the elongated scleral shell of myopia or even to the stretching of the retina (if this occurs)—a fine distinction but a necessary one.

INFLAMMATORY CAUSES ⁶⁸

Chorioretinitis, cystic and senile degeneration of the retina, edema of the retina, as seen in association with nephritis, and peripheral chorioretinitis, uveitis and scleritis are all conditions characterized by two types of pathologic change, degeneration and atrophy. It is probable in these instances that the mechanism of retinal separation is similar to that of the separation associated with myopia. Certainly a tear must initiate the condition, and histologic sections have been described wherein patches of chorioretinitis were the exact site of a retinal rent. The central hole in the macula following contusio bulbi and the similar holes which follow actinic retinitis seem to prove this sequence of events conclusively. It is not certain whether tuberculous chorioretinitis and syphilitic retinitis are the cause of retinal separation. Conjectures may be made, but as yet no definite proof is present. Diabetic retinitis, excluding subretinal hemorrhages, if not nephritic in nature does seem to be a cause, but here also in individual cases other nondiabetic factors could be the true underlying reason for the separation.

SYMPTOMATIC SEPARATION

Retinal separation which occurs with sarcoma of the retina and of the choroid and with cysticercosis and hydatid cysts is purely symptomatic and is of interest only in reaching a correct differential diagnosis.

⁶⁸ See "Myopia."

APHAKIA AND RETINAL SEPARATION

The retinal separation following early after operation for cataract is almost a distinct and definite type. Certainly preexisting retinal conditions play a part, for it has been known for years that the myopic eye is prone to retinal separation following the extraction of cataract. Cases of this type are to be considered probably and essentially as instances of separation due to myopia, and operative treatment should be employed the same as for any other detachment not basically traumatic. The elongated scleral shell, the preexisting myopic pathologic process, the probable development of a larger optically empty space behind the contracted vitreous, anterior to the retina (because of the trauma and the structural changes following extraction of cataract), the probability of a fluid vitreous and the possibility of the loss of some of this are the physiologic and structural factors responsible for this form of detachment. Of all factors, these are least commonly associated with a demonstrable retinal tear. For this reason it is likely, though not proved, that many of these tears are the result of uncovered retinal disinsertions. Further, they occur shortly after the extraction of cataract and seldom if ever late, in fact, a late development would place them in the second class of postoperative (cataract) separations.

This second group of postoperative separations include those instances which follow late after the extraction of cataract and after discissions, sometimes single but more often multiple, they are occasionally seen after linear extractions for traumatic cataracts and no doubt play a part in those cases in which the patient has a stormy postoperative convalescence with a more or less long drawn out iridocyclitis.

The etiologic factors in the first group of postoperative separations are probably connected with Anderson's theories of distention and perhaps also of depression. This second group, however, is probably, with the separations due to trauma, the best example for demonstrating the theory of attraction. The operation which antedated this second group has resulted in a demonstrable contraction of the vitreous, with cicatrices within the vitreous body and chamber. In 3 instances a postoperative separation has been correctly prophesied following earlier operation of this type simply because one could see with the slit lamp a contracting vitreous with tension lines. These arose in the neighborhood of the temporal portion of the ora serrata above and diverged downward and backward, like the veins of a palmleaf fan. In 2 instances rents were found in the retina, but only after the separation had been found. In 1 instance the separation occurred during an interval of five days and in the second, within a period of thirty-six days. In neither of these was operation successful. The third instance was discovered suddenly by the patient, though it probably was present for from a week to ten days.

The patient had had a linear extraction for traumatic cataract, a disinsertion was found, and operation was successful in completely reattaching the retina

These separations are considered hopeless by many operators. This is an unduly pessimistic attitude. Etiologically, they differ so little from any of the other types of separation that all similar rules for surgical treatment apply.

CONCLUSIONS

1 The presence of a retinal tear or of a disinsertion is necessary to the development of retinal separation.

2 Histologically, the theories of distention, of depression, of attraction and of exudation, play equally important roles in the causation of retinal separation, though under varying circumstances and peculiar to individual cases.

3 Myopia itself may not be the etiologic cause but the retinal changes (retinosis), degenerative and trophic, which result from myopia.

4 The retinal separation in aphakic eyes is caused by the same factors which are responsible for retinal separation in nonaphakic eyes.

5 The retinal separation of directly applied traumatisms is probably to be explained by the theory of distention plus the damage to the ocular tissues from the impact, the deformity-producing blow (affecting the shape of the eyeball) to be considered as distinct from the damage to the tissues themselves from the impact. The laws governing the transmission of energy through a complex fluid body, as is the eyeball and its contents, apply as rigidly here as with any less complex mass of fluid or semifluid.

1930 Chestnut Street

News and Notes

SOCIETY NEWS

New York Academy of Medicine—At the November meeting of the Section of Ophthalmology of the New York Academy of Medicine, held November 21, an instructional hour on perimetry of chiasmal lesions was conducted by Dr Ralph I Lloyd. Slit lamp demonstrations were made by Drs Milton Berliner, Wendell L Hughes, Giralamo Bonaccolto and Gordon M Bruce.

Dr Mark Schoenberg reported a case of recurrence of ocular hypertension eighteen years after an Elliot trephining.

Dr Phillips Thygeson gave a report on the use of sulfanilamide in the treatment of inclusion conjunctivitis.

A paper on the use of the grenz ray in the practice of ophthalmology was read by Dr Raymond Pfeiffer and discussed by Dr Gustave Bucky.

The paper of the evening was presented by Dr Walter Ivan Lillie, of Philadelphia, on the interpretation of fundal changes associated with arterial hypertension.

Dr James W White is chairman of the section and Dr Rudolph Aebli, secretary.

Swiss Ophthalmological Society—At the annual meeting of the Swiss Ophthalmological Society in Zurich, Professor Vogt presented the society with 50,000 francs to celebrate the thirtieth anniversary of the society and in memory of his son, Alfred Vogt, who died ten years ago. The fund is to be known as the Alfred Vogt Foundation, and the income is to be used every year in the form of a prize for the best scientific contribution by a member of the society and to facilitate scientific publications.

Ophthalmological Society of the United Kingdom—The annual congress of the Ophthalmological Society of the United Kingdom will be held at the headquarters of the Royal Society of Medicine, Wimpole Street, London, W 1, on April 20-22, 1939. The subject for discussion will be "The Problems of Refraction." The discussion will be opened by Mr Affleck Greeves, Mr Charles Goulden and Mr A Harold Levy.

The Bowman Lecture will be delivered by Professor Weve, his subject being "Diathemy in Ophthalmic Practice."

The International Organization Against Trachoma and the International Association for the Prevention of Blindness will hold their annual meetings on Wednesday, April 19, 1939, at the same venue as that of the Ophthalmological Society of the United Kingdom. These meetings will be accessible to members of the congress.

The annual dinner of the society will be held on Thursday, April 20. The president of the society is T Harrison Butler Esq, M A, D M.

National Society for Prevention of Blindness—The annual meeting of the National Society for the Prevention of Blindness was held on

Thursday, December 1, at the Russell Sage Foundation Building in New York. The following addresses were given: "Thirty Years in Saving Sight," by Lewis H. Carris, and "Prevention of Blindness from the Ophthalmologist's Point of View," by Dr. Ellice M. Alger. The presentation of the Leslie Dana Gold Medal to Dr. Ellice M. Alger was made by Dr. John N. Evans.

First de Schweinitz Lecture—Dr. Edward Jackson, of Denver, delivered the first de Schweinitz lecture of the Section on Ophthalmology of the College of Physicians of Philadelphia on November 17 on "The Development of Ophthalmology in One Lifetime." The lecture was established in honor of Dr. George E. de Schweinitz, who died on August 22.

General News

Archiv fur Augenheilkunde—The *Archiv fur Augenheilkunde*, at the conclusion of its one hundred and tenth volume in November 1937, was combined with the von Graefe's *Archiv fur Ophthalmologie*. The new journal carries on its frontispiece the following title: "Albrecht von Graefe's Archiv fur Ophthalmologie, vereinigt mit Archiv fur Augenheilkunde. Redigiert von A. Wagenmann, Heidelberg, W. Lohlein, Berlin, and E. Hertel, Leipzig."

This means the termination of the *Archiv fur Augenheilkunde* as an independent journal. This journal was founded in 1869 by Herman Knapp (New York) and Moos (Heidelberg) as the *Archives of Ophthalmology and Otology* and as the *Archiv fur Augenheilkunde und Ohrenheilkunde*. It appeared in English and German editions up to 1911. In 1879 the journal was divided into two journals, the *Archives of Ophthalmology* and the *Archives of Otology*, and the *Archiv fur Augenheilkunde* and the *Zeitschrift fur Ohrenheilkunde*. The ophthalmic journal was edited by Herman Knapp and Hirschberg (Berlin). In 1882 Schweigger (Berlin) took Hirschberg's place as the German editor, in 1905 Hess (Munich) became editor, he held this position until his death in 1925, when Wessely (Munich) took his place and was later joined by Hertel (Leipzig). An important part of the *Archiv fur Augenheilkunde* was the review of the literature. This was abandoned in 1925, when the *Zentralblatt fur die gesamte Ophthalmologie* was founded.

The *Archives of Ophthalmology* was continued by Arnold Knapp and W. A. Holden from 1912 until 1928, when the *Archives* was taken over by the American Medical Association, with the following editorial board: Arnold Knapp, chief editor, G. S. Derby, W. Zentmayer, S. R. Gifford, F. H. Adler and J. H. Waite.

New Editors for Zeitschrift fur Augenheilkunde—A change has taken place in the editorship of the *Zeitschrift fur Augenheilkunde*, which has been conducted, with such well known success by G. Behr (Hamburg) and J. Meller (Vienna). The names of A. Bruckner (Basel) and H. Weve (Utrecht) have been added to the list of editors, and it is reported that the latter two will be the chief editors of the journal beginning with January 1939, when certain changes in the publication are being considered. The high standing of the *Zeitschrift*

für Augenheilkunde has been due to the indefatigable work of J. Meller, and it is to be hoped that for the future success of the journal his cooperation will not be lost.

New Ophthalmic Journal.—Welcome is extended to a new bimonthly ophthalmic journal, *Archivos brasileiros de oftalmologia*. The articles will appear in Portuguese, French, Spanish and Italian, with a résumé in Portuguese. The editors are Drs. W. Bedford Mattos, S. Paula Santos and Duval Prado, and the address of the publication office is Caixa Postal 4086, São Paulo, Brazil.

Acta Ophthalmologica Oriental.—Dr. Aryeh Feigenbaum writes that he and his whole staff of collaborators have withdrawn from the editorship of the *Folia ophthalmologica orientalia*, founded and edited by him for a number of years, and that he has founded a new journal, the *Acta ophthalmologica orientalia*, of which the first issue has just appeared.

UNIVERSITY NEWS

Course in Basic Ophthalmology.—A new course in basic ophthalmology began November 1 at Wayne University College of Medicine, Detroit, to continue each Tuesday and Friday throughout the year. Designed for residents and those interested in increasing their basic knowledge in ophthalmology, the first course is on histology, comparative and human, while the second will be on physiologic optics, physiology of the eye, and gross anatomic sections of the head and neck. There is no fee for the courses. Dr. Parker Heath is professor and head of the department of ophthalmology.

Postgraduate Course in Biomicroscopy.—A one week course for licensed physicians specializing in diseases of the eye will be given at Wayne University College of Medicine, Detroit, December 12 to 17 inclusive, under the direction of Dr. Parker Heath, professor of ophthalmology. Gullstrand's principles and their modifications will be discussed. Practical training in the use of various instruments for slit lamp microscopy will be emphasized. The class will be limited to 14. The registration fee is \$50. For details, write the Dean, Wayne University College of Medicine, 1512 St. Antoine Street, Detroit.

Prize for Ophthalmic Work.—The University of Buffalo awards annually a gold medal for a work on an ophthalmologic subject. Recent awards went to Dr. J. G. Bellows, of Chicago, Dr. Joseph Globus, of New York, and Dr. J. N. Evans, of Brooklyn. For details, write Dr. H. W. Cowper, 543 Franklin Street, Buffalo.

Obituaries

GEORGE EDMUND DE SCHWEINITZ, M D

1858-1938

Dr George Edmund de Schweinitz died at his home in Philadelphia on Aug 22, 1938, at the age of 79 Dr de Schweinitz held the admiration and respect of all who knew him He was tall and erect, with kindly blue eyes, and he possessed a voice that, while mild, had an agreeable and penetrating power that was unusual His charm and powerful personality made him conspicuous in any group of men, and he well deserved the title so often given him—Dean of American Ophthalmology

His ancestry goes back to Moravian nobility He was born on Oct 26, 1858, in Philadelphia, the son of Bishop Edmund de Schweinitz and Lydia de Schweinitz He received the degree of Bachelor of Arts in 1876 and the degree of Master of Arts in 1878 from Moravian College, Bethlehem, Pa, and the degree of Doctor of Medicine from the University of Pennsylvania in 1881 He was first honor man in his class After being graduated, he was resident physician at the Children's Hospital in 1881 and at the University Hospital from 1881 to 1883 From 1882 to 1887 he was quiz master on therapeutics at the Medical Institute, and from October 1883 until June 1888, prosector of anatomy at the University of Pennsylvania in the service of the late Professor Leidy For a time he devoted himself to general practice, but soon, under the direction of the late Dr William F Norris, he began the study of ophthalmology In 1885 he was elected ophthalmic surgeon to the Children's Hospital and in 1886, ophthalmologist to the Orthopedic Hospital and Infirmary for Nervous Diseases, where he was associated with the late Dr Weir Mitchell He was appointed ophthalmic surgeon to the Philadelphia General Hospital in 1887 and professor of ophthalmology at the Polyclinic and College for Graduates in 1891 He resigned from the latter position in 1892, when he was elected clinical professor in ophthalmology at the Jefferson Medical College In 1888 he was elected one of the editors of the *University Medical Magazine*, afterward published as the *University of Pennsylvania Medical Bulletin* While editor of the *University Medical Magazine*, he served in this capacity first with the late Dr Hobart A Hare and later with the late Dr Edward Martin He was appointed professor of ophthalmology at the Jefferson Medical College in 1896, which position he held until 1902, when he was made professor of ophthalmology at the University of Pennsylvania

In this position he served with distinction until 1924, when he resigned. He later became a member of the board of trustees of the university.

He was a member of the Pathological Society of Philadelphia, the Philadelphia County Medical Society, the Philadelphia Neurological Society, the College of Physicians, of which institution he was president from 1910 to 1913, the American Ophthalmological Society, of which he was president in 1916, and the American Medical Association, of which he was president in 1922. He was vice president of the Pennsylvania Institute for Instruction of the Blind at Overbrook and a member of the Academy of Natural Science, the American Philosophical Society, the Ophthalmological Society of the United Kingdom, the



GEORGE EDMUND *de* SCHWEINITZ, M.D.
1858-1938

Société française d'ophtalmologie and the Société belge d'ophtalmologie, he was an honorary member of the Royal Society of Medicine of London and of the Hungarian and Egyptian ophthalmologic societies.

As one of the outstanding ophthalmologists in the world, Dr. de Schweinitz was honored at home and abroad. He was the recipient of the Alvaenga Prize in 1894, awarded by the College of Physicians of Philadelphia, for his essay on toxic amblyopia. He received the bronze plaque of the Société française d'ophtalmologie in 1923, the title of his address being "Ocular Manifestations of Focal Infections." The Howe Medal in Ophthalmology was awarded to him in 1927 and the Huguenot Cross in 1928. He gave the Bowman Lecture before the Ophthal-

mological Society of the United Kingdom in 1923, the title of his address being "Concerning Certain Ocular Aspects of Pituitary Body Disorders, Mainly Exclusive of the Usual Central and Peripheral Hemianopic Field Defects" He was awarded the Leshe Dana Medal for meritorious work among the blind, which was presented at St. Louis in 1930, when he delivered a notable address on "The Heritage of Sight, Its Conservation" The medal bore the inscription of "wise, learned, patriotic, teacher and guide" The University of Pennsylvania conferred on Dr. de Schweinitz the honorary degree of Doctor of Laws in 1914, the University of Michigan conferred the degree of Doctor of Science in 1922, and Harvard University conferred a similar degree in 1927

Dr. de Schweinitz belonged to the Philadelphia Club, the University Club of Philadelphia and the University of Pennsylvania Club of New York

His contributions to medicine were varied and epoch making He was the author of many textbooks, brochures, treatises and scientific papers and did extensive research on the eye He was the author of "The American Textbook on Diseases of the Eye, Ear, Nose and Throat," in collaboration with the late Dr. B. A. Randall, and of a standard work on "Toxic Amblyopias" His book entitled "Diseases of the Eye" went through ten editions and has been the outstanding American textbook for many years At one time he was American editor of Haab's "Ophthalmoscopy and External Diseases of the Eye and Operative Ophthalmology" He wrote several communications in association with the late Dr. Thomas B. Holloway, one of the most important of which was on "Pulsating Exophthalmos," and with Dr. Edward Jackson he edited the "Ophthalmic Year Book" from 1905 until 1909

When the United States entered the World War in 1917, Dr. de Schweinitz was made a member of the Council of National Defense and later entered the medical corps of the army as major and did meritorious work both in this country and abroad After the war he joined the Medical Officers' Reserve Corps and became a member of the editorial board of "The Medical Department of the United States Army in the World War" At the time of his death he held the rank of brigadier general in the Medical Officers' Reserve Corps

He was president of the International Congress of Ophthalmology held in Washington in 1922, and at that time was designated as one of two to represent this country on an international committee, the object of which was to reorganize the International Congress of Ophthalmology He took a prominent part in the deliberations of this committee and was later made a member of the council

He enjoyed a large and influential private practice in addition to the usual opportunities for clinical and pathologic research in general and special hospitals with which he was connected As a clinician, he was painstaking, thorough and skilful and never lost sight of the fact that

the eye and its diseases were closely allied to general diseases and diseases of the central nervous system. He displayed sound surgical judgment as an operator and did his work with meticulous care, always having the best interest of his patients at heart. He was an indefatigable worker and a diligent student and found little time for relaxation. He never married but devoted himself assiduously to his chosen profession. As a consultant, he held a most enviable reputation. His reports were meticulous, and his attitude toward the members of the medical profession was ideal. He was essentially the doctors' consultant. He was in great demand as a public speaker, and his after-dinner speeches were prepared with the same care that he gave his scientific addresses. He was fond of walking but never participated in any of the popular sports. His principal diversion was reading, and he liked a good detective story.

The Spartan type of character that was his was well illustrated in the manner in which he met his attack of glaucoma. Quite by accident he discovered that both his pupils were dilated. Investigation revealed that he had early simple glaucoma in both eyes. In spite of treatment the tension remained high, and he decided on an early operation. The trephine operation was performed on both eyes, and an excellent result was obtained. His answer to an inquiry concerning the effect of the disease on his vision was very descriptive. He said "I can best describe my seeing ability by stating that where I previously took a glance I now have to take a look." His central vision remained nearly normal until the time of his death.

All who were privileged to know Dr. de Schweinitz had a real affection for him. His loss will be keenly felt by a large circle of acquaintances, while to his many friends his death leaves a void as teacher, author, clinician, surgeon, administrator and friend that will be impossible to fill. Sincere sympathy is extended to the remaining relatives and friends in their irreparable loss.

WALTER R. PARKER, M.D.

For the past fifty years George de Schweinitz was the most widely known and the most erudite of American ophthalmologists.

It is not necessary for me to catalogue his numerous services to his country or to list the many honors heaped on him by the societies and universities of his country and of foreign countries, because some of these are to be found in "Who's Who," but it may be of interest for me to recall an occurrence characteristic of the man, drawn from his and my intimacy, early established and steadily maintained for half a century. We had by appointment an annual meeting, either at his home, presided over by that remarkable woman his mother, or at his club. The purpose of the colloquy that followed was simply, *à la* Callimachus, "to tire the sun with talking and send him down the sky."

To me these informal trysts were inspiring occasions I feel that I owe to them much of whatever use I have been in the world, moreover, they permitted me to commune with the real de Schweinitz, who was not only a prince charming with a vivid personality but a Gamaliel at whose feet I sat in wonder and admiration My friend that has gone was in the best sense of that term a great man whose memories "death, who taketh all away, *them* he cannot take"

CASEY WOOD, M D

Rome, Italy

A mere catalogue of de Schweinitz's attainments gives an inadequate idea of his personality and of his outstanding position, not only in the history of ophthalmology but in the history of medicine in the United States He was both a great surgeon and a great physician, and it was eminently apt that he should have held the presidency of the Philadelphia College of Physicians His life was unremittingly devoted to ophthalmology and to the encouragement of medicine in the famous and old established University of Pennsylvania An object which he had much at heart was the enrichment of the magnificent medical library in Philadelphia, and much of the time of his annual visits to England was devoted to seeking out medical incunabula in London and elsewhere De Schweinitz belonged to the type of American one associates with New England but who also flourishes in Pennsylvania—that erudite and highly cultured type, whose distrust of humbug and anything cheap engenders a reserve which can be as impenetrable as armor plate Only those who succeeded in overcoming this reserve can appreciate the sterling worth which it masked To his accepted friends, de Schweinitz was a delightful companion and a staunch comrade

As an Englishman who was proud of his friendship for many years, it is fitting that I should express the profound admiration all ophthalmologists on this side of the Atlantic—British and Continental—had for de Schweinitz At the meetings of the Ophthalmological Society of the United Kingdom he was an honored guest, until in due time he became Bowman Lecturer and one of the few honorary members of the society By his death the world has lost a great ophthalmologist and a great gentleman

J HERBERT PARSONS

London, England

As a student, de Schweinitz had been well prepared by heredity and teaching His father, the Moravian bishop, had been able to give him a most excellent intellectual start, but he was unable to contribute largely to the financial side of his medical education There was a legacy of

\$500 left by his grandmother, but this was not sufficient, so to add to it he taught school for two years at Nazareth Hall, thus largely or perhaps altogether paying his own way. This, too, was good mental exercise, preparing him for his success as a teacher in later years.

While teaching school he became associated with a physician in Nazareth and got a good start in practical medicine. His popularity in his class at the university is shown by his election as class president. He made friends easily. He had no fads. In those days poker was a favorite indoor pastime. He was not at all fond of the game and was but an indifferent player. He was a good speaker and conversationalist, not given to florid oratory but employing careful, well chosen, convincing words. His writing was of the same nature, carefully thought out beforehand. He rarely had to alter a manuscript.

On his graduation he was the second class of the three year term. He was appointed intern at the Children's Hospital, largely through the friendship of Dr. John Ashhurst Jr. This internship ran for a year, but it was the usual custom to divide it into two terms. At the end of six months I followed him there, and he went to the University Hospital, this appointment being obtained as the result of a competitive examination.

After his year there he had a room with me in the house where I had started practice. He had not settled definitely what line to pursue, probably general practice, when an offer for an assistantship came from Dr. Norris. After studying over this offer for some time, he finally accepted it as a practical way of starting. Dr. Norris had watched him as intern and had recognized his capabilities.

De Schweinitz had no predilection toward ophthalmology, it was this offer that influenced him. Without it he would probably have drifted into surgery, for his preceptor, Dr. Wharton, was a surgeon, and he afterward held the position of surgical registrar at the University Hospital and was professor of anatomy under Dr. Leidy. I think that his greatest objection to general practice was that he would have to do obstetric work. His first office was at 1330 Spruce Street, and here his luck held good in that among his fellow boarders were several men, slightly older than he, who had most excellent social contacts, and they, after recognizing his worth, introduced him to their friends, thus helping to start his social career which was such a success. At first he slept on the couch in his office, using the sitting room of the house as his waiting room. In the beginning he looked after all the general practice he could get in addition to his ophthalmic work. His income from practice was at first small. He added to it by sitting up at night with patients of older practitioners. This was before the days of trained nurses. He also established a successful quiz class on therapeutics among the students. To illustrate how close the wolf often was to his door, here is a story he used to tell.

He was walking home, since he did not wish to pay streetcar fare, when he met Dr Agnew, who asked him to come to the hospital and administer ether to a patient. He received \$10 for this and so was able to pay his office rent, about which he had been in some doubt. He had been in practice about six years before he could afford a secretary. He then employed one, at first part time and afterward constantly. She remained with him until his death and became quite a factor in the smooth running of his practice. All of his colleagues know her as Miss Mary. He changed his office from 1330 Spruce Street to 1401 Locust Street to establish a home for his stepmother and stepsister, his father having died.

Looking back over his life, one can see that his success was due to his native ability plus his unswerving devotion to his profession.

W E HUGHES, M D

LADISLAS DE BLASKOVICS
1869-1938

After receiving his medical degree in 1893 in Budapest, Hungary, Blaskovics began his ophthalmic training as assistant to de Grosz in the First University Eye Clinic, where he remained for ten years. He then acted as director of the Hungarian State Eye Hospital for twenty years. In 1928, on the death of von Hoor, he was appointed professor of ophthalmology and director of the Second University Eye Clinic in Budapest. In 1936, on the retirement of de Grosz, the two university clinics were combined, and Blaskovics acted as director, though his health was far from good. He died on Oct 17, 1938, of pneumonia.

His talents lay in the surgical field. It is stated that even in his student days his dexterity in operations on animals was remarkable. The wealth of material in the University Hospital in Budapest gave him the opportunity of developing many new operative procedures and established his reputation as one of the great ophthalmic operators in the world. He was particularly interested in plastic surgery, in which field he was a pioneer and devised many new operative procedures. He is perhaps best known for his operation for ptosis. Although he was first opposed to the intracapsular operation for cataract, he later became an enthusiastic convert and suggested a special capsule forceps for this procedure. It was a revelation and a pleasure to watch him at work in the operating room. Always quiet and cool, working with practically no assistance and without fuss and helping himself to the instruments which were spread out on a side table, he conducted his operations on the soundest surgical principles and without trauma. He also made contributions to other fields of ophthalmology and devised a new unit for measuring vision, oxyoptry, in which a 1 degree angle replaced that of 1 minute. He wrote a text on operations on the eyes in Hungarian, which appeared in several editions. An enlarged edition of this book in German, written in conjunction with A. Kreiker, appeared only a few months ago.

ARNOLD KNAPP

Abstracts from Current Literature

EDITED BY DR WILLIAM ZENTMAYER

Biochemistry

CHEMICAL AND HISTOCHEMICAL INVESTIGATION OF UREA IN THE LENS AND OCULAR MEMBRANES I BORSOTTI, *Ann di ottal e clin ocul* 66: 32 (Jan) 1938

The xanthidrol test of Fosse as modified by Vercellana was employed, crystals of xanthic urea being formed in the section of the tissue under investigation. When the whole eye of the rabbit was fixed with xanthidrol, crystals were found in the vitreous, cornea, sclera and retina, especially in the internal layers. When the cornea alone was removed before the death of the animal, however, and fixed in the same way, no urea crystals were found. This indicated that the crystals found in the cornea of eyes fixed after death are due to rapid post-mortem diffusion from the aqueous, which does not take place during life while the cornea is exercising its function of selective permeability. The same is apparently true of crystals found in the sclera and retina, since when the aqueous and vitreous were rapidly removed before death, or when the fixative was injected into the globe during life few crystals were found in the sclera or retina.

These observations suggested that the findings of Schmorl on the urea content of the crystalline lens might be in part explained by post-mortem diffusion. To control this factor, the author estimated urea quantitatively in the lenses of cattle, sheep and rabbits extracted immediately after enucleation of the eye and twelve hours after enucleation. The lenses extracted immediately showed a urea content of 16.1, 18.1 and 13.9 mg per hundred cubic centimeters for beef, sheep and rabbit lenses, respectively. When left in the eye twelve hours before extraction of the lens, the figures were 22.9, 20.4 and 21.8 mg per hundred cubic centimeters. The definite increase in urea in the lens must be attributed to increased permeability of the lens capsule occurring after death. The author believes that his work offers additional proof of the selective permeability of certain living tissues, such as the cornea and lens capsule.

S R GIFFORD

Congenital Anomalies

CONGENITAL ANOPHTHALMOS VEILING AN OPHTHALMOTERATOID REPORT OF A CASE E SCHRECK, *Klin Monatsbl f Augenh* 100: 74 (Jan) 1938

Schreck reports in detail the clinical, operative and histologic data with reference to a girl aged 17 who was examined at the ophthalmic clinic of Heidelberg University for absence of her left eye at the age of 10 days. At that time congenital anophthalmos with a vicarious orbital tumor was found. A plastic operation, performed when the girl was 17 years old, revealed a tumor, the size of a walnut, which was con-

nected with a coloboma of the lower lid. The tumor presented the pathologic picture of an early malformation of the ocular "anlage" and a teratoid formation.

The relation between congenital anophthalmos and teratoma is discussed and evaluated, the result of this evaluation was a separation of congenital anophthalmos into genuine and apparent. The diagnosis of the genuine type should be made only after exact microscopic examination of the orbital contents reveals the total absence of ocular rudiments. All other cases should be recorded under the group of apparent anophthalmos, which type forms a transition to microphthalmos, in which remnants of the globe may be recognized microscopically.

In Schreck's case there was complete absence of ocular rudiments, but pathologicoanatomic proof was furnished of the presence of an undeveloped and malformed anlage of the sclera, choroid and retina. Glia cells were found in abundance, and their provenience was doubtlessly traced to retrograding retinal tissue. In addition, a teratoid tumor was found in the orbit which consisted of underdeveloped derivatives of two embryonic layers. This tumor did not belong to the fourth group of teratomas of Mizno, as it did not contain any tissues which might not occur in a malformed ocular anlage. Other considerations, too, pointed to a tumor derived from an ocular malformation. Schreck thinks that in his case an early disturbance of the ocular anlage produced a severe ocular malformation, associated with a teratoid tumor in the orbit, which belongs to the group of the apparent anophthalmos.

K. L. STOLL

Cornea and Sclera

THE TREATMENT OF INFECTED ABRASIONS OF THE CORNEA. C. F. KUTSCHER, Pennsylvania. *M. J.* 41:979 (Aug.) 1938.

Kutscher calls attention to the value of sodium hypochlorite in the management of infected abrasions of the cornea. The chemical is employed in a strength of 1:5,000 and is instilled every second hour day and night for twenty-four hours. After twenty-four doses, the instillations are made every fourth or sixth hour. If the infection is not checked, delimiting keratotomy is done.

W. ZENTMAYER

KRUKENBERG'S PIGMENTED SPINDLE AND THE PIGMENT RING IN THE CORNEA. REPORT OF CASES. SOMMER, *Klin. Monatsbl. f. Augenheilk.* 99:468 (Oct.) 1937.

Sommer reports 3 cases of disseminated pigmentation on the endothelium of the cornea associated with other symptoms. The first patient, a woman aged 31, presented bilateral Krukenberg spindles in the presence of an otherwise perfectly normal cornea and normal vision. The second patient, a woman aged 61, had similar bilateral spindles, which, however, were less distinct than those of the first patient. No changes were observed after the extraction of cataracts from the eyes, which were myopic. The patient did not have diabetes. In the third case the spindles were associated with chronic glaucoma. They became less conspicuous after bilateral trephining after the manner of Elliot. The atrophic retina in these slightly hyperopic eyes showed numerous scattered pigment cells. Tension was normal after the operations but

the cupping of the disks persisted. The cause of the spindles is discussed by the author, who arrives at no definite conclusions.

K L STOLL

Experimental Pathology

AN EXPERIMENTAL STUDY OF CHOKED DISC IN THE RAT W A JEFFERS, J Q GRIFFITH JR, W E FRY and A G FEWELL, *Am J Ophth* 20: 881 (Sept) 1937

On the basis of experiments on rats, the authors give the following summary

"1 Rats developing increased intracranial pressure and vascular hypertension following the intracisternal injection of kaolin were found to develop no ocular changes upon ophthalmoscopic or histologic examination

"2 Ten rats developed a condition simulating papilledema upon the implantation of cerebellar tumors, as confirmed by the same examinations

"3 Eight rats that were known to have increased intracranial pressure after the intracisternal injection of kaolin failed to develop signs of papilledema in the presence of large cerebellar tumors

"4 The evidence presented is interpreted as support for the view that papilledema is due to an increased flow of fluid from the cerebrospinal space to the eye, and that it is not due to a block in the flow from the eye toward the cerebrospinal space

"5 Our findings are discussed with reference to hypotheses concerning the nature of papilledema "

W S REESE

HISTOLOGIC STUDY IN EXPERIMENTAL WOUNDS AND SUTURES OF THE OPTIC NERVE C TITA, *Ann d'ottal e clin ocul* 66: 51 (Jan) 1938

The author sectioned one optic nerve of each of 60 rabbits with care to avoid the central vessels. The fragments were held in close apposition by a suture and by sparing the posterior portions of the sheath. Sections were made at various intervals from three to thirty days later. Regeneration of fibers could be observed by the seventh day and was more extensive by the fourteenth and twenty-first days. By the twenty-first day, a few fibers could be traced through the cicatricial zone. By the thirtieth day, however, secondary degeneration was already evident in the regenerated fibers, which were obviously being compressed by condensation of the scar tissue. Studies of the later processes have been made by Rossi, who showed further degeneration of the new fibers and never saw fibers traverse the scar. The author concludes that regeneration of fibers in a functional sense after grafting of the eye is entirely impossible.

S R GIFFORD

General

ANNIVERSARY NUMBER *Ann d'ocul* 175: 1 (Jan) 1938

In January 1938 the *Annales d'oculistique* reached the hundredth anniversary of its foundation. In January 1838 Florent Cunier issued

the first number, and in the present issue is found a complete table of the articles published in the first volume with an exhibit of the title page. There are also photographs of Cunier, Warlomont, Desmaires, Dor, Panas, DeWecker, Gayet, Javal, Badal, Abadie, Coppez, Nuel, Parinaud, Landolt, Lagrange, Truc and Van Duyse. There are also articles on "A Hundred Years of Franco-Belgian Ophthalmology" by Rochon-Duvigneaud, "Paralysis of Associated Movements of the Eyes," published by Parinaud in 1883, "Iridectomy and Sclerectomy Combined in the Treatment of Chronic Glaucoma. New Procedure for the Establishment of a Filtering Cicatrix," published by Lagrange in 1906, "Vision" by Nuel, published in 1904, and an unpublished article by Morax on "Gonococcus Infection."

The editorial board of the present *Annales* consists of Magitot, chief editor, Rochon-Duvigneaud, Baillart, Hartmann, Leplat, Dupuy-Dutemps, Redslob, Jeandelize and Hambresin.

S H McKEE

General Diseases

TULAREMIA FROM THE CLINICAL, AND ESPECIALLY FROM THE OPHTHALMOLOGIC, STANDPOINT. A JESS, *Klin Monatsbl f Augenh* 99: 577 (Nov) 1937

The advance of tularemia in Germany prompted Jess to report on the symptoms of this disease in a short, but precise article. The oculist is first consulted in many cases, as the ocular symptoms frequently prevail. Trachoma and tuberculous conjunctivitis will have to be considered from the point of view of the differential diagnosis. The early general symptoms, such as fever, malaise, lack of appetite and involvement of the preauricular and submandibular glands, are characteristic. The glands may remain swollen for weeks after the ulcers of the conjunctiva disappear. This oculoglandular form resembles Parinaud's conjunctivitis, the origin of which has been traced to animal infection, as has been tularemia. Three other forms of tularemia occur: the ulceroglandular form, which begins with ulcers in the epidermis and swelling of the glands; the glandular form, in which infection of the glands presents first; and the typhoid, or the septic form. Conjunctivitis infectiosa necroticans, described by Pascheff, appears to be related to tularemia. The death rate from tularemia is 4 per cent according to Jess. The point of entrance is the eye, and in many cases the disease is apparently due to eating insufficiently cooked meat of infected animals. Cases of tularemia have been reported in the United States, Sweden, Norway, Japan and Russia and recently in the Balkans, Moravia, Austria and Bavaria. A total of 396 cases of human tularemia were recorded in Bohemia up to the end of March 1937. Aside from rabbits and other rodents, pheasants, partridges, foxes, dogs, cats, hogs and sheep are subject to tularemia. Human beings may become infected by furs. The exact anamnesis, inoculation of animals, an agglutination test and a test for cutaneous allergy are essential in arriving at a correct diagnosis. The agglutination test is not positive prior to the second week. Infection from man to man has not been observed. The therapy consists of the use of antiserum and local ocular treatment. Permanent damage to the eyes has not been reported.

K L STOLL

SPECIFIC DIAGNOSIS OF TUBERCULOUS DISEASES OF THE EYES REPORT
OF CASES W ROHRSCHEIDER, Klin Monatsbl f Augenh 99.
682 (Nov) 1937

In cases of miliary and other forms of tuberculosis of the uvea the diagnosis of ocular tuberculosis may be readily arrived at by the observation of the clinical picture. In many ocular diseases, however, which are ascribed to tuberculosis, diagnostic tests, such as roentgenograms of the lungs, serologic reactions and intracutaneous and subcutaneous tests, are not reliable. The evaluation of focal reactions may be still more difficult. This evaluation seems to be facilitated by observation of the intraocular pressure, which drops after injections of tuberculin used for diagnostic purposes. Rohrschneider refers to the research in point done by Samojloff and Mexina, who, he contends, have not considered spontaneous undulations of the intraocular pressure in inflamed eyes. Such undulations occurred in the third case described. Rohrschneider shows tables with data on the intraocular tension and temperature for 20 patients. He concludes that undulations of the intraocular tension after injections of tuberculin cannot be depended on for the diagnosis of a tuberculous focal reaction. It is necessary, in his opinion, to observe the intraocular tension for a few days prior to the diagnostic injection of tuberculin. The tension barely changed or did not change at all in some cases presenting clinical evidence of an ocular focal reaction of tuberculosis, his second case furnishes proof of this observation. Therefore, undulations of the intraocular tension after tuberculin tests are of limited use in the diagnosis of tuberculosis.

K L STOLL

Glaucoma

THE PHOTOCROMATIC INTERVAL IN GLAUCOMA AND CAVERNOUS
ATROPHY R PICKARD, Brit J Ophth 22. 391 (July) 1938

The author undertook the examination of the photochromatic interval in cases of glaucoma and cavernous atrophy. The photochromatic interval is the condition in which a color produces stimulus of light but not of color. The test was applied by moving the stimulus from the periphery of the field to fixation. The author concludes that it may fairly be said that if the red field is diminished and the photochromatic interval is under 5 degrees, there is a five to one chance that the condition is glaucoma. Conversely, if the red is reduced and the photochromatic interval is over 5 degrees, the chances are one to four that it is cavernous atrophy.

The article is somewhat technical and should be read in the original.

W ZENTMAYER

CONTROL OF THE INTRAOCULAR PRESSURE IN INFANTILE GLAUCOMA
W KAPUSCIŃSKI, Arch f Ophth 138. 673 (May) 1938

The object of the studies on which this paper is based was to determine in which forms or in what phases of glaucoma atropine is not contraindicated or even indicated. First, the author confirmed the well

known fact that mydiatics or miotics exert no appreciable influence on the intraocular tension of normal eyes of children. In 3 cases of typical hydrophthalmos (children from 2 to 6 years of age) the tension with the use of atropine was certainly no higher than that when no medication was used or when miotics were employed. In 1 case of hydrophthalmos combined with nevus flammeus of the face pilocarpine had practically no effect, whereas the first dose of atropine after a period during which miotics were used several times produced an acute though transient drop in pressure. Still more striking was the pressure-lowering effect of atropine in a case of glaucoma simplex (a girl aged 20) in which cyclodialysis of each eye was necessary five times in order to bring about lasting hypotony (tension of each eye, between 8 and 13 mm of mercury). About a year after the last cyclodialysis, an acute attack of glaucoma suddenly developed in one eye with a tension of 58 mm of mercury and a deep anterior chamber. Pilocarpine narrowed the pupil without any effect on the tension. After one dose of atropine the pupil dilated and the tension dropped at first to 26 and, a few hours later, to 6 mm of mercury. During a two months' stay in the hospital, the patient had six more acute attacks, each of which was promptly controlled by the use of atropine. A striking subjective symptom which preceded each attack was a sudden lowering of the refraction caused by deepening of the anterior chamber.

P C KRONFELD

Hygiene, Sociology, Education and History

LIGHT RESERVE FOR OCCUPATIONS IN SIGHT-SAVING CLASSES R A KAZ, Brit J Ophth 22:482 (Aug) 1938

Kaz emphasizes the role of illumination in relieving the ocular and reflex symptoms of ocular fatigue.

W ZENTMAYER

CHILDREN WITH DEFECTIVE VISION, IN NEED OR NOT OF SIGHT-SAVING CLASS R A KAZ, Brit J Ophth 22:486 (Aug) 1938

Kaz points out that treatment directed to the cause of the reduced vision which has resulted in a child being relegated to a sight-saving class may in time bring sufficient improvement to permit the child to return to schools for normal sighted pupils.

W ZENTMAYER

INSTRUCTION AND PROFESSIONAL ORIENTATION OF AMBLYOPIC CHILDREN. CREATION OF SPECIAL SCHOOLS V ORTIZ, Arch de oftal de Buenos Aires 13:7 (Feb) 1938

In this article an appeal is made for the establishment of special schools for the education of amblyopic children. Such schools were first established in Great Britain by Bishop Hauman and James Keir. The author specifies the conditions required for admission and for the selection of the teaching personnel and the advantages to be derived therefrom.

The teaching methods are next detailed, with classification of the pupils into elementary, medium and high groups. A good general education and proper manual training are advocated, with stress on the importance of the proper selection of a profession.

C E FINLAY

Injuries

SURGICAL TREATMENT OF CAUSTIC BURNS OF THE EYELID. REPORT OF CASES, WITH ILLUSTRATIONS. O. THIES, *Klin Monatsbl f Augenh* 99:764 (Dec) 1937

In an exhaustive manner Thies describes the caustic injuries of the face, lids and eyeball. A detailed synopsis is given of the evolution of the surgical treatment which was begun by Thiersch and was continued and improved by such men as Kuhnt, Czeismack, Stellwag, Axenfeld and others. Short histories are added of a number of cases observed by Thies, which are illustrated by 21 pictures. The course of caustic injuries is described, and the opinion is expressed that only surgical procedures at an early stage will produce results. Early transplantation of the mucous lining of the lip is considered essential. It is evident by the description rendered that all the authors had to follow their own road in the quest for a useful surgical method, many of which were replaced by more practical ones later. The plastic operations on the lids have been perfected to such an extent today that Thies can claim 100 per cent successful results. The duration of extensive plastic operations has been reduced from two hours to one-half hour, with the assistance of one nurse. Several cases are reported of caustic burns of the eyeball. Early plastic operation after Denig's method succeeded in removing disfiguring scars, in saving the sight of some patients and in stopping the progress of destruction in the cornea. The number of these injuries has been greatly reduced in recent years by forced precautionary measures at the factories.

K. L. STOLL

Lacrimal Apparatus

SOME RESULTS OF INTRANASAL DACRYOCYSTORHINOSTOMY. T. E. WALSH and L. BOTHMAN, *Am J Ophth* 20:939 (Sept) 1937

Walsh and Bothman give the following summary and conclusions of their experiences with intranasal dacryocystorhinostomy.

"Twenty-one tear sacs have been operated on by intranasal dacryocystorhinostomy with 85.7 per cent successful results.

"For cases of dacryocystitis with obstruction at the lower end of the sac, operation via the nasal route is preferable to dacryocystectomy, which does not often relieve tearing, and to the Toti operation, which leaves a scar."

W. S. REESE

SYPHILIS OF THE LACRIMAL SAC SIMULATING ACUTE DACRYOCYSTITIS. P. DESVIGNES, *Bull Soc d'ophth de Paris* 50.13 (Jan) 1938

Syphilitic lesions in the vicinity of the lacrimal sac are rare, the chancre being exceptional. Periostitis with exostosis occurs at times during the tertiary stage. Cases of gummatous tumor of the lacrimal sac were reported by Kalt, Rollet and Colrat reported 1 case and Michaesco reported 3. The latter are similar to Desvignes' case in that the mass was soft and painful. When first seen, the patient, 36 years of age, complained that the condition had been present for a

number of days. The lesion had all the characteristics of acute dacryocystitis. Hot applications had little effect, and on incision much blood and a few drops of yellowish, viscid fluid gushed forth. The red tumefaction, a slight amount of pain and a fistula persisted. On exploration of the fistula, a gummatous process in the ethmoid bone was noted. The necessity of a nasal examination and the elimination of a possible diagnosis of tuberculosis are emphasized.

L. L. MAYER

Lens

TOTAL EXTRACTION OF THE OPACIFIED CRYSTALLINE LENS WITH SNELLEN'S LOOP. F. P. ALLNDE, Arch de oftal de Buenos Aires 13: 89 (Feb.) 1938

After reference to the current methods of extraction of the lens in toto, the author describes his method of extraction with Snellen's loop. He reports 15 cases in which good results were obtained.

C. E. FINLAY

EXPERIMENTAL STUDIES ON THE CONTRACTILITY OF THE LENS FIBERS. P. GILLESSEN, Arch f Ophth 138: 598 (May) 1938

The anatomists Leuwenhoek and Pemberton called the lens "musculus crystallinus" because they thought that the lens fibers were contractile. At the beginning of this century this view was revived by W. Krauss, his pupil GillesSEN now reports experiments in which small quantities of acetylcholine or histamine (80 micrograms of active substance, not stated in how much water) were injected into the equatorial portions of the lenses of rabbits, cats, dogs and monkeys. The animals were under a general anesthetic and had received a certain amount of atropine (obviously not enough to produce cycloplegia). The intralental injections were made with a glass capillary, which was introduced through the periphery of the cornea into the anterior chamber and from there through the dilated pupil in the periphery of the lens. If the pupil remained the same width during the injection and for a half hour after it, the author concluded that all of the active substance had been deposited in the lens and that none of it had escaped into the aqueous. The refraction was determined skiascopically before and several times after the injections. Fifteen minutes after the injection the author found a considerable increase, and half an hour later a decrease, in the refraction of the eye treated in this way. "Acetylcholine and histamine, if deposited in the lens cortex near the equator, are capable of eliciting contraction and relaxation of the lens fibers. The lens fibers are, therefore, no plasmatic tubes nor elastic fibers, but contractile elements similar to smooth muscle fibers."

P. C. KRONFELD

Methods of Examination

BINOCULAR FIXATION FOR SCIOPTICARY AND OPTOMETRIC. J. FOSTER, Brit J Ophth 22: 426 (July) 1938

Binocular fixation at longer distances than 33 cm is still rather a problem, and existing apparatus is likely to interfere with the field of the examined eye, to be somewhat inaccurate or to be complicated.

and expensive The author states, however, that Cowan and Markov have produced a simple method which overcomes these difficulties, but their device is somewhat unwieldy Pinned to the Bjerrum screen is a gray circle 5 inches (12.7 cm) in external diameter, painted on a 7 inch (17.8 cm) square of black paper, so that the center of the circle overlies and conceals the fixation point on the screen The screen should have an even illumination of at least 7 foot candles The second part of the apparatus consists of the cross projector, resembling an ordinary ophthalmoscope The patient holds the instrument in one hand and sees an illuminated image of a cross which appears as if it were situated on the Bjerrum screen The article is illustrated

W ZENTMAYER

Neurology

HYPERTENSIVE MENINGEAL HYDROPS L M DAVIDOFF and C G DYKE, *Am J Ophth* 20 908 (Sept) 1937

A syndrome characterized by increased spinal fluid pressure, headache and papilledema is discussed, the literature is reviewed and 15 cases are presented The authors suggest that this syndrome be called "hypertensive meningeal hydrops" The characteristics to be noted in the pneumoencephalogram are a normal ventricular system, difficulty in filling the ventricles during ventriculography and the escape of gas into the cisterna subarachnoidales and cerebral sulci during ventriculography Subtemporal decompression is an important therapeutic measure, especially if elevation of the optic disks is great and the patient's vision is threatened The prognosis is uniformly good, although the cure does not occur rapidly The authors believe that it takes months or even years before a normal equilibrium between production and absorption of the cerebrospinal fluid is established

W S REESE

OPTOCHIASMIC ARACHNOIDITIS DUE TO PNEUMOCOCCUS M GUILLERMIN and J PESME, *Bull Soc d'opht de Paris* 50:16 (Jan) 1938

A young soldier in the course of his training noticed a sudden and marked loss of vision Examination showed bilateral choked disks, greater on the right side, an absence of hemorrhages and wide but reactive pupils The visual acuity in the right eye was 1/100 and in the left eye 1/50 The results of the entire general examination were negative Exploratory visualization of the optic chiasm revealed marked thickening and redness of the arachnoid When the adhesions were broken, the cerebrospinal fluid in this region appeared cloudy Recovery was complicated by epileptiform seizures and an increase in temperature Frequent spinal taps showed cloudy fluid, which, when cultured, revealed a strain of pneumococcus The patient died, and on the basis of the results of autopsy the authors are of the opinion that a localized pneumococcic arachnoiditis was disseminated by operative intervention

L L MAYER

Ocular Muscles

ON SO-CALLED REFUSION K RAAB, *Klin Monatsbl f Augenh* 99: 475 (Oct) 1937

Haploscopic research showed that accommodation and convergence fail often in adjusting the visual axes for the intended point of fixation, even in the presence of emmetropia and orthophoria. This so-called physiologic exophoria is compensated by fusional convergence. The convergence may be increased beyond the necessary measure, while the degree of accommodation remains unaltered. The rotations possible in obtaining increased or decreased convergence, while the accommodation remains unchanged, is called the relative width of convergence. The upper limit of relative convergence used to be considered the point at which the convergence changed into diplopia. Pascal found that prior to the advent of diplopia a moment arises in which the binocularly observed single picture appears indistinct. This indistinctness is due to excess accommodation and may be overcome by means of weak concave lenses. The moment when the indistinctness appears is the actual upper limit of the relative convergence. The indistinctness disappears, and two clear pictures will be observed if fusion is prevented by forcing exaggerated convergence. The double images will fuse as soon as the prisms are reduced. This moment is called the refusion point, which is identical with what Pascal called the recovery point. The results of the research done by Pfimlin, Strubin and others are discussed.

Raab arrived at the following conclusions. The refusion point depends on the existing tonus of the muscle, which presents individual differences. The position of the point of indistinctness, which is the upper boundary line of the width of relative convergence, determines the amount to which the tonus may be increased. A primary and a secondary point of refusion can be distinguished in some persons. The position of the primary point can be determined by a persistence of an increased accommodation on convergence, while the secondary point depends on the tonus of the muscle. There are simple hypotonic forms and accommodatively compensated hypotonic forms of insufficiency of convergence, these differ clinically. The crossing of the curves of indistinctness and refusion, as described by Pfimlin and Strubin, are not the rule.

K L STOLL

Physiology

ANISEIKONIA IN EMMETROPIA W L HUGHES, *Am J Ophth* 20: 887 (Sept) 1937

This is a study of selected patients examined for aniseikonia at the New York Eye and Ear Infirmary. Hughes arrives at the following conclusion:

"The relative size of the ocular images of a group of 43 patients having symptoms that were thought to be ocular in origin was determined. In 14 the images were equal. In 29 they were asymmetrical (aniseikonia)."

"The results in 14 cases in which complete data were obtainable are reported. Relief was obtained in 10 by the wearing of iseikonic zero power lenses designed to correct the aniseikonia present. It has been shown that the presence of aniseikonia is independent of any refractive error."

W S REESE

Retina and Optic Nerve

ANATOMICOPATHOLOGIC RESEARCHES ON A GLOBE WITH PARTIAL
RETINAL DETACHMENT OF TWENTY DAYS' DURATION MAGITOT
and LENOIR, Bull Soc d'opht de Paris 50: 34 (Jan) 1938

The eye of a young woman 35 years of age was removed because an intraocular tumor was suspected. Loss of vision was noted suddenly without a history of trauma. The media were clear, the tension was normal, and the detachment was located temporally near the disk. No holes or hemorrhages were found, but a few white spots on the surface and a slight haziness on transillumination were enough to cause tumor to be suspected.

Cystic degeneration was noted in the macular area. The retinal rods and cones were in good condition. The pigment epithelium showed some curious lesions. Islands of atrophy were found in the detached area together with degeneration of the hyaloid fossa. In places, the pigment epithelium seemed to have entirely disappeared. The choroid was in good condition. The suprachoroidal area was filled with an albuminous fluid. The authors have no hypothesis as to the causative agent in idiopathic retinal detachment but simply wish to offer their observations for what they are worth.

L L MAYER

RETINAL BLOOD PRESSURE IN CASES OF NASAL DISEASES AND THE
EFFECTS OF SURGICAL INTERVENTION IN THE NOSE AND NASO-
PHARYNX CATTANEO and LASAGNA Ann di ottal e clin ocul
66: 1 (Jan) 1938

Pressure in the retinal arteries was recorded with Bailliar's dynamometer and in the capillaries by entoscopy during pressure with the same instrument. Although actual measurements are not given, a more or less marked hypertension in the retinal vessels as compared with the brachial blood pressure was noted in 57 per cent of persons with various nasal diseases. Immediately after nasal operations, the incidence of relative hypertension rose to 76 per cent, while after a month or more only 27 per cent of the patients were found to show relative hypertension. This is considered to illustrate a vasomotor reflex between the retinal vessels and the nasal mucosa.

S R GIFFORD

DISCIFORM MACULAR DEGENERATION OF JUNIUS-KUHNT G FAVALORO,
Ann di ottal e clin ocul 66: 81 (Feb) 1938

The author reports 3 cases of disciform macular degeneration in patients 60, 53 and 55 years of age. The condition in 1 case was seen in an early stage, with little elevation of the newly formed tissue. That in the other cases was typical of a more advanced stage. In 1 case only one eye was affected. All patients showed a moderate degree of hypertension. The literature is reviewed, and especial attention is given to the few cases in which histologic examination was made. On account of its characteristic ophthalmoscopic picture, the author believes that the condition is a definite clinical entity which should be distinguished from other changes affecting the macula. He considers senile macular disease and familial macular disease to be a purely degenerative process,

while the exudative processes described by Axenfeld as retinitis externa is of an inflammatory nature. Disciform macular degeneration, he believes, shows a primary reactive transudative phase, followed by degenerative changes. The part played by the choroid in the early stages is emphasized.

S R GIFFORD

MEASUREMENTS OF THE CALIBER OF THE RETINAL VESSELS OF
PATIENTS WITH HYPERTENSION AND NEPHRITIS G BADTKE,
Klin Monatsbl f Augenh 99:655 (Nov) 1937

In this detailed paper Badtke renders an account of the technical considerations of measurements of the retinal vessels. He adds a short and clear division of the types of hypertension and nephritides and of their symptoms. Tables are presented which show notes on the condition of the disk, the retina and its vessels and the blood pressure. The caliber of the retinal vessels was measured with Lobeck's *Messokular*. Table 5 shows the width of the retinal veins and the relation of the veins to the arteries in the various types of hypertension and nephritis. The results of Badtke's research follow. The width of the retinal arteries was smaller than that of the veins of healthy persons, even if the difference was slight. The arteries were somewhat wider than normal in cases of red hypertension. In some cases the artery was wider than the vein. The arteries were never contracted in cases of genuine red hypertension. Transition from benign red hypertension to malignant pale hypertension was evidenced by contraction of the arterial system. It was evident in the periphery in the beginning but could not be measured with Lobeck's method. The arteries near the disk showed unevenness of the caliber and occasional contraction for certain distances, these could usually be measured well with Lobeck's method. The contraction of the arteries was most evident in cases of complete pale hypertension. In cases of chronic nephritis no contraction of the arterial system was noted as long as the systolic and diastolic pressure remained low and as long as no ophthalmoscopic changes were present, the measurements equaled those found in healthy persons. The picture resembled that of red hypertension in some cases of high systolic blood pressure. Measurements of the caliber in these cases showed slight distention of the arteries.

K L STOLL

CONGESTION OF THE RETINA INDICATING INVOLVEMENT OF THE
RETINAL VESSELS IN FAILURE OF THE RIGHT SIDE OF THE HEART
REPORT OF CASES G JANKE, *Klin Monatsbl f Augenh* 99:
756 (Dec) 1937

Janke refers to a previous report of a case of cyanosis of the retina which was not coupled with polycythemia (*Klin Monatsbl f Augenh* 96:605 [May] 1936). He reports 3 similar cases of congestion of the retina in which polycythemia, hitherto considered a regularly concomitant symptom of this disease, was absent. The patients suffered from emphysema of the lungs and cardiac failure. The circulation of their pulmonary blood was retarded by atrophy of the alveolar walls, lack of capillaries and inactivity of the thorax. This condition produced

compensatory hypertrophy of the right side of the heart, followed by dilatation of the chamber on that side. The result was retention of an amount of residual blood during each systole, as the heart was unable to handle the normal quantity of blood. As a consequence, stasis developed, which finally extended from the right atrium of the heart to the veins of the body. Simultaneously with asphyxia, cyanosis and congestion of the liver, considerable increase of the pressure in the venous system was noted. Primary polycythemia existed in none of the 3 cases, and secondary polycythemia of a low degree in 1. A high degree of distention and tortuousness of the retinal veins was observed in each case. The disks were slightly congested in the cases of milder involvement, and edema and hemorrhages of the disks and retina were present in the cases of severe involvement. The gravity of the pathologic changes in the fundus permit no prognostic conclusions. Decompression of the vascular system followed treatment in juvenile patients. Janke disagrees with Sobanski, who believes that choked disk will result from lack of correlation between the increased venous tension due to increased cerebral tension and the arterial pressure in the retina. It is Janke's opinion that in congestion of the retina the venous stasis is primary and the edema of the disk secondary, in choked disk, on the other hand, the edema of the optic nerve is primary and the venous stasis secondary.

K L STOLL

TREATMENT OF RETINAL DETACHMENT BY MEANS OF DIATHERMY
AND INJECTION OF AIR INTO THE VITREOUS B ROSENGREN, *Acta
ophth* 16:3, 1938

Since 1930 operations for the relief of retinal detachment have been performed with increasing frequency. In spite of improvements in technic, there has not been a corresponding increase in the number of cures obtained. They amount to about 50 per cent, as in Gonin's time. The author has interested himself in the reason for the numerous failures which still occur. They may be attributed, he believes, not to improper localization of retinal tears or inadequate cauterization but to failure to secure proper apposition of the retina to the underlying structures. Therefore, in addition to the usual procedures of diathermy and drainage, the author injects 1 or 2 cc of air into the vitreous. The bubble acts as a tampon, which exerts pressure on the retina and serves to prevent seepage of vitreous through the retinal tear. The air itself, because of factors of capillarity, does not penetrate beneath the retina. It is of the utmost importance to keep the air bubble in contact with the retinal tear. This is effected by means of appropriate sutures beneath the rectus muscles and by suitably altering the position of the patient. Arruga's failures with injection of air are attributed by the author to nonobservance of this detail. The air is injected by means of a needle 0.36 mm in diameter, inserted 5 or 6 mm behind the limbus.

In a series of 20 operations on 18 patients cures were obtained in 14 instances.

O P PERKINS

Trachoma

STUDIES ON THE INFECTIVITY OF TRACHOMA VII FURTHER OBSERVATIONS ON FILTERABILITY OF THE INFECTIOUS AGENT L A JULIANELLE, M C MORRIS and R W HARRISON, *Am J Ophth* 20: 890 (Sept) 1937

This is the seventh article on the filterability of the infectious agent of trachoma. The following summary and conclusions are given:

"1 Further studies have been made on the filterability of the infectious agent of trachoma, employing Berkefeld (kieselguhr), Elford (collodion), Kramer (plaster of Paris), and Seitz (asbestos) filters.

"2 In all of five experiments on filtration of infectious tissues through Elford filters alone, the filtrates were noninfectious.

"3 In seven additional experiments with active material, parallel filtrations with all four filters yielded on two occasions filtrates which were infectious, both resulting from Berkefeld V filtrates only.

"4 Filterability has been demonstrated, therefore, in two of 12 trials, as compared with one of 11 trials in a former study.

"5 The evidence indicates that the infectious agent of trachoma is irregularly filterable, and that the difficulties in filtration may be attributable to variations in the infectious tissues themselves, as well as in the composition of the filter employed.

"6 The possible factors involved in unsuccessful filtration are discussed and a hypothetical explanation is offered."

W S REESE

Tumors

FIBROMA OF THE SCLERA V CERCKEZ, *Ann d'ocul* 175: 390 (May) 1938

Tumors of the sclera are rare, and fibromas are especially so. All authors class fibroma of the sclera in the group of orbital tumors.

V M, a farmer aged 22, was seen because of exophthalmos of the right globe, with severe pain and loss of vision on that side. For the preceding two years he had had intermittent pain on the right side of the head. The pain grew gradually worse, and in the meantime he had been sent to a military hospital, where the Wassermann reaction was found to be negative, and roentgen examination of the orbit showed nothing abnormal. After four weeks of rest, the pain had improved, but a slight exophthalmos remained. The patient was returned to his regiment, but his condition grew worse, until he was unable to see well, the eye was then enucleated and examined pathologically. It showed a fibroma which came from the sclera.

Of particular importance is the pathogenesis, development and evolution of fibroma of the sclera. In this case the patient did not receive any trauma to the eye, which led the authors to believe that the tumor was of congenital origin. Extension of the tumor manifested itself behind in the orbit as well as in front, making it difficult for them to examine the back of the eye. The development and breaking down of the inflammatory phenomena were due probably to a special topography of the blood vessels, especially the veins. A bibliography accompanies the article.

S H McKEE

VOLUMINOUS CHONDROMA OF THE LEFT ETHMOID REGION WITH
MUCOCELES IN ALL SINUSES AND A GRAVE OCULAR SYNDROME
E RUBALTELLI, Riv oto-neuro-oftal 14: 48 (Jan -Feb) 1937

A boy of 16 years, in good health, noted a progressive divergence of the left eye, protrusion of the globe and a marked reduction in vision. The disk was red and slightly elevated, with well defined edges and with markedly dilated and tortuous veins. Examination of the nose gave negative results. Palpation revealed a hard painless mass in the superomedial portion of the orbit. It was firmly fixed to the medial wall, from which it seemed to rise. Roentgenographically, an opaque mass could be demonstrated occupying the ethmoid cells, extending into the nasal fossa, the orbit and the maxillary sinus of the same side. In addition, the frontal and maxillary sinuses were clouded.

The mass was removed by a transmaxillary and central facial approach of Citelli. It had an osteocartilaginous consistency and histologically showed the characteristics of a chondroma. In addition, two large mucocèles were found occupying the frontal and the maxillary sinuses. Postoperative examination showed that vision, fundus and mobility had returned to normal. The text is illustrated with 9 photographs and drawings.

F P GUIDA

TUMORS OF THE ORBIT CYLINDROMA OF THE LACRIMAL GLAND
Q DI MARZIO, Riv oto-neuro-oftal 14 65 (Jan -Feb) 1937

Di Marzio reviews the literature on the subject and reports 7 cases of cylindroma of the lacrimal gland. He presents the clinical histories, operative procedures and results of histologic examination. Of these 7 patients, the first was well after sixteen years, the follow-up studies on the second and third were incomplete. The fourth patient showed a recurrence after twenty years and three years after a second operation died of cerebral metastasis. In the fifth case roentgen therapy was used after the third recurrence, with a cure of eight years' duration. The other 2 cases were recent.

After discussing the theories of the origin of these tumors, Di Marzio concludes that the elements which go to form the tumor vary, arising from the different tissues that go to make up the lacrimal gland embryologically, hence, the endothelial form of the growth at one time and the epithelial at another time. Early complete excision, including the lacrimal gland, followed by roentgen irradiation to avoid recurrence is advised, as the tumor is of a malignant nature.

F P GUIDA

Vision

A VISUAL-ACUITY SURVEY C W RAINEY, Am J Ophth 20 930
(Sept) 1937

Rainey calls attention to the lack of a clinical report of a visual efficiency survey using the percentage method since the publication by the American Medical Association in 1925 of a standard method of

appraisal of loss of visual efficiency He submits a survey of 721 men between the ages of 20 and 75 years, giving the following conclusions

"1 Because the visual efficiency rapidly decreases with age, due to the loss of accommodation and the prevalence of subnormal and abnormal conditions of the eyes, the need for periodic visual-efficiency examination at one- to two-year intervals is apparent

"2 Homatropine (4 percent) with cocaine (2 percent) can be used conveniently in determining the refractive state of the eyes at any age, without risk of inducing acute glaucoma

"3 The use of 0.5-percent atropine solution, four drops a day for four days, is recommended to supplement the effect of homatropine in cases of high refractive error

"4 Cylinder skiascopy, including the rotatory trial, was especially useful in groups 2 and 3, for patients whose vision could not be brought to normal and who were often unable to help in the subjective tests at the trial case

"5 The manifest method of refraction is inadequate and inefficient as compared with the method using cycloplegia

"6 The visual efficiency of each of the three groups could be increased by refraction and spectacles The efficiency of the whole group was raised approximately 25 per cent for distance and 45 per cent for near The gain of the group already wearing spectacles was 14 percent for distance and 17 percent for near

"7 The tests for color blindness were an aid to diagnosis

"8 The ophthalmologist best serves the interests of the employee and employer

"9 The Standard Method of Appraisal of Loss of Vision should be used as the basis of all group examinations

"10 Examinations of the visual fields with the tangent curtain should be made in all cases in which the vision is not normal after refraction has been corrected"

W S REESE

Sympathetic Ophthalmia

IS PERFORATION OF AN EYE NECESSARY FOR THE PRODUCTION OF SYMPATHETIC OPHTHALMIA? EINAR ANDERSSON, *Acta ophth* 16: 119, 1938

There have been reported a number of cases of sympathetic ophthalmia in traumatized eyes wherein no penetrating wound has been found The author makes the statement that in none of these cases has the eye been serially sectioned, which, he believes, is the only sure method by which to eliminate the possibility of the presence of a perforation

In the case reported here, that of a 3 year old child, the history obtained from the parents was that of contusion by means of a stone to the right eye No signs of perforation were evident clinically Hemophthalmos and secondary glaucoma were present Later, the cornea was stained with blood, and the eye became soft Three and a half

months after the injury a severe sympathetic ophthalmia occurred and progressed unremittingly despite all treatment, including enucleation of the exciting eye

A minute needle-like penetrating wound involving the sclera, choroid and retina was found only after serial sections of the globe were made

Although in this case the object which struck the eye was probably something other than a stone, the author reminds one that contusions may produce minute tears in the coats of the eye

An interesting feature of the case was that the child did not react to tuberculin, nor could any evidence of tuberculosis be found on clinical or roentgen examination

O P PERKINS

Society Transactions

EDITED BY W L BENEDICT

AMERICAN OPHTHALMOLOGICAL SOCIETY

FREDERICK H VERHOEFF, M D, *President*

Seventy-Fourth Annual Meeting, San Francisco, June 9-11, 1938

EUGENE M BLAKE, M D, *Secretary*

SHALL WE USE CYCLOPLEGICS? DR W H CRISP, Denver

Every refractionist should be able to make an approximately accurate measurement without the use of a cycloplegic. In many cases the difference between careful examinations without and with cycloplegia is slight, but there is always a risk of important differences being overlooked without cycloplegia. This is true even in cases of myopia, especially in the presence of astigmatism or of inequality of the two eyes. In cases of hyperopia careful use of bilateral fogging demonstrates that the amount of so-called latent hyperopia is less significant in most instances than is sometimes assumed. But cycloplegia is important for accuracy in estimating astigmatic error and in the exceptional cases of concealed hyperopia. As to astigmatism, the most important reason for the use of a cycloplegic is not its value in retinoscopic examination but rather the greater certainty of accuracy in the selection of cylindric strength and axis.

DISCUSSION

DR GEORGE F LIBBY, San Diego, Calif. I use a 3 per cent solution of homatropine hydrobromate at about body temperature and apply it by raising the upper lid and putting a drop at the upper edge of the cornea. It is my custom to put 1 drop in each eye every five minutes until I have applied 6 drops, which gives satisfactory cycloplegia.

DR W B LANCASTER, Boston. I agree with Dr Crisp that there is a too general adoption of the idea that cycloplegia should be used in almost all cases, and I favor dispensing with it in many instances, even for children. One wishes to control accommodation because, by contraction and relaxation of the ciliary muscle, accommodation changes the refraction which one is trying to measure, increases myopia and decreases hypermetropia. Contraction of the ciliary muscle does not produce an astigmatic change in the lens so that it can correct an existing astigmatism, however, it is known that accommodation can neutralize astigmatism in some cases. It simply moves the whole conoid of Sturm forward or backward, so that different portions of it fall on the retina. If the interfocal circle falls on the retina, all the lines will be equally distinct or equally slightly blurred, but accommodation does not neutralize the astigmatism by an unequal contraction of the ciliary muscle.

DR H S GRADLE, Chicago I do not agree with some of the statements made by Dr Lancaster in regard to the lack of necessity for the use of cycloplegics in the majority of cases. It is true that refraction can be done adequately in a large percentage of cases without the use of a cycloplegic, but it is equally true that no eye can be examined thoroughly without its use. It is my belief that blindness could be prevented in a large percentage of cases of glaucoma by the early detection through the use of a cycloplegic.

DR EDWARD JACKSON, Denver The question of whether or not to use cycloplegics depends on whether the ophthalmologist wants to learn all that he can about the eye for which he is prescribing or whether he is content to follow his long experience and certain rules that have been laid down without finding out what might be learned about a particular eye or patient. It is always possible to learn more with the use of a cycloplegic than without. From the practical standpoint, the use of a cycloplegic often depends on the patient's resistance to having anything of that sort done.

Two things have often been overlooked in practice. First, a partial correction is not a correction of the error of refraction. The patient with a partial correction of myopia supplements this correction by looking obliquely through the lenses, thereby getting the effect of stronger lenses. Often he gets that effect at the cost of introducing astigmatism. A similar condition applies in cases of hyperopia.

One other point that is overlooked is that every person probably is able to relax his accommodation completely when he goes to sleep. He can learn to relax it completely and look through his glasses much more quickly than he can learn to use a lesser amount of correction and see clearly through his glasses. It is a general rule that all persons can relax accommodation when they go to sleep and that it is easier for them to learn to relax accommodation with the full correction before the eye than to relax it partially and still see clearly.

There are surprises all the time. There are children who take a +1 diopter lens, and in spite of all efforts of the parents to have them wear glasses faithfully they will notice a definite blurring in reading the letters after wearing the glasses for some time. One must be guided in the individual case, but the patient is not being given a fair chance unless one tries to have him wear the full correction.

SUBJECTIVE STUDY OF VISUAL ABERRATIONS DR EDWARD JACKSON, Denver

Newton explained the rainbow, and Helmholtz pointed out how the focusing power of the crystalline lens might change. But some common phenomena of vision still remain to be understood and explained. The conventional figure of a star, recognized for thousands of years, depends on aberrations presented in every dilated pupil. This can be studied by shutting off different parts of the pupil, this became known only when the practice of skiascopy was begun.

The radiating lines seen about the reflection of the sun from any polished surface are caused in the eye by reflections from the crystalline lens, near the anterior pole. They can be studied by looking at such

reflections through a small aperture. They probably assist the nutrition of the retina by distributing light to its pigment layer, without interfering with the usual retinal images. The lines are best seen against a black background. Subjective studies of the blindspot and visual fields, as described in the 1935 *Transactions of the American Ophthalmological Society*, have shown that certain primitive ocular movements are not coordinated with the retinal sense of direction and magnitude, which serves for most of the important functions of the eyes. The facts revealed by subjective study of vision are of practical value in diagnosis and in explaining to patients symptoms that have alarmed them.

DISCUSSION

DR W B LANCASTER, Boston. All the rays that traverse the lens do not focus at a point, but in Stium's conoid there is a plane which can be selected by the eye for a good retinal image. (A diagram was used for purposes of demonstration.) The function of accommodation is to pick out the place which makes the best image, and that is what the eye does all the time. If the accommodation is paralyzed, the eye is unable to do this. With a mixture of rays the eye has a habit of picking out one place which suits it best, and when one introduces other rays at the periphery of the lens by dilating the pupil, the eye may still be able to select its original preferred point, as was mentioned by Dr Crisp. The eye may do this in some instances, but if one wants to find out what the eye really does, one must examine it under the conditions under which it really works and not under cycloplegia, because then the aberrations are different and the eye must make a little different adjustment.

MIXED CELL TUMOR OF THE LACRIMAL SAC DR JOSEPH L. MCCOOL, San Francisco

Mixed tumors occur commonly in the salivary, parotid and lacrimal glands and in the buccal mucosa. Mixed tumors of the lacrimal sac are rare. These neoplasms are of complex structure, usually presenting epithelial elements in the form of cell strands and neoplastic tissues, chiefly cartilage, mucous tissue and connective tissue. Any one of these elements may predominate, giving nearly pure chondromas, sarcomas or carcinomas, but usually all of the cell types are represented. Age appears to play no part, cases being reported in persons of from 11 months to 73 years of age.

A man, aged 54, complained of lacrimation of six months' duration and a slowly growing mass in the region of the left tear sac. Inspection revealed a hard, solid mass 1 by 1 by 0.5 cm. in the lacrimal fossa. The skin over the mass was normal in color and not particularly tense. The mass was not tender on palpation, and no secretion exuded through either punctum on pressure. The tumor was removed without difficulty. It was a soft, yellowish, translucent mass. Pathologic examination revealed at one edge of the section ciliated epithelium with marked round cell infiltration in the underlying tissues, which in places formed lymphoid nodules. Separated from this by a dense connective tissue septum was a mass of more neoplastic-looking tissue composed of a

mixture of fibroblastic tissue, round cells and cells of indefinite shape with rather large, clear, oval or round nuclei and without distinct nucleoli. Mitotic figures were common in some areas among the cells. Clear myxomatous areas were present in a small amount. There was infiltration of all the types of tissue between the striated muscle fibers at the edge.

A diagnosis of mixed tumor of the lacrimal sac was made. Radium was implanted in the cavity from which the tumor was removed. There was no recurrence ten months after the operation.

DISCUSSION

DR JOHN W. BURKE, Washington, D. C. A man, aged 73, seen on March 18, 1936, complained of sticking of the lids and epiphora. Examination showed a small growth protruding from the lower right punctum. Pressure on the lid would make the growth protrude further, on relaxation, it would drop back into the dilated canaliculus. On palpation, a rather firm, solid growth was felt. On April 23, with local anesthesia, the canaliculus was split to the inner canthus. The growth was lifted out. It did not seem to be adherent, and there was no bleeding when it was removed. The mucous membrane of the canaliculus was stretched and slightly hyperemic but otherwise showed no changes. The tumor was pear shaped and measured 8 by 5 mm. It was pinkish yellow, slightly vascular and not pigmented.

Microscopic examination showed a papillary tumor with small connective tissue cores covered by multiple layers of stratified squamous epithelium with some leukocytic infiltration in the tumor and in the stalks. There was no evidence of infiltration, and the lesion had all the appearance of a benign tumor. A diagnosis of epithelial papilloma was made.

PANOPHTHALMITIS AND SYMPATHETIC OPHTHALMIA DR BERNARD SAMUELS, New York

This article appeared in full in the November issue of the ARCHIVES, page 804.

METASTATIC SEPTIC ENDOPHTHALMITIS WITH RING ABSCESS OF THE CORNEA. REPORT OF A CASE WITH DESCRIPTION OF ANATOMIC CHANGES. DR C. W. TOOKER, St. Louis

A man, aged 53, entered the hospital in June 1932 for the treatment of a bilateral mastoiditis. After operation he seemed to have made a good recovery but was brought to the hospital again in January 1933, when a staphylococcic septicemia was discovered. Vision in the left eye was limited to perception of light. The cornea was clouded. The iris and anterior chamber were filled with exudate. Blood cultures on two occasions showed the presence of staphylococci. The patient lived three days, and a complete autopsy was done. The left eye was removed and sectioned.

(Sections and photomicrographs were shown to demonstrate the anatomic changes.)

ORBITAL HYPEROSTOSIS ITS OCCURRENCE IN TWO CASES OF MENINGIOMA OF THE SKULL DR ARNOLD KNAPP, New York

This article appears in full, with discussion, in this issue of the ARCHIVES, page 996

(a) LEIOMYOMA AND (b) BLOOD CYST OF THE IRIS REPORT OF CASES DR E C ELLETT, Memphis Tenn

This article will appear in full with discussion, in a later issue of the ARCHIVES

OCULAR MANIFESTATIONS IN BRUCELLOSIS (UNDULANT FEVER) DR JOHN GREEN, St Louis

This article will appear in full, with discussion, in a later issue of the ARCHIVES

SYPHILITIC OPTOCHIASMIC ARACHNOIDITIS DR DERRICK VAIL, Cincinnati

A review of the literature revealed numerous reports of cases of optochiasmic arachnoiditis in persons who showed clinical or laboratory evidence of syphilis. A man, aged 58, who was thought to have been tabetic for five years and had received long courses of antisiphilitic treatment, complained of progressive loss of vision in spite of the treatment. The right eye became entirely blind, and vision in the left eye was reduced to ability to count fingers at 3 feet (91 cm) because of a large central scotoma. The outline of the disks was sharp, except for nasal blurring and a depressed center, and there was a visible, greenish white lamina cribrosa, the vessels were markedly reduced in caliber. A diagnosis of optochiasmic arachnoiditis was made. Craniotomy in the right frontal region, performed on April 4, 1938, by Dr Nolan Carter, of Cincinnati, disclosed an old leptomeningitis, involving especially the under surface of the frontal lobes. The chiasm and optic nerves were hidden and entangled in sheetlike bands of old inflammatory tissue. Examination of a fragment of the thin inflammatory membrane showed a thickened arachnoid with epithelioid cells and secondary fibrosis.

DISCUSSION

DR RALPH I LLOYD, Brooklyn. I had a case of specific chiasmic arachnoiditis in which the change came on suddenly. The right eye became blind overnight. Examination of the other eye showed temporal quadrupia. The patient was seen by an otolaryngologist, who removed the ethmoid cells on the affected side. The tissue was sent to a pathologist, who reported hyperplastic rhinitis and said that it was a sufficient cause to explain the sudden loss of vision. In about a month the other quarter of the affected half-field disappeared, leaving a picture typical of that seen in disease of the pituitary gland, i. e. one blind eye and hemianopia in the other eye. The vision before the operation was nearly 20/20 and the knee jerks were normal. At the end of the following month vision in the macular area of the remaining half-field was com-

pletely gone In the course of the next three months the patient was totally blind His Wassermann reaction was 4 plus He first said that he had never had a syphilitic infection, but he finally admitted having had one thirty-five years previously

(The manner in which the visual field became constricted to the vanishing point was demonstrated by slides, which revealed particularly the similarity of the changes in the visual field to those associated with lesions of the pituitary body)

DR DERRICK VAIL, Cincinnati I am grateful to Dr Lloyd for pointing out the progressive loss of the field of vision in these cases from beginning to end He pointed out the importance of the chiasmal area in certain lesions and called attention to chiasmic defects in the visual field, which can explain many other ramifications, for example, the traumatic type of optochiasmic arachnoiditis and the suggestion that syphilitic atrophy of the optic nerve is due to a lesion arising in the chiasmic cisterna and affecting the optic nerve

LESION OF THE OPTIC TRACT PROBABLY THE RESULT OF INFECTED SPHENOID SINUSES DR ALBERT N LEMOINE, Kansas City, Mo

This article appears in full, with discussion, in this issue of the ARCHIVES, page 966

CORRECTION OF ENTROPION BY TRANSPLANTATION OF THE ORBICULARIS MUSCLE OF THE LOWER EYELID DR JOHN M WHEELER, New York

Many procedures have been suggested for the correction of spastic entropion Most of them, but not all, depend for their effectiveness on a downward pull calculated to unroll the inverted lower lid and to prevent turning in of the margin by downward traction on the skin

The operation described depends for its effect on pressure at the lower margin of the tarsus of the lower lid, obtained by tightening some of the fibers of the orbicularis muscle The operation prevents entropion and at the same time gives support to the eyelid

DISCUSSION

DR EDWARD JACKSON, Denver Is there any difficulty in isolating the portion of orbicularis muscle that is wanted, and how does one judge this portion in cutting the strips of muscle before proceeding to use them?

DR J E WEEKS, Portland, Ore For many years I have employed a simple operation for the correction of spasmodic entropion of the lower eyelid with most satisfactory results An incision is made 3 mm below and parallel to the margin of the lid, extending from the punctum almost to the outer commissure A curved incision is now made connecting with the extremities of the first incision, including as much integument below the first incision as is thought desirable The subcutaneous tissue and the underlying fibers of the orbicularis muscle are excised with the skin The margins of the wound are approximated by means of fine silk sutures Three sutures are then passed through the margin of the

lid from above downward, beginning in the intermarginal space and emerging on the cutaneous surface 2 mm below, one suture being in the middle of the lid and one on each side, about halfway to the canthi. Each suture is then passed into the skin of the cheek about 4 mm directly below its point of first emergence, again emerging 2 mm farther down. A small roll (3 mm in diameter) of iodoform gauze is passed beneath the sutures, and the ends of the sutures are tied, causing marked ectropion of the margin of the lower lid. The sutures are removed at the end of six or seven days.

DR RAMON CASTROVIEJO, New York. With no knowledge of Dr. Wheeler's operation but simply after watching him work I decided to operate in a couple of cases. I did not find the procedure difficult. I have also been able to correct entropion of the upper lid by the same method.

DR JOHN M. WHEELER, New York. Picaine hydrochloride is thoroughly injected into the lower lid, so as to get good magnification of the fibers and to reduce the entropion while the operation is going on. In the second procedure I spoke of it is perfectly feasible to clamp the lid so that there will be no hemorrhage. Since one is almost sure to cut the palpebral arteries during dissection, one should clamp them. I like to leave the orbicularis muscle in place and with the scissors cut down against the tarsus in which the strips are outlined, I then lift the outlined strips with a pair of forceps and cut under them with very fine scissors. By holding the strip on a hook it is easy to carry on the dissection toward the end of the lid and then to put in the sutures in such a way as to be sure of the accurate overlapping and the position of the tightened strips of orbicularis muscle.

A NEW METHOD FOR TRANSPLANTING PTERYGIUM DR EDWIN M. NEHER, Salt Lake City

This article will appear in full, with discussion, in a later issue of the ARCHIVES.

INTERIOR IRIDOTOMY IN OPERATIONS FOR CATARACT ON EYES WITH POSTERIOR SYNECHIAE OR PUPILLARY MEMBRANE. VALUE OF OPERATION DR PAUL A. CHANDLER, Boston

This article appeared in full in the October issue of the ARCHIVES, page 641.

DISCUSSION

DR DERRICK VAIL, Cincinnati. Dr. Chandler's contribution is going to be of great value to any one who does operations for cataract, and I know that I will follow his precepts in such cases as he described. For a number of years I have performed inferior iridotomy in other cases, particularly in those in which there has been loss of vitreous following the extraction of cataract. After loss of vitreous the pupil always assumes a hammock shape or a boat shape. This produces a drag on the pillars of both sides which may be obviated by doing an inferior iridotomy at the time of the extraction, in spite of the danger of further loss of vitreous. If the operation is skilfully done with sharp Noyes scissors, further loss of vitreous is not great.

OPHTHALMOLOGY COMES OF AGE I THE YEARS 1908-1915, UP TO
THE ESTABLISHMENT OF THE EXAMINING BOARD DR SYLVESTER
JUDD BEACH, Portland, Me

History relates that in 1913 the three national ophthalmologic societies appointed a joint committee. After due consideration, this committee evolved the scheme of an examining board. As a consequence, in 1915 what is now the American Board of Ophthalmology was organized. Even in the chronicles of the board there is no hint that there was anything more complicated than this about the origin of a movement which has revolutionized postgraduate medical education. In reality, for a period of years several groups had in divergent ways been wrestling with an unsavory situation. It was by deft use of the momentum they created that this solution was finally reached.

DISCUSSION

DR GEORGE F LIBBY, San Diego, Calif. In 1912, under Dr Jackson's directions, there was established in Denver a postgraduate school for advanced teaching in ophthalmology. There were 11 students, 1 from Pennsylvania, 2 from the Middle West and 8 from Denver. The course was of six weeks' duration. In the following year, after a year's reading in ophthalmology under Dr Jackson's direction, 3 members of the class received the degree of Doctor of Ophthalmology at the state university. This movement, leading to the certification of men who had taken advanced work in ophthalmology, such as the course given in Pennsylvania and similar courses in the University of Minnesota, and to the degree of Doctor of Science in Ophthalmology, had an important bearing on the development of the board.

DR W B LANCASTER, Boston. The society is grateful to Dr Beach for this important historical contribution. Perhaps the ophthalmologists here think that it is a simple matter to write such a paper, that all one has to do is to look up the records of the *Transactions* to find what was done year by year! There is a great deal more to it than that. The historian has to evaluate what he finds and interpret it, but even the records are meager. They simply say "it was moved and carried, etc." Take the epoch-making meeting of last night, when the future historian tries to find out the facts he will simply be confronted with the record "moved and voted." Nothing will be said of how Horatio held the bridge, who fought on his right hand and who on his left hand, what soldiers stood behind him or how they met the onslaughts of the reactionaries and the die-hards! Perhaps every one thinks that when the American Board of Ophthalmology was formed everything was peaceful, but I can assure them that that baby was born with tears and travail and that when the board first made its appearance many a finger of scorn and ridicule was pointed at it by men in high position. Fortunately in those days the leaders were men of vision and of courage, but of vision especially, who could see the future when others were in the fog and who led on to great achievement, so that now ophthalmologists are proud to have the stone which was originated by many of these men made the headstone of the corner ophthalmology leading all the specialties, from otolaryngology to surgery.

TRAUMATIC RETINAL ANGIOPATHY DR ARTHUR J BEDELL, Albany, N Y.

The change in the fundus in cases of traumatic retinal angiopathy, consisting of large fluffy white masses with many superficial and deep hemorrhages about the disk, is pathognomonic. This condition was mentioned by Jacobi in 1868 and was completely described by Purtscher in 1910, since which time his name has been commonly associated with it.

(Lantern slides of previously published illustrations accompanied a review of the literature.)

I wish to add 3 new cases to the small number reported. The condition in 1 was caused by a severe injury to the side of the head, in another, by compression of the chest, traumatic asphyxia and fracture of the dorsal vertebrae, and in the third, by fracture of the third and fourth lumbar vertebrae.

(Serial color photographs of the fundus were shown to demonstrate the clinical history.)

THE CULTIVATION OF HUMAN CONJUNCTIVAL AND CORNEAL EPITHELIUM IN VITRO DR PHILLIPS THYGESON, New York

Certain viruses affecting the human conjunctiva and cornea have failed to grow in cultures of chick embryo tissue, and many require human epithelial tissues. Experiments on the cultivation of human conjunctival and corneal epithelium are reported, including notes on the histologic structure of the developing tissues. A technic suitable for use in virus studies is outlined.

DISCUSSION

DR DANIEL B KIRBY, New York. Dr Thygeson is to be congratulated on the development of this method of cultivation of tissues from the human eye. Dr Carrel and Dr Ebeling have both expressed themselves as admiring very much the progress which Dr Thygeson had made in this research. It will be of further value in researches in psychology and in pathologic studies of other human ocular tissues.

DR RAMON CASTROVIEJO, New York. I was particularly interested in Dr Thygeson's presentation, because he has brought out some points that had been the object of debate. He has illustrated that epithelial tissue cultivated in vitro gives origin only to epithelial tissue. In the same way, connective tissue can give origin only to connective tissue cells.

In reviewing the literature on the histologic picture of keratoplasty, published by some French authors, I was surprised to find the statement that connective tissue cells which developed to repair corneal wounds originated from epithelial cells. This observation entirely disagrees with Dr Thygeson's findings and with my own microscopic study of corneal grafts both in animals and in human beings. I found, like Dr Thygeson, that epithelial cells can reproduce only epithelial cells and that connective tissue cells were produced by preexisting cells of the same nature.

Another point stressed by Dr Thygeson in his presentation was that the growth of tissue cultures was the same, whether the tissue was obtained from fetuses, stillborn infants or adults. This observation also disagrees with the clinical finding that transplants obtained from stillborn infants behaved in exactly the same manner as transplants obtained from adults' eyes.

Dr Thygeson deserves to be congratulated for his thorough and most interesting presentation.

RESULTS OF AUTOTRANSPLANTATION OF CORNEA INTO ANTERIOR CHAMBER THEIR SIGNIFICANCE REGARDING CORNEAL NUTRITION DR TRYGVE GUNDERSEN, Boston

This article appeared in full in the October issue of the ARCHIVES, page 645.

SOME PHARMACOLOGIC REACTIONS OF ISOLATED IRIS MUSCLES DR PARKER HEATH, Detroit

An experimental study was made of some of the actions of iris dilators and sphincters in animals. The method used consisted of a delicate suspension of isolated sphincter or dilator in solution, the changes were electrically amplified and then recorded by light beams on sensitized paper. Various stimuli and depressors were used, especially those classed as sympathetic and parasympathetic. Physiologic or spontaneous reactions were noted.

DISCUSSION

DR H. S. GRADLE, Chicago: About twelve years ago I worked on the time reaction of the pupil. I employed a similar method in the human being in the hope of establishing a more accurate timing of the pupillary reaction. The apparatus employed was a modification of the ordinary clinical electrocardiograph, and the method was similar to that described by Dr Heath. Interestingly enough, the electrodes were applied to both sides of the sclera (the difference in potential was what was desired), and the results were negative. One can obtain satisfactory graphs on the human subject by the use of nonpolarizing electrodes, which show clearly the electrical reactions of contraction of the sphincter and also of the dilator fibers. It is true that the stimulus used here was merely the stimulus of light, but different values for the sphincter and dilator effects were obtained which led to the impression that the dilator fibers are infinitely weaker than the sphincter. The experimental method described by Dr Heath is applicable to the living human eye, with the exception that side currents are set up which form almost uncontrollable reactions of the extraocular muscles which tend to cloud the issue. Consequently, Dr Heath's method is a marked advance in using the isolated iris muscle and in eliminating all extraneous influences.

ANISEIKONIA DR WALTER B LANCASTER, Boston

This article appears in full in this issue of the ARCHIVES, page 907

ANISEIKONIA A CLINICAL STUDY OF 836 PATIENTS DR CONRAD BERENS, New York

The data obtained in this study seem to indicate that correction of aniseikonia may be a factor in some cases in the relief of ocular complaints and general symptoms which apparently are not eliminated by the wearing of ordinary correcting lenses, orthoptic training and other forms of treatment. The correction for ametropia was changed in some cases, in others, accommodation seemed to be improved for some unknown reason. Muscle balance was undoubtedly changed in some instances, and fluctuations in the physical condition may have been coincident with the wearing of the iseikonic lenses, these and other factors should be taken into consideration in evaluating results.

DISCUSSION ON PAPERS BY DRs LANCASTER AND BERENS

DR EDWARD JACKSON, Denver. Studies on aniseikonia as reported in the papers of Drs Lancaster and Berens have far-reaching effects. They are close to the question of cerebral action and association, beyond the boundaries of what the great mass of people have learned to do with their eyes. Individual differences must be considered before the significance of the tables that were presented is understood.

DR HERMAN N BURIAN, Hanover, N D. I have studied the importance of peripheral fusional stimuli for the relative position of the two eyes, for the process of fusion is in itself a well rounded problem of physiologic optics, and its investigation was not undertaken with specific regard to aniseikonia. As a side result, however, I obtained, as Dr Lancaster pointed out, the conclusive proof that the measurements of artificially induced differences in size are identical, no matter whether they are taken with or without ocular movements. In other words, an actual difference in size was measured, not a possible anisophoria.

One has to assume that the relative difference in the sizes of the ocular images gives rise to difficulties in fusing the images of the two eyes. The result is that in persons affected with aniseikonia the constant struggle to bring about and maintain fusion produces the well known symptoms. One has to rely on the reports of the patients as to the relief they obtained by wearing an aniseikonic correction and from these reports to draw conclusions concerning the validity of one's assumption of the effectiveness of size corrections. There is, however, an objective sign which seems to me especially convincing as to the value of aniseikonic corrections for the achievement of perfect binocular vision. This is a fact that a marked improvement of depth perception can be obtained by the wearing of an aniseikonic correction. A number of intelligent patients who are good observers report spontaneously that they have noticed such an improvement or that they have experienced for the first time real stereoscopic three-dimensional vision. They often report in connection with this a considerable improvement in vision for fine, close work and for certain outdoor activities.

Objectively, this subjective impression of the patients has been checked with the Keystone chart, and it has been found that there actually was a marked improvement in their depth perception. If such patients do not wear their glasses for a few hours, their depth perception will be more or less reduced. Immediately after putting on the glasses, they show 100 per cent depth perception and do not lose it for a longer or shorter time after taking the glasses off.

Finally, I should like to mention a last point. Dr. Beiers has mentioned that in his cases there was no noticeable influence of aniseikonic corrections on the phorias of the patients. This is not astonishing, and it is evident that one cannot expect such an influence if one defines the phorias as the position of rest of the eyes. This position, so far as it is due to mechanical factors, cannot be expected to change. Under normal conditions of seeing it is not true that all innervational influences are excluded, and one can well imagine that it will be much easier for a patient with a considerable amount of phoria to overcome the anomaly in the position of rest if the process of fusion is facilitated by matching the images of the two eyes. This is probably another important factor which contributes to the great benefit some patients derive from the wearing of aniseikonic corrections.

ALEUKEMIC LYMPHOSIS INVOLVING THE UPPER LIDS. PATHOLOGIC CHANGES. DR. FREDERICK T. TOOKE, Montreal, Canada

The patient, an elderly, corpulent person, had heavy pendulous eyelids that were everted with difficulty. The palpebral conjunctiva was ischemic, appearing somewhat like moist blotting paper. The skin was not attached to what felt like a generalized involvement of the meibomian glands. The lids measured from 7 to 7.5 mm. A contrast measurement of a patient of the same body weight was 2 mm.

Clinical examination revealed a generalized glandular involvement, including the posterior cervical glands, the preauricular glands, the epitrochlear glands and the axillary and inguinal glands. Adiposity precluded palpation of the spleen. Biopsy was performed on the left upper lid and on one cervical gland.

Microscopic sections of lymph gland showed loss of normal structure due to an intense proliferation of closely packed, small, round lymphoid cells. The cells were uniform, tending to be a trifle angular in some areas with a suggestion of clinging to the reticulum. The picture presented corresponded to that found on the borderline between a lymphatic leukemia and a relatively mature form of lymphosarcomatosis. One is inclined to classify the condition among the katasplasias, i. e., as a rather immature form of lymphatic leukemia.

Sections from the eyelid showed densely packed lymphoid cells which were of medium size, they exhibited occasional mitotic figures and corresponded to the elements met with in the lymph gland described here. One also places the proliferation among the more immature forms of kataplastic processes, with an appreciation of the fact that it borders on a lymphosarcomatosis. The pathologic changes were similar to, though somewhat more immature than, those frequently described under the term "lymphatic leukaemia cutis."

The blood picture showed marked anemia, with a tendency for the cells to be somewhat larger than normal and of poor regenerative ability. The white blood cells showed a deficiency in the number of myeloid elements, with some immature forms. The lymphocytes were increased, and this increase appeared to be due to the presence of occasional lymphoblastic cells, similar to those met with in the lymph glands.

The blood picture was not typical of lymphatic leukemia or of lymphosarcomatosis. One is inclined to believe that the condition probably lies on the borderline between these two diseases. It is understandable that such an intermediate type might well occur when one considers the intimate relationship between these two conditions and the fact that the latter is simply a pathologic process of a more immature undifferentiated nature. For the purpose of classification the condition described here is probably best considered aleukemic lymphosis.

DISCUSSION

DR JOHN E WEEKS, Portland, Ore. Could the condition in this case be Mikulicz' disease, as described by Professor Mikulicz, of Breslau, Germany, some years ago?

DR EDWARD JACKSON, Denver. Although radiation therapy has been tried on the lymph glands in cases such as this, I should try the effect of vitamins, especially vitamin B.

DR JOHN E WEEKS, Portland, Ore. A case similar to that reported by Dr Tooke came under my observation a year and a half ago. I was surprised at the enlargement of the glandular elements and particularly of the eyelids themselves. Under irradiation the lymphomas entirely disappeared or were reduced in size at least during a period of about nine months, and the patient's general health appeared to improve.

DR FREDERICK T TOOKE, Montreal, Canada. In reply to Dr Weeks' question, I thought that the pathologic changes in Mikulicz' disease were much more definitely sarcomatous than those manifest in this case, also the blood picture as described here should answer Dr Weeks' question more clearly.

The suggestion of Dr Jackson regarding treatment might have been tried, but probably it would not have had much effect, considering the rapid decline of the patient from the time he came under observation.

ORBITAL MECHANICS AND LIMITATIONS AND MOVEMENT OF THE EYE, OPTIC NERVE, OBLIQUE MUSCLE AND MULLER'S MUSCLE WITH RELATION TO EXOPHTHALMOS, SEPARATION OF THE RETINA, THROMBOSIS AND POSTOPERATIVE MALAISE. DR CLIFFORD B WALKER, Los Angeles.

Several problems are encountered in work on the separated retina, the more notable of which follow: (1) absolute blindness immediately after traumatic separation which persists without interruption and (2) nausea and vomiting, apparently of the cerebral type, following more or less difficult operations in certain quadrants, with perhaps excessive tension on the optic nerve in spite of the fact that tenotomy is not performed.

Some of the factors involved, together with a consideration of the accessory function of the smooth and oblique muscles, deduced from a common type of retinal separation with the tear in the neighborhood of the attachment of the superior oblique muscle, can be studied to advantage in the light of orbital mechanics exhibited by models. Visualization of these mechanics has been found inefficient with large heavy models used almost exclusively heretofore. An attempt has been made, therefore, to make skeletonized or semitransparent models of exact size which are differentiated with color and enlarged by means of photoprojection and have various degrees of motion and tint.

Book Reviews

Methodik des optischen Raumsinnes und der Augenbewegungen.
Abderhalden's Handbuch der biologischen Arbeitsmethoden.
By Armin Tschermak-Seysenegg Section V, part 6, no. 10.
Pp. 325, with 88 illustrations Berlin. Urban and Schwarzenberg,
1937

Since F. B. Hofmann's contribution to Tigerstedt's "Handbuch der physiologischen Methodik" (1914), no comprehensive work concerning the methods used in the investigation of the sense of space and of the ocular movements has been published. This void is now filled by Armin Tschermak's exhaustive article, which is all the more welcome since the work of E. Hering has been developed and continued by this author more than by any other living physiologist. Nobody can compete with Tschermak's enormous theoretic knowledge and practical experience in the field under discussion. This also accounts for the facts that in his article theoretic discussions take up more space than one would expect to find in a book dedicated to the methods of investigation and that it gives in detail particularly the methods used and developed by that author himself, although all the other current methods are mentioned.

In an introductory chapter Tschermak gives a brief recapitulation of the basic ideas of what he has termed the exact subjectivism in the physiology of the senses, emphasizing the necessity of strict discrimination between the objective geometric distribution and the subjective impression of the position in space.

The first part of the work deals with the general methods in the investigation of the sense of space, the fixation of the head, the direction of gaze of the observer, etc. The author emphasizes especially the great advantage of self-recording devices, with the help of which, particularly of an appropriate punch, permanent records of the measurements can be obtained on paper without the necessity of reading the scales after each setting. A special chapter is devoted to the mathematical method of the theory of probabilities.

The special discussion of the sense of space is subdivided into the methods devised for the investigation of the perception of space in monocular and in binocular vision. To the first pertain the methods of determining the visual field; these methods are not treated exhaustively, since they have been considered in detail in other, mostly clinical, publications. It is, however, interesting to note that Tschermak reemphasizes the fact that the restriction of the color sense in indirect vision is purely relative and that one is not warranted in speaking of absolute color blindness in the periphery of the visual field. The relative localization in space without movements of the eyes is discussed more exhaustively. The theoretic significance of the various asymmetries in localization (angular discrepancies, discrepancies in direction, Kundt's partition experiment, the radial shrinkage of a test object in indirect vision, etc.), which play such an important role in the foundation of the subjectivistic conception of physiologic optics, is treated thoroughly, and the methods for their

determination are described. Tschermak's deductions from these discrepancies concerning a difference between the nodal projection center of the retina and the prenodal functional center as point of reference for a division of the retina are worthy of consideration. He emphasizes that the significance of the discrepancies in question can be evaluated properly only when it has been cleared up whether, and to what extent, they depend on asymmetries of the dioptric apparatus of the eyes.

The examination of the sensorial and motor orientation in space, i. e., the absolute and egocentric localization, disclination and conclination of the vertical and the horizontal meridian with various positions of the head and body and under the influence of acceleration, is discussed at length. Much space is taken up by interesting theoretic considerations, which are warranted because of their importance for the physiology of flying and for clinical examinations.

The methods concerned with the investigation of binocular space perception are subdivided into a number of chapters. The first chapter deals with the study of the haploscopic and stereoscopic effects produced without the use of instruments. The conception of the sensorial cyclopean eye is discussed and explained with the aid of a diagram. Of special importance for the clinician is the method wherein haploscopic and stereoscopic effects are achieved with the aid of colored (red and green) devices, the method of Haidinger's polarization brushes and, above all, the after-image method. The last method becomes especially important in the investigation of the visual act of squint, because it permits the determination of the retinal (sensorial) correspondence. Methods are described for measuring the angle of squint and examining the dominance of one eye by means of retinal rivalry.

A detailed description is given of the mirror haploscope, especially in the form developed by the author.

The methods for determining the corresponding retinal points, particularly those based on the determination of the apparent frontal plane ("horopter determinations"), are treated at length, and the instruments and their use are described. The methods based on other criteria (single appearance of a test object, constancy of the visual directions and maximal sensitivity to stereoscopic differences) are also taken into consideration. In this chapter mention is made of the influence which differences in size and shape of the ocular images (aniseikonia) have on the form of the horopter. The last chapter of the methods concerned with space perception deals with binocular depth perception. After an introduction giving the basic ideas concerning depth perception, there is a description of a number of arrangements and instruments for the investigation of this function. The various factors which influence stereopsis and have a bearing on the stereoscopic methods—time factor, factors concerning the localization of the images, dioptric factors and oculomotor factors—are discussed and their importance evaluated. The chapter on binocular vision closes with a discussion of secondary, empiric and interpretive movements which lead to the sensation of depth not based on vision with disparate retinal points.

The next part of the work deals with the investigation of movements of the eyes. The question of the center of rotation of the eyes and of its determination on the axes of rotation as well as characteristics

of the ocular muscles and the laws governing the movements of the single eye are discussed in detail. The special methods for testing the laws of Donders and Listing are presented, and particular emphasis is placed on the after-image method which Tschermak considers best and, in general, quite adequate. An exhaustive description is given of Tschermak's model illustrating Listing's law. For the examination of the voluntary movements of the eye, as they occur under the ordinary conditions of seeing, one has to resort to one of the various methods of photographic registration, which are described in full.

The discussion of the associated movements of the two eyes is begun with Hering's law of equal innervation of the oculomotor apparatus. This is followed by a discussion of the complex of accommodation, convergence and pupillary reaction, i. e., the adaptation of the eyes for near and for distant vision. Special emphasis is given to those movements which Tschermak designates as "adaptative movements." They bring the eyes comparatively slowly into a suitable tonic equilibrium, in which they remain for a certain length of time. These movements are (1) corrective movements, i. e., movements which tend to overcome differences in the oculomotor apparatus of the two eyes (heterophorias), (2) fusion movements, which are produced under artificial conditions (prisms, etc.) and serve the purpose of maintaining binocular single vision, and (3) static changes in the position of the eyes, the purpose of which is to maintain the normal position of the meridians of the eyes in spite of the influence which changes of the position of head or body have on the oculomotor apparatus.

For the investigation of the heterophorias, Tschermak describes, besides mentioning other methods, his own phorometer and his after-image method, which allows the determination of heterophorias with purely monocular fixation. The methods for investigating the fusion movements (with the haploscope or prisms) and the static and optokinetic changes in the position of the eyes are described briefly.

The work closes with a discussion of cinematographic devices, i. e., devices with the aid of which sensation of movement can be produced (stroboscope, strobostereoscope, moving pictures, Archimedes' spiral, Dvorak-Nach's phenomenon, Pulfrich's stereo-effect, etc.), and with a short mention of the methods for the production of optokinetic nystagmus.

In the scope of a short review it is impossible to do justice to Tschermak's work, with its abundance of knowledge, facts and valuable suggestions. Whoever is interested, or wants to do research work, in the field of space perception and the physiology of ocular movements will have to refer to Tschermak's work in the original.

A. BIELSCHOWSKY

Ottalmologia dei paesi caldi. By Prof. Vittoria Ruata, Italy. Cloth. Price, 45 lire. Pp. 362, with 92 illustrations and 4 colored plates. Milano: Ulrico Hoepli, 1938.

Vittorio Ruata, an associate at the Royal Eye Institute of the University of Rome, has written a concise, practical, theoretic volume of moderate size on tropical ophthalmology. Ruata has had the opportunity

of living and practicing in North Africa (Libya and Egypt) and of studying in other tropical countries. In the preface he states that he was urged to undertake this task because of the lack of a similar manual, not only in the Italian medical literature but in that of some other countries as well.

The author adheres strictly to his purpose—to be practical—referring to experimental theories only when he thinks that they may be of some help to the reader.

The volume is divided into nine chapters. In the first chapter Ruata describes the effects of climatic and atmospheric elements—wind, dust, light, etc.—on the eye in tropical countries, their consequences and their causal relationship to diseases of the conjunctiva or to the other structures of the eyeball.

After a review of the lesions caused by insects or by animal bites, the animal parasites which frequently cause ocular disease are listed.

As would be expected, the chapter on trachoma is the piece de résistance of the book. Ruata has had a great deal of experience with this problem in European countries and in Egypt, in which, he asserts, trachoma affects a greater percentage of the population than in other countries. The reader will find of interest the observation of the author that in Tunisia, Libya, Egypt and Ethiopia the black race is less affected by trachoma than the yellow or Mongolian race. While the native Egyptian population is affected nearly 100 per cent, as one goes into the Egyptian Sudan (where the population is mostly Negro) trachoma is found in only about 10 per cent of the population. Also, in Ethiopia and in Somaliland trachoma is almost absent among the natives, while on the opposite side of the Red Sea (where the population is mostly of Arabic blood) trachoma is found in almost 100 per cent of the population. For these reasons the author thinks that race rather than climate is the principal factor determining the incidence of trachoma in the tropics.

The last few chapters of the book are devoted to tropical diseases which affect the ocular adnexae.

In my opinion the author has succeeded in presenting a practical volume on tropical ophthalmology which should be a worthy addition to the library of any student of ophthalmology, whether or not he is practicing in tropical countries.

G BONACCOLTO

An Introduction to Clinical Perimetry By H. M. Traquair, M.D.,
Edinburgh. Third edition, revised and enlarged. Price, 30 shillings.
Pp. 320, with 227 illustrations and 3 colored plates. London:
Henry Kimpton, 1938.

Dr. Traquair's textbook on perimetry, which has become a classic, now appears in a third edition. In a foreword by Norman M. Dott, the neurosurgeon, the value of quantitative perimetry is emphasized, and it is pointed out that perimetry has now become an integral part of neurologic investigation, that speed of examination is particularly essential and that fatigue of the patient must always be reckoned with. The book is revised and enlarged, with 48 new illustrations, and new advances in this field are recorded. Simplicity of expression and the

demonstration of essentially practical methods remain outstanding features. It is one of Dr Traquair's great merits that the procedures which he advocates are simple, both as to instrument and as to technic, and within the reach of all and that it is not the elaboration of apparatus that is important but the understanding of the one who takes the field. Emphasis is justly laid on the interpretation and evaluation of the findings. The author believes that it is unnecessary to burden clinical procedures with elaborateness of doubtful practical value. Interpretation depends on knowledge of the anatomy of the visual paths and of the various ways in which different pathologic conditions act. Finally, as the examination is subjective, the psychic conditions which may be of influence must be recognized.

Ophthalmologists are indebted to Dr Traquair not only for a splendid work but for the admirable manner in which he has presented a difficult subject.

ARNOLD KNAPP

An Introduction to Clinical Scotometry By John N. Evans, M.D., F.A.C.S. Cloth Price, \$4. Pp. 266, with 57 illustrations, appendixes and bibliography index. New Haven, Conn. Yale University Press, 1938.

In the first six chapters of this book the author gives in detail the history of angioscotometry, a description of the equipment and the technic. In the remaining eight chapters he describes in detail the normal angioscotoma and compares it with angioscotomas found in primary ocular disease and with those associated with lesions of the nerve bundles. The appendixes contain much important data.

The author believes that an angioscotoma is not the actual measurement of vessel shadows but is a measure of the perivascular space. A hypothesis has been devised correlating the angioscotoma with the widening of the perivascular space which produces a dysfunction of the synapses in the retina due to oxygen deprivation. This theory is supported clinically in that although the retinal arterioles are narrowed by pressure on the globe, the angioscotoma becomes wider.

Angioscotomas produced by retinal edema, diseases of the nasal accessory sinuses, glaucoma, true neuritis and pseudoneuritis, intracranial vascular lesions, complicated pregnancy and retinal arteriosclerosis and those associated with strabismus are differentiated from those produced by variations in the normal eye.

As all defects of the visual field cannot be explained adequately by the angiogenic (perivascular space) hypothesis, the neurogenic (nerve fiber bundle) hypothesis has been considered and the respective defects differentiated. The author feels "that wedge-shaped defects with the apex pointing toward the blind spot are ocular and angiopathic in origin, but where the apex is toward the macula the lesion is primarily in the pathway. The picture is rendered atypical, however, where increased intracranial pressure is superimposed." This apparent argumentative point increases the value of the book, which is indispensable to the ophthalmologist who does not relegate the examinations of the visual fields to his secretary or office nurse.

The subject is presented completely in this book and is a challenge to ophthalmologists that better work in a generally neglected portion of an ocular examination can and should be done. This is best described by the author. "Had angioscotoma no other value in a diagnostic sense, its study would still be justified, because it provides a topographical outline which not only helps in the localization of various processes but also supplies a guide to show whether this or that area has been covered in a particular study. If one has succeeded in outlining a few vessel stumps about a blind spot, it is a guaranty that the blind-spot has been carefully and reliably mapped."

All ophthalmologists should own and study this book so that desired additional data will be available in the near future. The author is to be complimented for a work well done.

W I LILLIE, M D

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President Dr D D Sathaye, 127 Girgaum Road, Bombay 4, India
Secretary Dr H D Dastur, Dadar, Bombay 14, India
Place H B A Free Ophthalmic Hospital, Parel, Bombay 12 Time First
Friday of every month

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OXFORD OPHTHALMOLOGICAL CONGRESS

Hon Secretary-Treasurer Dr F A Anderson, 12 St John's Hill, Shrewsbury,
England

Time July 1939

PALESTINE OPHTHALMOLOGICAL SOCIETY

President Dr Arieh Feigenbaum, Abyssinian Str 15, Jerusalem
Secretary Dr E Sinai, Tel-Aviv

POLISH OPHTHALMOLOGICAL SOCIETY

President Dr W Kapuscinski, 2 Waly Batorego, Poznan
Secretary Dr J Sobanski, Lindley'a 4, Warsaw
Place Lindley'a 4, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President Dr Malcolm Hepburn, 111 Harley St, London, W 1, England
Secretary Dr C Dee Shapland, 15 Devonshire Pl, London, W 1, England

SOCIETE FRANÇAISE D'OPHTALMOLOGIE

Secretary Dr Rene Onfray, 6 Avenue de la Motte Picquet, Paris, 7^e

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President Prof K G Ploman, Stockholm
Secretary Dr K O Granstrom, Sodermalmstorg 4 III tr, Stockholm, So

TEL-AVIV OPHTHALMOLOGICAL SOCIETY

President Dr D Arieh-Friedman, 96 Allenby Str, Tel-Aviv, Palestine
Secretary Dr Sadger Max, 9 Bialik Str, Tel-Aviv, Palestine

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman Dr Eugene Chan, Cheeloo University School of Medicine, Tsinan,
Shantung, China
Place Cheeloo University School of Medicine Time Last Thursday of alter-
nate months

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON
OPHTHALMOLOGY

Chairman Dr S Judd Beach, 704 Congress St, Portland, Maine
Secretary Dr Derrick T Vail Jr, 441 Vine St, Cincinnati
Place St Louis Time May 15-19, 1939

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY,
SECTION ON OPHTHALMOLOGY

President Dr Harry S Gradle, 58 E Washington St, Chicago
Executive Secretary-Treasurer Dr William P Wherry, 1500 Medical Arts
Bldg, Omaha
Place Chicago Time Oct 8-14, 1939

AMERICAN OPHTHALMOLOGICAL SOCIETY

President Dr Frederick Tooke, 1482 Mountain St, Montreal, Canada
Secretary-Treasurer Dr Eugene M Blake, 303 Whitney Ave, New Haven, Conn
Place Hot Springs, Va

CANADIAN OPHTHALMOLOGICAL SOCIETY

President Dr S Hanford McKee, 1528 Crescent St, Montreal
Secretary-Treasurer Dr J A MacMillan, 1410 Stanley St, Montreal
Place Montreal Time June 19-23, 1939

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President Mr. William Fellowes Morgan, 50 W. 50th St, New York
 Secretary Miss Regina E Schneider, 50 W 50th St, New York

SECTIONAL

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr R O Ebert, 104 Main St, Oshkosh, Wis
 Secretary Dr G L McCormick, 626 S Central Ave, Marshfield
 Place Oshkosh Time May 1939

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President Dr Edwin B Goodall, 101 Bay State Rd, Boston
 Secretary-Treasurer Dr Trygve Gundersen, 243 Charles St, Boston
 Place Massachusetts Eye and Ear Infirmary, 243 Charles St, Boston Time
 8 p m, third Tuesday of each month from November to April, inclusive

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President Dr. F. C Cordes, 384 Post St, San Francisco
 Secretary-Treasurer Dr C Allen Dickey, 450 Sutter St, San Francisco
 Place San Francisco Time June 19-22, 1939

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr H L Goss, Cobb Bldg, Seattle
 Secretary-Treasurer Dr Purman Dorman, 1115 Terry Ave, Seattle
 Place Seattle or Tacoma, Wash Time Third Tuesday of each month, except
 June, July and August

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President Dr L J Friend, 425 E Grand Ave, Beloit, Wis
 Secretary-Treasurer Dr Thorsten E Blomberg, 501-7th St, Rockford, Ill
 Place Rockford, Ill, or Janesville or Beloit, Wis Time Third Tuesday of
 each month from October to April, inclusive

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Don M Howell, Alma, Mich
 Secretary-Treasurer Dr Louis D Gommon, 308 Eddy Bldg, Saginaw, Mich
 Place Saginaw or Bay City, Mich Time Second Tuesday of each month,
 except July and August

SIoux VALLEY EYE AND EAR ACADEMY

President Dr R A Kelly, 304 N Main St, Mitchell, S D
 Secretary-Treasurer Dr J C Decker, 515 Frances Bldg, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman Dr Grady E Clay, Medical Arts Bldg, Atlanta, Ga
 Secretary Dr John R Hume, 921 Canal St, New Orleans

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President Dr Carl Wencke, Battle Creek
 Secretary-Treasurer Dr A K Zinn, Battle Creek
 Time Third Thursday of alternate months

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr C M Harris, Johnstown
 Secretary-Treasurer Dr C Wearne Beals, 41 N Brady St, DuBois

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President A presiding officer is selected for each meeting alternately until all members have served

Secretary Dr John C Long, 324 Metropolitan Bldg, Denver
Place Capitol Life Bldg, Denver Time 7 30 p m, third Saturday of each month, October to May, inclusive

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR,
NOSE AND THROAT

President Dr William M Good, 63 Center St, Waterbury
Secretary-Treasurer Dr S J Silverberg, 201 Park St, New Haven

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President Dr Grady E Clay, 384 Peachtree St, N E, Atlanta
Secretary-Treasurer Dr J Mason Baird, 511 Medical Arts Bldg, Atlanta

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr C W Rutherford, 23 E Ohio St, Indianapolis
Secretary Dr Marlow W Manion, 23 E Ohio St, Indianapolis
Place Indianapolis Time First Wednesday in April

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr H H Lamb, American Bank Bldg, Davenport
Secretary-Treasurer Dr B M Merkel, 604 Locust St, Des Moines
Place Davenport

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr Francis E Le Jeune, 632 Maison Blanche Bldg, New Orleans
Secretary-Treasurer Dr Edley H Jones, 1301 Washington St, Vicksburg, Miss
Place Gulfport, Miss Time May 8, 1939

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY
AND OTOLARYNGOLOGY

Chairman Dr B Fralick, 201 S Main St, Ann Arbor
Secretary Dr O McGillicuddy, 124 W Allegan St, Lansing

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Frank N Knapp, 318 W Superior St, Duluth
Secretary-Treasurer Dr George E McGear, 920 Medical Arts Bldg, Minne
apolis
Time Second Friday of each month from October to May

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President Dr Roy Grigg, Bozeman
Secretary Dr A W Morse, 507 Phoenix Bldg, Butte

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY,
OTOLOGY AND RHINOLARYNGOLOGY

Chairman Dr Norman W Burritt, 30 Beechwood Rd, Summit
Secretary Dr A Russell Sherman, 671 Broad St, Newark
Place Atlantic City Time June 1939

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman Dr Algernon B Reese, 73 E 71st St, New York
Secretary Dr Chester C Cott, 333 Linwood Ave, Buffalo

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr J M Lilly, 302 Old St, Fayetteville
 Secretary-Treasurer Dr Frank C Smith, 106 W 7th St, Charlotte

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr N A Youngs, 322 De Mers Ave, Grand Forks
 Secretary-Treasurer Dr F L Wicks, 516-6th St, Valley City
 Place Fargo Time May 1939

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr L O Clement, 406 State St, Salem
 Secretary-Treasurer Dr Paul Bailey, 833 S W 11th Ave, Portland
 Place Good Samaritan Hospital, Portland Time Third Tuesday of each month

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President Dr N Darrell Harvey, 112 Waterman St, Providence
 Secretary-Treasurer Dr Linley C Happ, 124 Waterman St, Providence
 Place Rhode Island Medical Society Library, Providence Time 8 30 p m,
 second Thursday in October, December, February and April

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr S B Fishburne, 1430 Marion St, Columbia
 Secretary Dr J W Jervey Jr, 101 Church St, Greenville
 Place Columbia Time Nov 1, 1938

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr Kate Savage Zerfoss, 165-8th Ave, N, Nashville
 Secretary-Treasurer Dr W D Stinson, 805 Medical Arts Bldg, Memphis

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President Dr T E Fuller, 100 W Board St., Texarkana
 Secretary Dr O M Marchman, 1719 Pacific Ave, Dallas

UTAH OPHTHALMOLOGICAL SOCIETY

President Dr V P White, 143½ S Main St, Salt Lake City
 Secretary-Treasurer Dr E B Fairbanks, Boston Bldg, Salt Lake City
 Time Third Monday of each month

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President Dr Charles T St Clair, 418 Bland St, Bluefield, W Va
 Secretary-Treasurer Dr M H Williams, 30½ Franklin Rd, S W, Roanoke

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE
 AND THROAT SECTION

President Dr George Traugh, 309 Cleveland Ave, Fairmont
 Secretary Dr Welch England, 621½ Market St, Parkersburg

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON
 EYE, EAR, NOSE AND THROAT

President Dr Andrew Rados, 31 Lincoln Park, Newark
 Secretary Dr William F McKim, 317 Roseville Ave, Newark
 Place 91 Lincoln Park South, Newark Time 8 45 p m, second Monday of
 each month, October to May

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr L E Brown, Second National Bldg, Akron, Ohio
 Secretary-Treasurer Dr C R Anderson, 106 S Main St, Akron, Ohio
 Time First Monday in January, March, May and November

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President Dr J Mason Baird, Medical Arts Bldg, Atlanta, Ga
 Secretary Dr Alton V Hallum, 478 Peachtree St, Atlanta, Ga
 Place Academy of Medicine, 38 Prescott St Time Second Friday of each month from October to May

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

Chairman Dr Frank B Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore
 Secretary Dr Fred M Reese, 6 E Eager St, Baltimore
 Place Medical and Chirurgical Faculty, 1211 Cathedral St Time 8 30 p m, fourth Thursday of each month from October to May

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President Dr E Clifford Place, 59 Livingston St, Brooklyn
 Secretary-Treasurer Dr Frank Mallon, 1135 Park Pl, Brooklyn
 Place Kings County Medical Society Bldg, 1313 Bedford Ave Time Third Thursday in February, April, May, October and December

BUFFALO OPHTHALMOLOGIC CLUB

President Dr Ivan J Koenig, 40 North St, Buffalo
 Secretary-Treasurer Dr Meyer H Riwchun, 367 Linwood Ave, Buffalo
 Time Second Thursday of each month

CHATTANOOGA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Each member, in alphabetical order
 Secretary Dr A H Benz, 706 Medical Arts Bldg, Chattanooga, Tenn
 Place Mountain City Club Time Second Thursday of each month from September to May

CHICAGO OPHTHALMOLOGICAL SOCIETY

President Dr Georgiana Dvorak-Theobald, 715 Lake St, Oak Park, Ill
 Secretary-Treasurer Dr Earle B Fowler, 55 E Washington St, Chicago
 Place Medinah Michigan Avenue Club, 505 N Michigan Ave Time Third Monday of each month from October to May

CINCINNATI OPHTHALMIC CLUB

Chairman Each member, in rotation
 Secretary-Treasurer Dr E R Thomas, 819 Carew Tower, Cincinnati
 Place Holmes Memorial Library, Cincinnati General Hospital Time 8 15 p m, third Monday of each month except June, July and August

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman Dr Paul Moore, Republic Bldg, Cleveland
 Secretary Dr G Leslie Miller, 14805 Detroit Ave, Cleveland
 Time Second Tuesday in October, December, February and April

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman Dr Alexander G Fewell, 1924 Pine St, Philadelphia
 Clerk Dr W S Reese, 1901 Walnut St, Philadelphia
 Time Third Thursday of every month from October to April, inclusive

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman Dr Hugh G Beatty, 150 E Broad St, Columbus, Ohio
 Secretary-Treasurer Dr W A Stoutenborough, 21 E State St, Columbus, Ohio
 Place Deshler Wallick Hotel Time 6 p m, first Monday of each month

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman Dr Edgar G Mathis, 416 Chaparral St, Corpus Christi, Texas
 Secretary Dr E King Gill, 416 Chaparral St, Corpus Christi, Texas
 Time Second Thursday of each month from October to May

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr Lester H Quinn, 4105 Live Oak, Dallas, Texas
 Secretary Dr J Dudley Singleton, 1719 Pacific Ave, Dallas, Texas
 Place Dallas Athletic Club Time 6 30 p m, first Tuesday of each month
 from October to June The November, January and March meetings are
 devoted to clinical work

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President Dr E G Linn, 604 Locust St, Des Moines, Iowa
 Secretary-Treasurer Dr Grace Doane, 614 Bankers Trust Bldg, Des Moines,
 Iowa
 Time 7 45 p m, third Monday of every month from September to May

DETROIT OPHTHALMOLOGICAL CLUB

Chairman Members rotate alphabetically
 Secretary Dr Arthur S Hale, 1609 Eaton Tower, Detroit
 Time 6 30 p m, first Wednesday of each month

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President Dr L A Hulsebosch, 191 Glen St, Glen Falls
 Secretary-Treasurer Dr Joseph L Holohan, 330 State St, Albany
 Time Third Wednesday in October, November, March, April, May and June

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President Dr R A Gough, 602 W 10th St, Fort Worth, Texas
 Secretary-Treasurer Dr Charles R Lees, 806 Medical Arts Bldg, Fort Worth,
 Texas
 Place Medical Hall, Medical Arts Bldg Time 7 30 p m, first Friday of each
 month except July and August

GRAND RAPIDS EYE, EAR, NOSE AND THROAT SOCIETY

President Dr Dewey R Heetderks, 405 Medical Arts Bldg, Grand Rapids, Mich
 Secretary-Treasurer Dr Robert G Laird, 116 E Fulton St, Grand Rapids, Mich
 Place Various local hospitals Time Third Thursday of alternating months,
 September to May

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND
 THROAT SECTION

President Dr Louis Daily, 1215 Walker Ave, Houston, Texas
 Secretary Dr Herbert H Harris, 1004 Medical Arts Bldg, Houston, Texas
 Place Medical Arts Bldg, Harris County Medical Society Rooms Time
 8 p m, second Thursday of each month from September to June

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr J K Leasure, 23 E Ohio St, Indianapolis
 Secretary Dr Kenneth L Craft, 23 E Ohio St, Indianapolis
 Place University Club Time 6 30 p m, second Thursday of each month
 from October to June

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr E N Robertson, Concordia, Kan
 Secretary Dr John S Knight, 1103 Grand Ave, Kansas City, Mo
 Time Third Thursday of each month from October to June The November,
 January and March meetings are devoted to clinical work

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman Dr Ben K Parks, 619 Professional Bldg, Long Beach, Calif
 Secretary-Treasurer Dr Paul Nilsson, 211 Cherry Ave, Long Beach, Calif
 Place Professional Bldg Time Last Wednesday of each month from October
 to May

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr Clifford B Walker, 427 W 5th St, Los Angeles
 Secretary-Treasurer Dr John P Lordan, 2007 Wilshire Blvd, Los Angeles
 Place Los Angeles County Medical Association Bldg, 1925 Wilshire Blvd Time
 6 30 p m, fourth Monday of each month from September to May, inclusive

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President Dr Gaylord C Hall, Brown Bldg, Louisville, Ky
 Secretary-Treasurer Dr Charles K Beck, Starks Bldg, Louisville, Ky
 Place Brown Hotel Time 6 30 p m, second Thursday of each month from
 September to May, inclusive

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF
OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman Dr Earle Breeding, 1801 I St, N W, Washington
 Secretary Dr Elmer Shepherd, 1606-20th St, N W, Washington
 Place 1718 M St, N W Time 8 p m, third Friday of each month from
 October to April, inclusive

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman Each member, in alphabetical order
 Secretary Dr Sam H Sonders, Medical Arts Bldg, Memphis, Tenn
 Place Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital Time
 8 p m, second Tuesday of each month

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President Dr John E Mulsow, 231 W Wisconsin Ave, Milwaukee
 Secretary-Treasurer Dr John B Hitz, 411 E Mason St, Milwaukee
 Place University Club Time 6 30 p m, second Tuesday of each month

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman Each member, in alphabetical order
 Secretary Dr M C Pfunder, 645 Medical Arts Bldg, Minneapolis
 Place Hennepin County Medical Society rooms Time 6 30 p m, fourth
 Monday of each month, October to May, inclusive

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman Dr P H Kilbourne, Fidelity Bldg, Dayton, Ohio
 Secretary-Treasurer Dr Martland D Place, 981 Reibold Bldg, Dayton, Ohio
 Place Van Cleve Hotel Time 6 30 p m, monthly, first Tuesday from October
 to June, inclusive

MONTREAL OPHTHALMOLOGICAL SOCIETY

President Dr L de G Joubert, 690 Dunlop St, Montreal, Canada
 Secretary Dr K B Johnston, 1509 Sherbrooke St, W, Montreal, Canada
 Time Second Thursday of October, December, February and April

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman Dr Guy Maness, 119-7th Ave, Nashville, Tenn
 Secretary-Treasurer Dr Andrew Hollabaugh, Doctors Bldg, Nashville, Tenn
 Place St Thomas Hospital Time 8 p m, third Monday of each month from
 October to May

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President Dr E G Walls, 619 Maison Blanche Bldg, New Orleans
 Secretary-Treasurer Dr W B Clark, 1012 American Bank Bldg, New Orleans.
 Place Louisiana State University Medical Bldg Time 8 p m, second Tuesday
 of each month from October to June

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman Dr James W White, 15 Park Ave, New York
 Secretary Dr Rudolf Aebli, 30 E 40th St, New York
 Time 8 30 p m, third Monday of every month from October to May, inclusive

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President Dr Percy Fridenberg, 38 W 59th St, New York
 Secretary Dr David Alperin, 889 Park Place, Brooklyn
 Place Squibb Hall, 745-5th Ave Time 8 p m, first Monday of each month
 from October to May, inclusive

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND
 OTO-LARYNGOLOGICAL SOCIETY

President Dr Philip Romonek, 107 S 17th St, Omaha
 Secretary-Treasurer Dr W Howard Morrison, 1500 Medical Arts Bldg, Omaha
 Place Omaha Club, 20th and Douglas Sts, Omaha Time 6 p m, dinner,
 7 p m, program, third Wednesday of each month from October to May

OPHTHALMOLOGICAL SOCIETY OF THE UNIVERSITY OF PITTSBURGH

President Dr W W Blair, 121 University Pl, Pittsburgh
 Secretary Dr George H Shuman, 351-5th Ave, Pittsburgh
 Time Second Monday in November, January, March and May

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President Dr R N Berke, 430 Union St, Hackensack, N J
 Secretary-Treasurer Dr T A Sanfacon, 340 Park Ave, Paterson, N J
 Place Paterson Eye and Ear Infirmary Time 9 p m, last Friday of every
 month, except June, July and August

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman Dr Walter I Lilie, 255 S 17th St, Philadelphia
 Secretary Dr Edmund B Spaeth, 1930 Chestnut St, Philadelphia
 Time First Thursday of each month from October to May

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President Dr Edward Stieren, Union Trust Bldg, Pittsburgh
 Secretary Dr George H Shuman, 351-5th Ave, Pittsburgh
 Place Pittsburgh Academy of Medicine Bldg Time Fourth Monday of each
 month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President. Dr N. H Turner, 200 E Franklin St, Richmond, Va
 Secretary. Dr Richard W Vaughan, Medical Arts Bldg, Richmond, Va
 Place Westmoreland Club Time 6 p m, second Monday of each month from
 October to May

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President Dr R E Elliott, 78 S Fitzhugh St, Rochester, N Y
 Secretary-Treasurer Dr Raphael Farber, 280 Monroe Ave, Rochester, N Y
 Place Rochester Medical Association, 113 Prince St Time 8 p m, third
 Monday of each month from October to May

ST LOUIS OPHTHALMIC SOCIETY

President Dr Roy E Mason, Frisco Bldg, St Louis
 Secretary Dr Leslie Charles Drews, 508 N Grand Blvd, St Louis
 Place Oscar Johnson Institute Time Clinical meeting 5 30 p m, dinner and
 scientific meeting 6 30 p m, fourth Friday of each month from October to
 April, inclusive, except December

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President Dr Oscar H Judkins, 414 Navarro St, San Antonio, Texas
 Secretary-Treasurer Dr Wilfred E Muldoon, 414 Navarro St, San Antonio,
 Texas
 Place Bexar County Medical Library Time 8 p m, first Tuesday of each
 month from October to May

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE,
EAR, NOSE AND THROAT

Chairman Dr Russell Fletcher, 490 Post St, San Francisco
 Secretary Dr Avery Morley Hicks, 490 Post St, San Francisco
 Place Society's Bldg, 2180 Washington St, San Francisco Time Fourth
 Tuesday of every month except May, June, July and December

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President Dr R R Kirkpatrick, 6th and Walnut Sts, Texarkana, Ark
 Secretary-Treasurer Dr W L Atkins, 940 Margaret Pl, Shreveport, La
 Place Shreveport Charity Hospital Time 7 30 p m, first Monday of every
 month except July, August and September

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President Dr P B Greene, 422 Riverside Ave, Spokane, Wash
 Secretary Dr O M Rott, 421 Riverside Ave, Spokane, Wash
 Place Paulsen Medical and Dental Library Time 8 p m, fourth Tuesday of
 each month except June, July and August

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President Dr James F Cahill, 428 S Salina St, Syracuse, N Y
 Secretary-Treasurer Dr I Herbert Katz, 713 E Genesee St, Syracuse, N Y
 Place University Club Time First Tuesday of each month except June, July
 and August

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman Dr A Lloyd Morgan, 170 St George St, Toronto, Canada
 Secretary Dr W R F Luke, 170 St George St, Toronto, Canada
 Place Academy of Medicine, 13 Queen's Pk Time First Monday of each
 month, November to April

WASHINGTON, D C, OPHTHALMOLOGICAL SOCIETY

President Dr G Victor Simpson, 1710 Rhode Island Ave, N W, Washington,
 D C
 Secretary-Treasurer Dr Frank D Costenbader, 1726 I St, Washington, D C
 Place Episcopal Eye, Ear and Throat Hospital Time 8 p m, first Monday
 in November, January, March and May

INDEX TO VOLUME 20

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O and S, respectively.

Abnormalities and Deformities See under names of diseases and organs, as Acrocephaly, Eyes, absence of, Fingers and Toes, abnormalities, etc

Abscess See under names of organs and regions, as Cornea, abscess, etc

Accommodation and Refraction See also Astigmatism, Glasses, Myopia, etc
absorption of visible light by refractive media of human eye, *37
anisikonla, *907
anisikonla, clinical study of 836 patients, 1109
anisikonla in emmetropia, 1091
benzedrine in cycloplegia, 858
constitution of refracted light beam in accommodated eye, 670
dark adaptation and its differential diagnostic value in cases of neuritis and choked disk, 121
determination and significance of scotopic retinal visibility curve, *713
evaluation of homatropine-benzedrine cycloplegia, *585
induced size effect, new phenomenon in binocular space perception associated with relative sizes of images of 2 eyes, *604
intracapsular accommodation of lens, 513
nature and management of heterophorias, *375
neural correlations of vision and their significance for localization of tumors of brain, 509
ocular dominance, 105
physiologic and clinical ophthalmologic problems in relation to individual variability, *541, *726, *913
shall we use cycloplegics? 1099
simple accessory for dark room, 509
study of diet in relation to health, dark adaptation as index of adequate vitamin A intake, 512
testing fitness for night flying visual acuity, *58
use of paredrine in cycloplegia, 156, correction, 505
vertical prism values in commonly used bifocal lenses *95
visual defects in 11,021 school children of Milan, 671
yearly changes in refraction from birth to beyond middle life, 145

Acetylcholine See Choline and Choline Derivatives

Acid Ascorbic See Vitamins, C
Cevitamic See Vitamins, C
Phosphoric See Phosphorus and Phosphorus Compounds

Acrocephaly, dislocation of eyeball as complication of ocephaly, 330

Adaptometer See Accommodation and Refraction

Adie Syndrome See Reflex, pupillary

Agatston, S A Simple surgical procedure for relief of spastic entropion, 148
Unilateral retinitis pigmentosa, report of 2 cases, 680

Age, Old See Old age

Air, sea air and ophthalmology, 135

Alcohol, acute alcoholic amaurosis, *797

Alexander, R C Conjunctivitis associated with infection by *Streptococcus viridans*, clinical and bacteriologic observations in epidemic, *19

Alger, E M John Martin Wheeler, 885

Allen, T D Detachment of retina, summary of modern opinions, *307
Homonymous hemianopic paracentral scotoma, report of case, *846

Allergy See Anaphylaxis and Allergy

Amaurosis See Blindness

Amblyopia See Blindness

Ametropia See Myopia

Amsler, M Incipient (fruste) keratoconus, 124

Anaphylaxis and Allergy, allergic reaction of trachoma, 675
bilateral superficial lesions of cornea due to allergy, report of case, 334

Anesthesia, hypersensitivity to larocaine, *474

Aneurysm, aneurysmal dilatation of ophthalmic artery, clinical course, 133

Angioid Streaks See Retina, pathology

Angioma, angiomatosis retinae, report of case with pathologic study of enucleated eye, *958
juvenile glaucoma with angiomatosis of retina, 665

Angioscopy See Retina, blood supply

Angioscotometry See Scotoma

Angiospasm See Retina, blood supply

Anisikonla See Accommodation and Refraction

Anisocoria See under Pupils

Anomalies See under names of organs and regions, as Acrocephaly, Eyes, absence of, Fingers and Toes, abnormalities, etc

Anophthalmos See Eyes, absence of

Anthrax, attempts to develop in vitro oculo-tropism of avirulent anthrax bacillus and of *Proteus vulgaris*, 659

Antigens and Antibodies, antigenic properties of cornea and vitreous, 850

Apparatus See also Instruments
absorption of visible light by refractive media of human eye, *37
combined short wave and ionization apparatus, 146
for measuring and recording disturbances of ocular motility, 145
new, for recording depth perception, 678
ophthalmic carriage, *286

Aqueous Humor, circulation of aqueous, mechanism of secretion of intraocular fluid, *761
nature of, 331
osmotic pressure of aqueous humor in epidemic dropsy glaucoma, 853
permeability of capsule of lens and dextrose content of blood, aqueous and lens in animals with galactose cataract, 321

Arachnodactylia See Fingers and Toes, abnormalities

Arachnoid, opticochiasmic arachnoiditis, 127
optochiasmic arachnoiditis due to pneumococcus, 1090

INDEX TO VOLUME 20

- Arachnoid**—Continued
 optochiasmic arachnoiditis, importance of mixed type of atrophy of optic nerve as diagnostic sign, *384
 syphilitic optochiasmic arachnoiditis and pyretotherapy, 130
 syphilitic optochiasmic arachnoiditis 1103
- Arsphenamines**, temporary spasmodic myopia due to, 513
- Arteries** See Aneurysm, Blood pressure
 Thrombosis, etc
 Retinal See Retina, blood supply
- Ascorbic Acid** See Vitamins, C
- Astigmatism**, operative treatment of 143
 theory and use of cross cylinders 870
- Atrophy** See under names of organs and regions, as Cavernous Sinus, Nerves, optic, Retina, etc
- Autohemotherapy, Autoserotherapy** See Sero-therapy and Hemotherapy
- Aviation**, injuries attributed to glasses of aviators, 854
 testing fitness for night flying visual acuity, *58
- Avitaminosis** See under Vitamins and under names of deficiency diseases
- Bacilli** See Bacteria
- Bacteria** See also Staphylococci, Streptococci
 Anthrax See Anthrax
 Granulosis See Trachoma
 proteus group, attempts to develop in vitro oculotropism of virulent anthrax bacillus and of *Proteus vulgaris*, 659
- Bailliant** Examination of retinal vessels by dynamometry, 138
 Social service in ophthalmic hospitals, 148
- Beach, S J** Ophthalmology comes of age years 1908-1915, up to establishment of examining board, 1106
- Bedell A J** Traumatic retinal angiopathy 1107
- Bellows, J** Biochemistry of lens, effect of galactose on permeability of capsule of lens, *80
 Biochemistry of lens, studies on glutathione in crystalline lens, *417
 Use of sorbitol in glaucoma, *1036
- Benzedrine**, evaluation of homotropine benzedrine cycloplegia, *585
 in cycloplegia, 858
- Berens, C** Anisokonia, clinical study of 836 patients 1109
 Postoperative complication of cataract operations, 875
- Berger, A R** Conjunctivitis associated with infection by *Streptococcus viridans*, clinical and bacteriologic observations in epidemic *19
- Berner D E** Reading difficulties in children *829
- Berner, G E** Reading difficulties in children, *829
- Biedl-Laurence Syndrome** See Laurence-Biedl Syndrome
- Bielschowsky, A** Disturbances of vertical motor muscles of eyes, *175
- Bietti** Blood calcium in cases of juvenile cataract, 144
 Use of interferometer for estimation of endocrine function in ocular diseases, 142
- Biomicroscopy** postgraduate course in, 1073
- Birge, H L** Cancer of eyelids, conjunctiva and cornea, squamous cell epithelioma *254
- de Blaskovics, Ladislav** 1081
- Blatt** Cataract in relation to dysfunction of hypophysis 143
- Blepharitis** See Eyelids
- Blepharochalasis** See Eyelids diseases
- Blepharoplasty** See under Eyelids
- Blepharoptosis** See Eyelids, ptosis
- Blindness** See also Vision, etc
 acute alcoholic amaurosis, *797
 Color See Color Blindness
 discussion of national policy to be adopted in tropical country for prevention of, 147
 histologic examination of cerebrum in unilateral peripheral blindness, report of 2 cases, 116
 in infants without ophthalmoscopic changes (congenital forms), 133
 instruction and professional orientation of amblyopic children, creation of special schools 1087
 National Society for Prevention of annual meeting 1071
 of hypophyseal origin, 4 cases of adiposogenital syndrome with retinal degeneration and mental backwardness, 328
 pathologic changes in amblyopia following trypanamide therapy, 867
 prevention in tropical country, 147
 responsibility of ophthalmologist in conservation of sight and prevention of, 148
 role of social worker in program for prevention of, 148
 social aspect of prevention of 147
 social service in ophthalmic hospital 148
 spread in Dutch East Indies, 147
 struggle against blindness and new law of public assistance in Bulgaria, 147
- Blood**, alkaline reserve in idiopathic retinal detachment 859
 calcium in cases of juvenile cataract 144
 coagulation, advantages of use of coagulants in ocular operations especially in extraction of cataract and in plastic operations *406
- Diseases** See Leukemia
 permeability of capsule of lens and dextrose content of blood aqueous and lens in animals with galactose cataract 321
 pressure, arterial hypertension of retina, 137
 pressure arterial hypotony and retinal atrophy, 138
 pressure effect of extract of ciliary body on, 512
 pressure, high, autonomic hypertension 138
 pressure, high, diagnostic significance of retinal changes in cases of hypertension, 140
 pressure high, experimental hypertension 140
 pressure, high experimental hypertension, vascular changes in eyes *812
 pressure, high, histologic changes in eyes of patients with hypertension 138
 pressure, high, importance of ocular examination for understanding of hypertension and nephritis, 139
 pressure, high, latent processes in visual pathways in cases of hypertension 139
 pressure, high, significance of transitory arterial hypertension in genesis of retinal changes 138
 pressure, influence of light on arterial tension, 322
 pressure, variations in pressure of retinal arteries and in capillary pressure in relation to compression of globe, 859
 transfusion in uveitis 677
- Bogart D W** Intraocular foreign body report of case, 692
 Postoperative complication of cataract operations 875

INDEX TO VOLUME 20

- Bollack, I Opticohiasmic arachnoiditis, 127
- Bonaccollo, G Operation for congenital ptosis, 692
- Bones, growth, dysostosis multiplex with consideration of ocular condition, report of case, 322
- orbital hyperostosis, its occurrence in 2 cases of meningioma of skull, *996
- BOOK REVIEWS
- Bulletin of Ophthalmological Society of Egypt, 164
- Child's Graded Reading Book for Eye Specialists, H R Nesburn and D L Risley, 697, correction, 896
- Citric Acid Studies Referring to Eye, H Gronvall, 530
- Contribution from Eye Clinic of Odessa Medical Institute in Honor of Director Prof V P Filatov (Honorary Scientific Worker of U S S R), on His 60th Birthday, 161
- Diseases of Nervous System in Infancy, Childhood and Adolescence, F R Ford, 162
- Eingriffe am Auge, L von Blaskovics and A Kreiker, 893
- Eleventh Annual Report of Memorial Ophthalmic Laboratory, 528
- Fundus oculi Diagnostica oftalmoscopica, Q Di Marzo, 894
- Hypokizamide und Linse, A Meesmann 691
- Introduction to Clinical Perimetry, H M Triquair, 1116
- Introduction to Clinical Scotometry, J N Evans 1117
- Introduction to Mathematics of Ophthalmic Optics, P Boeder, 892
- Introduction to Ophthalmology, P C Kronfeld, 695
- Médications de choc en ophtalmologie, L Hambresin, 894
- Meningiomes supra-sellaires Diagnostic du syndrome chiasmatique, L Guillaumat, 895
- Methodik des optischen Raumsinnes und der Augenbewegungen Abderhalden's Handbuch der biologischen Arbeitsmethoden, A Tschermak-Seysenegg 1113
- Neuro-Ophthalmology, R L Rea, 893
- Oftalmologia del pnest child, V Ruzita, 1115
- Patient and Weather, W F Petersen with assistance of M E Milliken, 162
- Primer Congreso argentino de oftalmologia, 163
- Proceedings of All-India Ophthalmological Society, 697
- Stereoskopische Bilder fur schielende Kinder, C H Sattler, 348
- Studies on Physiology of Eye, J G Byrne, 164
- Text-Book of Ophthalmic Operations, H Grimsdale and E Brewerton, 344
- Textbook of Ophthalmology, S R Gifford, 163
- Text-Book of Ophthalmology, W S Duke-Elder, 159
- Untersuchungen uber die Augenhohlen des Menschen in verschiedenen Lebensaltern, P Pallin, 346
- Vitamine und Hormone in ihren Beziehungen zur Augenheilkunde (Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten no 25), K Wachholder, W Kyrieleis and R Braun, 347
- Borley, W E Influence of vitamins and dimittrophanol on production of experimental cataract, *30
- Bourdeaux Surgical treatment of glaucoma 126
- Bourneville-Pringle's Disease See Sclerosis, tubercous
- Bowen's Disease See Cancer, precancer
- Bowman Membrane See under Cornea
- Brain See also Meninges, Nervous System etc
- histologic examination of cerebrum in unilateral peripheral blindness, report of 2 cases, 116
- relation of sensory visual cortex to oculomotor cortex, 510
- tumors, neural correlations of vision and their significance for localization of tumors of brain, 509
- tumors pseudo albuminuric retinitis in cases of, 332
- tumors, studies of visual fields in cases of verified tumor of brain *137
- value of functional visual tests for localization of supratentorial tumors of brain, preliminary report, 524
- Brainstem Cataract in relation to dysfunction of hypophysis, 143
- Brown, E V L Yearly changes in refraction from birth to beyond middle life, 145
- Bruckner, A Physiologic and clinical ophthalmologic problems in relation to individual variability *511, *726, *913
- Bruning Graduation of Baillart and Sobanski dynamometers on human cadaver, 143
- Bucklers Causation of zonular cataract, 144
- Buphthalmos See Hydrophthalmos
- Burlan, H Fusional movements, role of peripheral retinal stimuli, 873
- Busacca, A Advantages of use of coagulants in ocular operations especially in extraction of cataract and in plastic operations, *406
- Cerebrospinal pressure in cases of retinitis, 140
- Development of trachoma in cornea, 147
- Research studies of eye, some technical and practical notes *395
- Cadavers graduation of Baillart and Sobanski dynamometers on human cadaver, 143
- transplantation of conjunctiva from cadaver's eye, 117
- Calcium content of crystalline lens in experimental parathyroparal cataract, 666
- in Blood See Blood, calcium
- Calhoun, F P John Martin Wheeler, 885
- Cancer See also Epithelioma, Neurocarcinoma, Sarcoma, Tumors, and under names of organs and regions, as Conjunctiva, Eyelids, Lacrimal Organs, etc
- metastases, differences in manner of perforation of sclera caused by primary melanoma and metastatic carcinoma of uveal tract, 676
- precancer, precancerous melanosis of lids and conjunctiva, 683
- Capillaries, isoperfusion and anisoperfusion of capillaries in cases of retinal hypertension, 140
- resistance to pressure in ocular tuberculosis, 126
- Carbon disulfide poisoning, *839
- Carman, H F, Jr Homonymous hemianopia paracentral scotoma, report of case, *846
- Carriage, ophthalmic, *286
- Carris Role of social worker in program for prevention of blindness, 148
- Carroll, F D Acute alcoholic amaurosis, *797
- Caruncle, Lacrimal See Lacrimal Organs
- Case, P H Intern's experiences with Verhoeff method of cataract extraction, *651
- Cataract, action of vitamin C on 142
- advantages of use of coagulants in ocular operations especially in extraction of cataract and in plastic operations, *406

INDEX TO VOLUME 20

- Cataract**—Continued
 and latent tetany, 325
 biochemistry of lens, effect of galactose on permeability of capsule of lens, *80
 calcium content of crystalline lens in experimental parathyroidal cataract, 666
 diabetic and tetanic cataract in relation to Krauss' theory of muscle function of cortical cells, 145
 due to dinitrophenol, 125
 due to electric shock, 113
 experimental dinitrophenol cataract in animals, 321
 experimental, influence of vitamins and dinitrophenol on production of, *30
 extraction corneo-scleral suture, its technic and advantages, 856
 extraction Intern's experiences with Verhoeff method *651
 extraction, psychoses complicating recovery from 324
 formation after reduction of weight with dinitro-bodies, 325
 galactose cataract, clinical observations, 506
 pH of lens in naphthalene cataract of rabbit, 855
 in relation to dysfunction of hypophysis, 143
 inferior iridotomy in operations for cataract on eyes with posterior synechiae or pupillary membrane, value of operation, *641, 1105
 intracapsular expression of, 855
 juvenile, blood calcium in cases of 144
 metabolism of lens after parathyroidectomy 113
 new capsule-grasping forceps, *1045
 nutritional, arrest by use of riboflavin, 507
 occurrence of so-called dinitrophenol cataracts without ingestion of dinitrophenol 854
 operation to reduce incidence of prolapse of lens *597
 operations in prehistoric age *1
 operations postoperative complication, 875
 parathyroid glands and eye 666
 permeability of capsule of lens and dextrose content of blood, aqueous and lens in animals with galactose cataract 321
 property of galactose cataracts to protect vitamin C from oxidation 506
 secondary operation for, 114
 stereophotography of incipient senile cataract, 143
 technic of total extraction with forceps 508
 total extraction of opacified crystalline lens with Sreellen's loop, 1089
 treatment of tetany cataract, 143
 treatment with preparations of strontium, 145
 zonular causation of 144
- Catarrh** See under Conjunctivitis
- Crusties** surgical treatment of caustic burns of eye, report of cases with illustrations 1088
- Cruterization** See under names of various diseases
- Cavernous Sinus** photochromatic interval in glaucoma and cavernous atrophy 1086
- Cells** See Tissue
- Cerebrospinal Fluid** pressure in cases of retinitis 140
- Cerebrum** See Brain
- Chandler P A** Inferior iridotomy in operations for cataract on eyes with posterior synechiae or pupillary membrane value of operation *641, 1105
- Charpentier, R** Retinal arterial spasm fatal forms, lack of acetylcholine 131
- Cheek**, rotation in ophthalmology, *410
- Choked Disk** See Neuritis, optic
- Cholesterol** metabolism of patients with retinal lesions, 144
- Choline** and Choline Derivatives retinal arterial spasm, fatal forms, lack of acetylcholine, 131
- Chondroma**, voluminous, of left ethmoid region with mucocoeles in all sinuses and grave ocular syndrome, 1096
- Chordoma**, ophthalmologic aspects of, 127
- Chorioretinitis** See Retinochoroiditis
- Choroid** choroidal hemorrhage simulating choroidal tumor, 520
 hereditary chorioretinal disease 865
 intraocular tension in cases of sarcoma of choroid and ciliary body *359
 topography and frequency of complications of uveal sarcoma, *290
- Ciliary Body** See Iridocyclitis, Uvea
- Coats Disease** See Retinitis, exudative
- Cohen M** Choroidal hemorrhage simulating choroidal tumor 520
Location of lens in anterior chamber of congenital origin, in 2 members of family followed by secondary glaucoma, extraction of lenses, 685
 Unilateral exophthalmos due to sclerosing sarcoma of anterior cranial fossa 686
- Collins Treacher Collins** prize, 883
- Coloboma** See under Iris, Macula Lutea, Nerves, optic
- Color Blindness** can deficiency of color perception be cured by neophan glasses? 107
 physiologic and clinical ophthalmologic problems in relation to individual variability *541, *726, *913
- Congresses** See under Societies
- Conjunctiva**, accidental vaccination of, *89
 cancer of eyelids conjunctiva and cornea squamous cell epithelioma, *254
 cultivation of human conjunctival and corneal epithelium in vitro 1107
 diplopia preceding marked increase of growth in dermoliopoma of conjunctiva 123
 histologic and clinical statistics on 80 cases of tumor of bulbar conjunctiva, 123
 lymphoid reaction of bulbar conjunctiva, reaction to tuberculin, 319
 precancerous melanosis of lids and conjunctiva 683
 subconjunctival cyst simulating prolapse of lacrimal gland 338
 transplantation from cadaver's eye 117
- Conjunctivitis** See also Ophthalmia
 acute membranous in brass worker 689
 antepositis conjunctivae fornix operation in cases of severe spring catarrh 143 850
 associated with infection by *Streptococcus viridans* clinical and bacteriologic observations in epidemic, *19
 chronic is provoked by undetected mycellan concretions in lacrimal canaliculus 660
 experimental production of local eosinophilia, particularly in relation to spring catarrh, 852
- Granular** See Trachoma
 limbic form of spring catarrh 850
 mannitol fermentation as indicator of conjunctival pathogenicity of *staphylococci*, *274
 phacocoele report of case, 114
 staphylococci, 108
 staphylococci treatment with staphylococcus toxoid preliminary note, *271
 tularemia from clinical and from ophthalmologic standpoint 1085
 use of strong solutions of zinc sulfate in Morax-Avenfeld conjunctivitis, 661

INDEX TO VOLUME 20

- Conjunctivitis--Continued
 vernal, 877
 vernal, corneal and scleral forms of, 145
- Contact Glasses. See Glasses
- Cooper W. L. Experimental iontophoresis of rabbits' corneas, report of 2 cases of corneal dystrophy with treatment by ionic medication, *624
- De Cori. Action of vitamin C on human cataract, 142
 Effect of pH on miotics and mydriatics, 145
- Cornel, abscess metastatic septile endophthalmitis with ring abscess of cornea, report of case with description of anatomic changes, 1102
 anatomic structure and evolution of irido-corneal angle in mammalia 141
 antigenic properties of cornea and vitreous, 850
 bilateral superficial lesions of cornea due to allergy, report of case, 334
 cancer of eyelids, conjunctiva and cornea, squamous cell epithelioma, *254
 conical, incipient (fruste) keratoconus, 124
 conical, preliminary report of case of keratoconus, successfully treated with organo-therapy, radium and short wave diathermy, 320
 conical, vitamin D complex in keratoconus, etiology, pathology and treatment of conical cornea, preliminary report, 852
 cultivation of human conjunctival and corneal epithelium in vitro, 1107
 development of trachoma in, 147
 dysostosis multiplex with consideration of ocular condition, report of case, 322
 events of vascularization and devascularization seen in, *974
 experimental iontophoresis of rabbits' corneas, report of 2 cases of corneal dystrophy with treatment by ionic medication, *624
 histology of Bowman's membrane in cases of glaucoma, 319
 Inflammation. See Keratitis
 marginal degeneration of, 108
 modified corneal clamp to facilitate insertion of stitches, *91
 obliteration of pericorneal blood vessels in treatment of various types of keratitis, further observations, 158
 Pannus. See Trachoma
 partial funnel detachment of Descemet's membrane, 109
 pigmentation, association of annular band of pigment on posterior capsule of lens with Krukenberg spindle *52
 pigmentation, Krukenberg's pigmented spindle and pigment ring in cornea, report of cases, 1083
 results of autotransplantation into anterior chamber, their significance regarding corneal nutrition *645
 sporothrix granuloma of limbus, 660
 Syphilis. See under Keratitis
 treatment of infected abrasions of, 1083
 treatment of Mooren's ulcer with liver extract, 320
 zonular opacity of, 687
- Cowen, T. Use of sorbitol in glaucoma, *1036
- Cramer, I. I. Oculoglandular tularemia, *88
- Cranium, enormous bony tumor of outer wall of orbit greater wing of sphenoid bone and temporal fossa, 134
 lesions of chiasma in case of cranial traumatism, 116
 orbital hyperostosis, its occurrence in 2 cases of meningioma of skull, *996
- Steeple Skull. See Acrocephaly
- unilateral exophthalmos due to sclerosing sarcoma of anterior cranial fossa, 686
- Crisp, W. H. Shall we use cycloplegics? 1099
- Crystalline Lens. See Lens, Crystalline
- Cuénod, A. Treatment of trachoma according to recent experimental research, 124
- Cullom M. M. New capsule grasping forceps, *1045
- Cyclitis. See Iridocyclitis
- Cycloplegia. See Accommodation and Refraction
- Cylindroma, tumors of orbit, cylindroma of lacrimal gland, 1096
- Cysts. See under Conjunctiva, Eyelids, Retina, etc
- Dacryoadenitis. See Lacrimal Organs
- Dacryocystitis. See Lacrimal Organs
- Dacryocystorhinostomy. See Lacrimal Organs
- Dark Adaptation. See Accommodation and Refraction
- David, M. Opticochiasmatic arachnoiditis, 127
- Davidson, M. Zonular opacity of cornea, 687
- Deformities. See under names of organs, as Lys, absence of, Fingers and Toes, abnormalities, etc
- Dehydrogenation. See under Retina
- DeJcan, C. Changes in fundus oculi of rabbits with rabies, 132
- Depth Perception. See Space Perception
- Dermolipoma, diplopia preceding marked increase of growth in dermolipoma of conjunctiva, 123
- Descemet Membrane. See under Cornea
- Dextrose, glycolysis, metabolism and glycolysis of lens after parathyroidectomy, 113
 permeability of capsule of lens and dextrose content of blood, aqueous and lens in animals with galactose cataract, 321
- Diabetes Mellitus, cholesterol metabolism of patients with retinal lesions, 144
 diabetic and tetanic cataract in relation to Krauss' theory of muscle function of cortical cells, 145
 ocular disorders observed in, 145
- Diathermy. See under names of various diseases and organs, i.e., Cornea, conical, Lys, diseases, Retina, detachment, etc
- Dinitrophenol. See Nitrophenol
- Diplegia. See Paralysis
- Diplopia preceding marked increase of growth in dermolipoma of conjunctiva, 123
- Directory of ophthalmologic societies, 165, 349, 531, 690, 897, 1119
- Disk, Optic. See Nerves, optic
- Dreyfus, G. Cataract due to dinitrophenol, 125
- Dropsy. See also Hydrophthalmos, etc
 osmotic pressure of aqueous humor in epidemic dropsy glaucoma, 853
- Drouet. Hypophysis and its relation to eye, 141
- Dublin G. T. Bilateral superficial lesions of cornea due to allergy, report of case, 334
- Dubois-Poulsen. Oxiometry of arteries of limbs in thrombosis of central retinal vein, 132
- Ductless Glands. See Endocrine Glands
- Duggan. Ocular disorders observed in diabetes, 145
- Dunnington, J. H. Intraocular tension in cases of sarcoma of choroid and ciliary body, *359
- John Martin Wheeler, 885
- Dutt, K. C. Cataract operations in prehistoric age, *1
- Dysostosis. See Bones, growth
- Eales' Disease. See under Retina, Vitreous Humor
- Ear, transplantation conchae auriculae to correct spastic entropion following total tarsectomy, 875
- Ectopia Lentis. See Lens, Crystalline
- Letropion. See under Eyelids

- Edema See under Retina
- Electricity, cataract due to electric shock, 113
- Electrocoagulation See under specific headings, as Retina, detachment, etc
- Electrolysis See Retina, detachment
- Electrosurgery See under specific headings as Retina, detachment, etc
- Elsberg, C A Preliminary report of value of functional visual tests for localization of supratentorial tumors of brain, 524
- Emmetropia See Accommodation and Refraction
- Endocrine Glands, endocrinology and eye 141
- ocular problems in field of endocrinology, 142
- retinitis pigmentosa and internal secretion treatment with estrogen, 142
- use of interferometer for estimation of endocrine function in ocular diseases, 142
- Endophthalmitis See Eyes, diseases, Uvea
- Entoptoscope See Eyes, examination
- Entropion See under Eyelids
- Eosinophilla, experimental production of local eosinophilla, particularly in relation to spring catarrh, 852
- Epilepsy, case associated with meningioma of optic nerve sheath compressing olfactory centers, dural calcifications and thalamic lesions, 327
- Euphora See Lacrimal Organs
- Epithelioma, cancer of eyelids conjunctiva and cornea, squamous cell epithelioma, *254
- treatment of epithelioma of eyelids 517
- Esophoria and anophoria in twins of same sex 330
- Esser J F S Rotation of cheek in ophthalmology *410
- Eyans, J N Ophthalmic carriage, *286
- Exophthalmos dysfunction of thyroid gland and its relation to eye, 141
- orbital mechanics and limitations and movement of eye, optic nerve, oblique muscle and Muller's muscle with relation to exophthalmos, separation of retina, thrombosis and postoperative malaise, 1111
- proptosis in 6 week old infant, 693
- smooth muscle of periorbital and mechanism of 669
- unilateral, analysis of 71 consecutive cases 857
- unilateral due to sclerosing sarcoma of anterior cranial fossa 686
- Extremities, blood supply, oscillometry of arteries of limbs in thrombosis of central retinal vein 132
- Eyelids, aleukemic lymphosis involving upper lids, pathologic changes, 1110
- blepharitis comb *658
- cancer of eyelids, conjunctiva and cornea squamous cell epithelioma, *254
- cicatization without ectropion following therapy with Carrel-Dakin solution, 114
- correction of entropion by transplantation of orbicularis muscle of lower eyelid 1104
- cysts, staphylococcal septicemia following suppurating meibomian cyst, report of case, 508
- diseases, blepharochalasis 115
- lichen planus of, 326
- malignant neoplasm of report of 2 cases, 682
- new method for rebuilding lower lid, 145, 315
- palpebral manifestations in delayed hereditary syphilis, 326
- plastic operations on lower lid with consideration of problem of carcinoma 668
- precancerous melanosis of lids and conjunctiva, 683
- ptosis, operation for congenital ptosis, 692
- repair of old laceration at medial end of lower lid 524
- rotation of cheek in ophthalmology *410
- Eyelids—Continued
- simple surgical procedure for relief of spastic entropion, 148
- total symblepharon with plastic repair, 338
- transplantation conchae auriculae to correct spastic entropion following total tarsectomy, 855
- treatment of cancer of, 879
- treatment of epithelioma of, 517
- tumor of lid in case of Bourneville-Pringle's disease 864
- use of auricular cartilage for correction of severe entropion following tarsectomy, 147
- Eyes See also Orbit, Vision, and under names of special structures and diseases
- absence of, case of primary bilateral anophthalmia, clinical and histologic report 851
- absence of, congenital anophthalmos veiling ophthalmotritoid, report of case 1082
- Accommodation and Refraction See Accommodation and Refraction
- anatomic structure and physiologic function of parathyroid gland in relation to eye 142
- and tooth, 663
- Anesthesia See Anesthesia
- Anomalies See under names of special structures of the eye and diseases as Acrocephaly, Fingers and Toes, abnormalities, etc
- apparatus for measuring and recording disturbances of ocular motility, 145
- Blood Supply See also Retina, blood supply
- blood supply, behavior of intraocular pressure in acute intraocular vascular reactions, 670
- changes in eye in herpes zoster ophthalmicus 329
- Cysts See under Conjunctiva, Eyelids, Retina, etc
- Diseases See also Iridocyclitis, Glaucoma, Ophthalmia Trachoma, etc
- diseases, attempts to develop in vitro oculo-tropism of virulent anthrax bacillus and of Proteus vulgaris 659
- diseases, carbon disulfide poisoning, *839
- diseases, dimethyl sulphate poisoning in relation to ophthalmology, 665
- diseases, fever therapy in ophthalmology, 518
- diseases in leprosy, 853
- diseases, metastatic septic endophthalmitis with ring abscess of cornea, report of case with description of anatomic changes, 1102
- diseases, ocular diseases connected with gonadal dysfunction 141
- diseases, ocular disorders observed in diabetes 145
- diseases, ocular problems in field of endocrinology, 142
- diseases, pinophthalmitis and sympathetic ophthalmia *804
- diseases, pathogenesis of ocular diseases of dental origin, 126
- diseases, sea air and ophthalmology 135
- diseases short wave therapy in ophthalmology, 679
- diseases, significance of heredity in ophthalmology, preliminary survey of hereditary eye diseases in Tasmania, 322
- diseases, use of interferometer for estimation of endocrine function in 142
- dislocation of eyeball as complication of oxycephaly 330
- dysfunction of thyroid gland and its relation to eye, 141
- endocrinology and eye 141
- Examination See also Accommodation and Refraction Vision tests, etc
- examination of, 143
- examination, presentation of small model entoptoscope, 112
- examination use of sodium and mercury vapor lamps in ophthalmology, 509

INDEX TO VOLUME 20

- Eyes—Continued
 experimental hypertension, vascular changes in eyes, *812
 Foreign Bodies in See Foreign Bodies
 fungi in normal and diseased eyes, 851
 fusional movements, role of peripheral retinal stimuli, 873
 hemorrhage into, disappearance of iris after prolonged hyphemia, 684
 hypophysis and its relation to eye, 141
 implantation of hollow grooved body into orbit for filling late after enucleation of eyeball, *709
 Injuries See also under Foreign Bodies
 injuries, exhibition at Royal Eye Hospital, 504
 intrascleral grafts with tendon from dead animals after excision of globe, 134
 luxation of eyeball with complete restoration of its function, report of case, 857
 Movements See also Nystagmus, Paralysis, Strabismus, etc
 movements, fundamental plan underlying ocular movements, conjugate horizontal movements, 329
 Muscles See also Heterophoria, Paralysis, Strabismus
 muscles, anatomic and clinical study of insertion of oblique muscles, 668
 muscles, disturbances of vertical motor muscles of eyes, *175
 muscles, muscle weakening by central tenotomy, 856
 obstetric ophthalmology, 340
 ocular leprosy, report of 22 cases, 507
 ocular thermometry, 110
 oculoglandular tularemia, *88
 orbital mechanics and limitations and movement of eye, optic nerve, oblique muscle and Muller's muscle with relation to exophthalmos, separation of retina, thrombosis and postoperative malaise, 1111
 Paralysis See Paralysis
 parasites, intraocular nematode worms, report of case and review of literature, *1006
 Physiology See Vision, physiology
 Pigmentation See Retinitis pigmentosa, and under special structures of eyes, as Cornea, etc
 Proptosis See Exophthalmos
 Refraction See Accommodation and Refraction
 research studies, some technical and practical notes, *395
 Sarcoma See Sarcoma
 successful hyperthermic treatment of ophthalmoblenorrhoea by application of steam spray, report of cases, 866
 Surgery See also Cataract, extraction, Glaucoma, therapy, etc
 surgery, advantages of use of coagulants in ocular operations especially in extraction of cataract and in plastic operations, *406
 surgical treatment of crustic burns of eye, report of cases with illustrations, 1088
 Tension See Glaucoma, Tension
 Tuberculosis See Tuberculosis
- Farmer, C J Biochemistry of lens, studies on glutathione in crystalline lens, *417
 Farmer, J as indicator of conjunctivitis, as indicator of conjunctivitis of staphylococci, *274
 Ferree, C E Testing fitness for night flying visual acuity, *58
 Fever, pyrexia and trachoma, 676
 Therapeutic See Eyes, diseases, Nerves, optic
- Fibroma of sclera, 1095
 Filters, cobalt blue filter for observation of fit of contact lenses, *657
- Fingers and Toes, abnormalities, bilateral congenital ectopia lentis with arachnodactyly (Marfan's syndrome), *371
 Fistula, congenital, of lacrimal gland, 685
 Foramen, Optic See Optic Canal
 Forceps, new capsule-grasping forceps, *1045
 Foreign Bodies, delayed removal of magnetic foreign bodies from vitreous, 152
 intraocular, report of case, 692
 Forea Centralis See Macula Lutea
 François, J Aneurysmal dilatation of ophthalmic artery, clinical course, 133
 Enormous bony tumor of outer wall of orbit, greater wing of sphenoid bone and temporal fossa, 134
 Syphilitic opticochiasmic arachnoiditis and pyretotherapy, 130
 Fread, B Intraocular mass, 151
 Frey, G Congenital fistula of lacrimal gland, 685
 Disappearance of iris after prolonged hyphemia, 684
 Friedenwald, J S Circulation of aqueous, mechanism of secretion of intraocular fluid, *761
 Fritz Blood supply in retinal vessels, 131
 Isoperfusion and anisoperfusion of capillaries in cases of retinal hypertension, 140
 Fundus Oculi See under Retina
 Fungi in normal and diseased eyes, 851
- Galactose, biochemistry of lens, effect of galactose on permeability of capsule of lens, *80
 cataract, clinical observations, 506
 permeability of capsule of lens and dextrose content of blood, aqueous and lens in animals with galactose cataract, 321
 property of galactose cataracts to protect vitamin C from oxidation, 506
 Genet, L Blindness in infants, without ophthalmoscopic changes (congenital forms), 133
 Retinal arterial spasm, fatal forms, lack of acetylcholine, 131
 Gibson, G G Clinical significance of retinal changes in leukemia, *364
 Gillesen Diabetic and tetanic cataract in relation to Krauss' theory of muscle function of cortical cells, 145
 Glasses See also Accommodation and Refraction
 can deficiency of color perception be cured by neophan glasses? 107
 cobalt blue filter for observation of fit of contact lenses, *657
 injuries attributed to glasses of aviators, 854
 muscle spasm caused by eyeglasses, 335
 selection of contact lenses by means of metallic prostheses, 122
 use of contact glasses for cosmetic purpose, 322
 use of new type of contact glass made of pyroxylin, 145
 vertical prism values in commonly used bifocal lenses, *95
- Glaucoma See also Hydrophthalmos, Tension
 action of yohimbine hydrochloride on intraocular tension, 125
 and diastolic pressure, 664
 association of cyclodialysis with fistulizing operations in certain cases of glaucoma, 323
 congenital buphthalmos complicated by dislocation of lens and hemorrhage into vitreous with complete recovery of central vision, *757
 exfoliation of lens capsule in, 508
 histology of Bowman's membrane in cases of, 319
 infantile, control of intraocular pressure in, 1086

INDEX TO VOLUME 20

- Glaucoma**—Continued
 juvenile, with angiomatosis of retina, 665
 luxation of lens in anterior chamber, of congenital origin, in 2 members of family, followed by secondary glaucoma, extraction of lenses, 685
 osmotic pressure of aqueous humor in epidemic dropsy glaucoma, 853
 pathogenesis, etiology and treatment of glaucomatous iridocyclitis 333
 photochromatic interval in glaucoma and cavernous atrophy, 1086
 spherophakia, luxation of lenses and secondary glaucoma relieved by extraction of lenses 324
 surgical treatment of 126
 therapy, spatula-forceps for Heine's operation 112
 use of sorbitol in, *1036
 with retinitis pigmentosa, 691
Glioblastospongloblastoma, anaplastic or differentiated, of retina, 863
Glutathione biochemistry of lens, studies on glutathione in crystalline lens, *417
Glycolysis See Dextrose, glycolysis
Goggles See Glasses
Goiter See also Thyroid
 pulsus of ocular muscles in case of toxic goiter, 335
Goldblatt H Experimental hypertension, vascular changes in eyes *812
Goldmann Improved slit lamp, 146
Goldstein D H Conjunctivitis associated with infection by *Streptococcus viridans* clinical and bacteriologic observations in epidemic, *19
Golgi apparatus, microstructure of epithelial cells and its importance for etiology of trachoma 862
Gonads, ocular diseases connected with gonadal dysfunction, 141
Gonioscope, new model, 502
Gonococci, pathogenesis, etiology and treatment of glaucomatous iridocyclitis 333
Gonorrhea, successful hyperthermic treatment of ophthalmoblennorrhoea by application of steam spray report of cases 866
Goodhart, R Acute alcoholic amaurosis, *797
Greenberg, M Proptosis in 6 week old infant, 693
Griffey, W P Retinal detachment with cyst formation, report of case, 523
 Total symblepharon with plastic repair, 338
Gronblad-Strandberg Syndrome See Pseudoxanthoma elasticum, Retina pathology
Grueter Microstructure of epithelial cell and significance of such cells in causation of trachoma, 146
Gundersen T Obliteration of pericorneal blood vessels in treatment of various types of keratitis, further observations 158
 Results of autotransplantation of cornea into anterior chamber, their significance regarding corneal nutrition, *645
Gunn Sign See Retina, blood supply
Guy L Simple dacryocystorhinostomy *954
Harada's Disease See under Uvea
Head, injuries, retinal arterial hypertension in cases of commotio cerebri 138
Heart congestion of retina indicating involvement of retinal vessels in failure of right side of heart report of cases, 1093
 effect of extract of ciliary body on isolated frog heart, 512
Heath, P Some pharmacologic reactions of isolated iris muscles, 1108
Heed C R Delayed removal of magnetic foreign bodies from vitreous 152
Heine Operation See Glaucoma, therapy
Hermans, R Pathogenesis of ocular diseases of dental origin, 126
Herpes zoster ophthalmicus, changes in eye in, 329
 zoster, specific treatment for, *304
Heterophoria See also Strabismus
 nature and management of, *375
Hildesheimer Possibility of decreasing myopia by scleral excision with electric knife 145
 Treatment of cataract with preparations of strontium, 145
von Hippel's Disease See Retina, tumors
Hirtz G Four cases of Laurence-Bardet-Biedl syndrome, treatment with total extracts of anterior lobe of pituitary gland, 134
Holmes, W J Congenital buphthalmos complicated by dislocation of lens and hemorrhage into vitreous with complete recovery of central vision, *757
Homatropine evaluation of homatropine benzedrine cycloplegia, *585
Horniker Diagnostic value of ophthalmodynamometry, 140
Hughes, W E George Edmund de Schweinitz, 1074
Hughes, W L New method for rebuilding lower lid, 145 315
 Repair of old laceration at medial end of lower lid, 524
Hydrogen Sulfide See Sulfides
Hydrogen Ion Concentration, effect of pH on miotics and mydriatics, 145
 pH of lens in naphthalene cataract of rabbit, 855
Hydrophthalmos, congenital buphthalmos complicated by dislocation of lens and hemorrhage into vitreous with complete recovery of central vision *757
Hypermetropia See Hyperopia
Hyperopia, familial hypermetropia of high degree, consideration of pathogenesis of ametropia 671
Hyperpyrexia See Fever
Hypertelorism, ocular, report of case, 683
Hypertension See Blood pressure, high
 Ocular See Tension
Hypheemia See Eyes, hemorrhage into
Hypophysis See Pituitary Body
Hypotony See Tension
Idiocy amaurotic, Try-Sachs' disease, report of case, 687
 juvenile form of familial amaurotic idiocy, 673
Igersheimer Latent processes in visual pathways in cases of hypertension, 139
Illumination See Lighting
Von Imre Ocular diseases connected with gonadal dysfunction, 141
Industrial Diseases, acute membranous conjunctivitis in brass worker, 689
 carbon disulfide poisoning *839
 exhibition of eye injuries at Royal Eye Hospital, 504
 miner's nystagmus from standpoint of etiology, 144
 superficial and deep-seated disciform keratitis in caisson workers after exposure to hydrogen sulfide, report of cases, 323
Infants, newborn, ophthalmoscopic study 144
Inflammation, Ocular See Ophthalmia, and under special structures of eyes
Injuries See under Cranium, Eyes, Head, etc
Inosite, inositol in ocular tissues, *299
Instruments See also Apparatus
 blepharitis comb *658
 comparison microscope 145
 graduation of Baillart and Sobanski dynamometers on human cadaver, 143
 improved slit lamp, 146

Instruments—Continued
 modified Briggs' retractor for dacryocystorhinostomy, 665
 modified corneal clamp to facilitate insertion of stitches, *91
 new capsule-grasping forceps, *1045
 new model gonioscope, 502
 new refractor suspension, *1044
 presentation of small model entoptoscope, 112
 simple accessory for dark room, 509
 Souter tonometer, 665
 spatula-forceps for Heine's operation, 112
 testing fitness for night flying visual acuity, *58

Internal Secretions See Endocrine Glands

Intraocular Tension See Tension

Ionization, experimental iontophoresis of rabbits' corneas, report of 2 cases of corneal dystrophy with treatment by ionic medication, *624

Iontophoresis See Ionization

Iridocyclitis See also Ophthalmia, sympathetic glaucomatous, pathogenesis, etiology and treatment, 333

Iris See also Pupils

disappearance after prolonged hyphemia, 684
 Foreign Bodies in See under Foreign Bodies
 gonioscopy of surgical colobomas of, 856
 inferior iridotomy in operations for cataract on eyes with posterior synechiae or pupillary membrane, value of operation, *641, 1105
 melanoma of iris with pathological findings, 517
 prolapse, cataract operation to reduce incidence of, *597
 some pharmacologic reactions of isolated iris muscles, 1108

Jackson, E Subjective study of visual aberrations, 1100

Theory and use of cross cylinders, 870

Jaffe, M Neuroparalytic keratitis, 688

Jameson, P C Subconjunctival section of ductules of lacrimal gland as cure for epiphora, 339

Jeandelize Hypophysis and its relation to eye, 141

Jones, L T Intraocular nematode worms, report of case and review of literature, *1006

Jordan, L W Intraocular nematode worms, report of case and review of literature *1006

Journals See Periodicals

Joviu Cataract in relation to dysfunction of hypophysis, 143

Jurisprudence, Medical, detachment of retina and accidents, 860

Katz, D Obstetric ophthalmology, 340

Kayser-Fleischer Ring See Cornea, pigmentation

Keith, N Arterial hypertension of retina, 137
 Keratitis, heredosyphilitic, information based on 349 cases of, 109

infection of cornea in irritation of peripheral nerves, 662

neuroparalytic, 688

obliteration of pericorneal blood vessels in treatment of various types, further observations, 158

superficial and deep-seated disciform keratitis in caisson workers after exposure to hydrogen sulfide, report of cases, 323

Trachomatous See Trichoma

traumatic parenchymatous, in congenital syphilis, 110

Keratoconus See Cornea, conical

Keratoplasty See under Cornea

Keves, I E L Experimental hypertension, vascular changes in eyes, 140, *812

Kidneys, Diseases See also Nephritis
 diseases, measurements of caliber of retinal vessels of patients with hypertension and nephritis, 1093

King, M J Scleromalacia perforans, report of case in which eye was examined microscopically, *1013

Klar Miner's nystagmus from standpoint of etiology, 144

Retinal arterial hypertension in cases of commotio cerebri, 138

Killen, B A Retinitis proliferans, clinical and histologic studies, *427

Knapp, A James A Spalding, 318

Ladislav de Blaskovics, 1081

Orbital hyperostosis, its occurrence in 2 cases of meningioma of skull, *996

Komoto, Juiiro, 504

Kornzweig, A L Anomalous retinal vein, 683

Koyanagi Histologic changes in eyes of patients with hypertension, 138

Krause, A C Inositol in ocular tissues, *299

Krausz Combined short wave and ionization apparatus, 146

Kravitz, D Studies of visual fields in cases of verified tumor of brain, *437

Kronenberg, B Topography and frequency of complications of uveal sarcoma, *290

Krukenberg Spindle See Cornea, pigmentation

Lacrimal Canal See Lacrimal Organs

Lacrimal Organs, chronic conjunctivitis is provoked by undetected mycellan concretions in lacrimal canaliculus, 660

dacryoadenitis, report of 2 cases, 689

dacryocystitis in lymphatic leukemia, *85

Fistula See Fistula

melanoblastoma of lacrimal caruncle, report of case and review of literature, 676

mixed cell tumor of lacrimal sac, 1101

modified Briggs' retractor for dacryocystorhinostomy, 665

nevocarcinoma of lacrimal caruncle, clinical and anatomic study, 517

recurrent pneumococcal dacryoadenitis, 112

simple dacryocystorhinostomy, *954

some results of intranasal dacryocystorhinostomy, 1088

subconjunctival cyst simulating prolapse of, 338

subconjunctival section of ductules of lacrimal gland as cure for epiphora, 339

syphilis of lacrimal sac simulating acute dacryocystitis, 1088

tumors of orbit, cylindroma of lacrimal gland, 1096

Lancaster, W B Anisokonia, *907

Larocaine See under Anesthesia

Last, M Coloboma of optic nerve and of macula, microscopic study *787

Lauber Arterial hypotony and retinal atrophy, 138

Laurence-Biedl Syndrome, blindness of hypophyseal origin, 4 cases of adiposogenital syndrome with retinal degeneration and mental backwardness, 328

Laurence-Bardet-Biedl syndrome, treatment with total extracts of anterior lobe of pituitary gland, 4 cases, 134

Laval, J Bilateral congenital ectopia lentis with arachnodactyly (Marfan's syndrome), *371

Tay-Sachs' disease, report of case, 687

Lecture, first de Schweinitz lecture, 1072

Leinfelder, P J Pathologic changes in amblyopia following triparamide therapy, 867

Lemoine A N Lesion of optic tract probably result of infected sphenoid sinuses, *966

- Lens, Crystalline, association of annular band of pigment on posterior capsule of lens with Krukenberg spindle, *52
 bilateral congenital ectopia lentis with arachnoidactyly (Marfan's syndrome), *371
 biochemistry of, effect of galactose on permeability of capsule of lens *80
 biochemistry of, studies on glutathione in crystalline lens, *417
 chemical and histochemical investigation of urea in lens and ocular membranes 1082
 congenital buphthalmos complicated by dislocation of lens and hemorrhage into vitreous with complete recovery of central vision *757
 exfoliation of lens capsule in glaucoma, 508
 experimental studies on contractility of lens fibers, 1089
 μ n of lens in naphthalene cataract of rabbit, 855
 incineration of sections of normal human lenses, 107
 intracapsular accommodation of, 513
 luxation of lens in anterior chamber of congenital origin, in 2 members of family followed by secondary glaucoma, extraction of lenses, 685
 luxation of lens through retinal tear into sub-retinal space, 112
 metabolism of lens after parathyroidectomy 113
 Opacity See Cataract
 permeability of capsule of lens and dextrose content of blood, aqueous and lens in animals with galactose cataract, 321
 relation of age and phosphoric acid esters content of lens, 659
 serologic research, specific properties of lens 319
 spherophakia, luxation of lenses and secondary glaucoma relieved by extraction of 324
 Lenz Stereophotography of incipient senile cataract, 143
 ✓ Leprosy, diseases of eye in, 853
 ocular, report of 22 cases 507
 Leukemia, aleukemic lymphosis involving upper lids, pathologic changes, 1110
 clinical significance of retinal changes in *364
 lymphatic, dacryocystitis in, *85
 Lewis, P Responsibility of ophthalmologist in conservation of sight and prevention of blindness, 148
 Lichen planus of eyelids, 326
 Light, influence on arterial tension 322
 photochromatic interval in glaucoma and cavernous atrophy, 1086
 toxicity, photophobia, 111
 Lighting, light reserve for occupations in sight-saving classes, 1087
 Limbus Corneae See Corner
 Lincz Comparison microscope 145
 Lindner Clinical study of vitreous 144
 Lippincott, S W Angiomatosis retinae, report of case with pathologic study of enucleated eye, *958
 Lo Cascio Anatomic structure and physiologic function of parathyroid gland in relation to eye, 142
 Loe, F Sulfanilamide treatment of trachoma preliminary report, 880
 Ludvig, E Absorption of visible light by refractive media of human eye, *37
 Determination and significance of scotopic retinal visibility curve, *713
 Lymphosis, aleukemic, involving upper lids pathologic changes, 1110
 McAndrews, L F Muscle spasm caused by eyeglasses, 335
 MacCallan Discussion of national policy to be adopted in tropical country for prevention of blindness, 147
 McCarthy, E F Absorption of visible light by refractive media of human eye, *37
 McCool J L Mixed cell tumor of lacrimal sac, 1101
 McDonald, R Angiomatosis retinae, report of case with pathologic study of enucleated eye, *958
 Carbon disulfide poisoning, *839
 McEwen, C Conjunctivitis associated with infection by *Streptococcus viridans*, clinical and bacteriologic observations in epidemic, *19
 McLean, J M New model gonioscope 502
 Macula Lutea, coloboma of optic nerve and of macula, microscopic study *787
 disciform macular degeneration of Junius-Kuhnt, 1092
 pathologic anatomy of disciform degeneration of, 674
 variations in pressure of retinal arteries and in capillary pressure in relation to compression of globe, 859
 Maisler, S New refractor suspension *1044
 Majima So-called paratrachoma a virus disease due to Halberstadter-Prowazek virus, 146
 Marfan's Syndrome See Fingers and Toes, abnormalities
 Martin Ophthalmologic aspects of case of choroidoma, 127
 Maxwell, J T Nature and management of heterophorias, *375
 Maziny Bey Social aspect of prevention of blindness, 147
 Meibomian Glands See Eyelids
 Melanin, pigmentation, precancerous melanosis of lids and conjunctiva, 683
 Melanoblastoma of lacrimal caruncle, report of case and review of literature, 676
 Melanoma of iris with pathologic findings 517
 Melanosarcoma, differences in manner of perforation of sclera caused by primary melanosis and metastatic carcinoma of uveal tract, 676
 Melanosis See Melanin, pigmentation
 Meninges See also Arachnoid
 case of epilepsy associated with meningioma of optic nerve sheath compressing olfactory centers, dural calcifications and thalamic lesions, 327
 hypertensive meningeal hydrops 1090
 suprasellar meningioma, 337
 Meningioma, case of epilepsy associated with meningioma of optic nerve sheath compressing olfactory centers dural calcifications and thalamic lesions 327
 orbital hyperostosis, its occurrence in 2 cases of meningioma of skull, *996
 suprasellar 337
 Mental Diseases psychoses complicating recovery from extraction of cataract 324
 Merrill Role of social worker in program for prevention of blindness, 148
 Methyl Sulfate, dimethyl-sulphate poisoning in relation to ophthalmology, 665
 Michail Histologic picture and pathogenesis of trachoma, 147
 Milcou Cataract in relation to dysfunction of hypophysis, 143
 Mills, L Ocular dominance, 105
 Mines and Miners miner's nystagmus from standpoint of etiology 144
 study of miner's nystagmus 511
 Minsky H Dacryoadenitis report of 2 cases 689

INDEX TO VOLUME 20

- Miosis See under Pupils
- Miyashita Work in schools in Japan for children with defective sight, 147
- Moon-Laurence-Biedl Syndrome See Laurence-Biedl Syndrome
- Mooren's Ulcer See under Cornea
- Muller Graduation of Baillart and Sobanski dynamometers on human cadaver, 143
- Mulock Houwer Acute trachoma, 12 cases, 147
- Muscles, Ocular See Eyes, muscles, Strabismus physiology, experimental study of influence of muscular work on intraocular tension, 119 smooth muscle of periorbital and mechanism of exophthalmos, 669 some pharmacologic reactions of isolated iris muscles, 1108 spasm caused by eyeglasses, 335
- Mydriasis See under Pupils
- Myelin See Retina, innervation
- Mylius Significance of transitory arterial hypertension in genesis of retinal changes, 138
- Myopia, case of transient inflammatory myopia, 858 familial hypermetropia of high degree, consideration of pathogenesis of ametropia, 671 possibility of decreasing myopia by scleral excision with electric knife, 145 temporary spasmodic, due to arsphenamine 513
- Naphthalene, pH of lens in naphthalene cataract of rabbit, 855
- Narcosis See Anesthesia
- Nasopharynx, retinal blood pressure in cases of nasal diseases and effects of surgical intervention in nose and nasopharynx, 1092
- Nastri Cholesterol metabolism of patients with retinal lesions, 144
- Nataf, R Treatment of trachoma according to recent experimental research, 124
- Nectoux, R Action of yohimbine hydrochloride on intraocular tension, 125
- Nematodes, intraocular nematode worms, report of case and review of literature, *1006
- Nephritis, importance of ocular examination for understanding of hypertension and nephritis, 139
- Nerves See also under Neuritis, Paralysis, etc
optic, calcium deposits in neighborhood of chiasm in disturbances of, 673
optic, case of epilepsy associated with meningioma of optic nerve sheath, compressing olfactory centers, dural calcifications and thalamic lesions, 327
optic, coloboma of optic nerve and of macula microscopic study, *787
optic, colobomata of optic nerve sheath in rats, 506
optic, fever therapy in ophthalmology, 518
optic, general considerations of psammomas apropos of case of cystic psammoma of optic papilla, 518
optic, histologic study in experimental wounds and sutures of, 1084
optic, intraocular neuroma (Recklinghausen's disease) of left optic nerve head, 510
optic, latent processes in visual pathways in cases of hypertension, 139
optic, lesion of optic tract probably result of infected sphenoid sinuses, *966
optic, optochiasmatic arachnoiditis, importance of mixed type of atrophy of optic nerve as diagnostic sign, *384
optic, orbital mechanics and limitations and movement of eye, optic nerve, oblique muscle and Muller's muscle with relation to exophthalmos, separation of retina, thrombosis and postoperative malaise, 1111
- Nerves—Continued
optic, perimetric studies in syphilitic optic neuropathies, *201
optic, relief therapy ("Entlastungstherapie") for tabetic optic atrophy, 865
optic, sarcoma of optic papilla, report of case, 677
Paralysis See under Paralysis
peripheral, infection of cornea in irritation of, 662
- Nervous System See also Brain, Nerves, Reflex, etc
ophthalmodynamometer and its diagnostic value, 667
- Neuritis, optic, dark adaptation and its differential diagnostic value in cases of neuritis and choked disk, 121
optic, experimental study of choked disk in rat, 1084
optic, experiments on papillary stasis, 662
optic, pathogenesis of choked disk, 515
retrobulbar, and chiasmal syndrome in pregnancy, 672
retrobulbar, in pellagra, 664
- Neurofibromatosis, intraocular neuroma (Recklinghausen's disease) of left optic nerve head, 510
- Neuroretinitis See Neuritis, optic, Retinitis
- Nevocarcinoma of lacrimal caruncle, clinical and anatomic study, 517
- Nitrophenol, cataract due to dinitrophenol, 125
cataract formation after reduction of weight with dinitro-bodies, 325
experimental dinitrophenol cataract in animals, 321
influence of vitamins and dinitrophenol on production of experimental cataract, *30
occurrence of so-called dinitrophenol cataracts without ingestion of dinitrophenol, 854
- Nose, ophthalmologic aspects of case of choroidoma, 127
retinal blood pressure in cases of nasal diseases and effects of surgical intervention in nose and nasopharynx, 1092
- Nystagmus, miner's, from standpoint of etiology, 144
miner's, study of, 511
- Obesity See also Laurence-Biedl Syndrome
cataract formation after reduction of weight with dinitro-bodies, 325
occurrence of so-called dinitrophenol cataracts without ingestion of dinitrophenol (following use of reducing agent, splendrets), 854
- OBITUARIES
de Blasovics, Ladislav, 1081
Komoto, Juiro, 504
de Schweinitz, George Edmund, 1074
Spalding, James A., 318
Wheeler, John Martin, 885
- Obrig, T E Cobalt blue filter for observation of fit of contact lenses, *657
- Occupational Diseases See Industrial Diseases
- O'Connor, R Simplification of O'Connor cinch operation, 315
- Ogle, K N Induced size effect, new phenomenon in binocular space perception associated with relative sizes of images of 2 eyes, *604
- Oguchi So-called paratrachoma a virus disease due to Halberstadter-Prowazek virus, 146
- Old age, relation of age and phosphoric acid esters content of lens, 659
- Olsho, S L Vertical prism values in commonly used bifocal lenses, *95
- Onfray, R Cataract due to dinitrophenol, 125
- Ophthalmia See also Conjunctivitis, Eyes, diseases
sympathetic, and panophthalmitis, *804
sympathetic, is perforation of eye necessary for production? 1097

INDEX TO VOLUME 20

- Ophthalmodynamometry See Retina, blood supply
- Ophthalmologic societies, directory of, 165, 319, 531, 699, 897, 1119
- Ophthalmology, anniversary number of *Annales d'oculistique*, 1084
 basic course in 1073
 history, our distinguished colleague Pope John XXI, 122
 ophthalmology comes of age, years 1908-1915 up to establishment of examining board, 1106
- Ophthalmoplegia See Paralysis
- Ophthalmoscopy See Eyes, examination
- Optic Canal clinical value of roentgenographic studies of optic foramen, 331
- Optic Chiasm calcium deposits in neighborhood of chiasm in disturbances of optic nerve, 673
 chiasmal syndrome and retrobulbar neuritis in pregnancy, 672
 lesions of chiasm in case of cranial traumatism 116
 optochiasmatic arachnoiditis 127
 optochiasmatic arachnoiditis due to pneumococcus, 1090
 optochiasmatic arachnoiditis, importance of mixed type of atrophy of optic nerve as diagnostic sign, *384
 syphilitic optochiasmatic arachnoiditis and pyretotherapy 130
 syphilitic optochiasmatic arachnoiditis, 1103
- Optic Disk Optic Papilla See Nerves, optic, Neuritis optic
- Nerves See Nerves, optic
- Tract See Nerves optic
- Optics optical decentration of eye, 670
- Orbit, enormous bony tumor of outer wall of orbit, greater wing of sphenoid bone and temporal fossa, 134
 external orbitotomy, 337
 implantation of hollow grooved body into orbit for filling late after enucleation of eyeball, *709
 orbital hyperostosis its occurrence in 2 cases of meningioma of skull, *996
 orbital mechanics and limitations and movement of eye, optic nerve oblique muscle and Muller's muscle with relation to exophthalmos, separation of retina thrombosis and postoperative malaise, 1111
 orbital varices 117
 tomography of, 118
 tumors, cylindroma of lacrimal gland 1096
- Orthoptic Training See Strabismus
- Oscillometry See Extremities blood supply
- Otitis Media, hypertensive meningeal hydrops, 1090
- Oxycephaly See Acrocephaly
- Pannus See Trachoma
- Parophthalmitis See Eyes diseases
- Papilledema See Neuritis optic
- Paralysis comparison of operations for paralysis of lateral rectus muscle, 149
 palsy of ocular muscles in case of toxic goiter, 335
- Parasites See Eyes, parasites
- Parathyroid anatomic structure and physiologic function in relation to eye, 142
 and eye, 666
- Parathyroidectomy, calcium content of crystalline lens in experimental parathyroidal cataract 666
 metabolism of lens after, 113
- Paratrachoma See Conjunctivitis
- Paredrine use in cycloplegia 156, correction 505
- Parker, F C Cataract operation to reduce incidence of prolapse of iris, *597
- Parker W R George Edmund de Schweinitz 1074
- Parsons J H George Edmund de Schweinitz 1074
- Pascheff Corneal and scleral forms of vernal conjunctivitis, 145
 Formation of follicles in mucous membrane graft applied by Denig technic for pannus, 147
 Ocular problems in field of endocrinology, 142
 Struggle against blindness and new law of public assistance in Bulgaria 147
- Paton R T Recurrent retinal and vitreous hemorrhages in young—Eales' disease, report of 2 cases *276
- Payne B F Comparison of operations for paralysis of lateral rectus muscle, 149
- Pellagra, retrobulbar neuritis in, 664
- Perera C A Retinitis pigmentosa with glaucoma 691
 Retinitis pigmentosa with 'hole' in macula, report of case, *471
- Perimetry See under Vision
- Periodicals, anniversary number of *Annales d'oculistique*, 1084
 Journal of Social Ophthalmology new journal 504
 merger of *Archiv fur Augenheilkunde* and *Archiv fur Ophthalmologie*, 1072
 new editors for *Zeitschrift fur Augenheilkunde*, 1072
 new ophthalmic journals, 1073
- Pesme P Four cases of Laurence-Bardet-Biedl syndrome, treatment with total extracts of anterior lobe of pituitary gland, 134
- pH See Hydrogen Ion Concentration
- Phosphorus and Phosphorus Compounds, relation of age and phosphoric acid esters content of lens, 659
- Photophobia See Light, toxicity
- Pillet, P Histologic and clinical statistics on 80 cases of tumor of bulbar conjunctiva 123
- Pituitary Body, blindness of hypophyseal origin, 4 cases of adiposogenital syndrome with retinal degeneration and mental backwardness, 328
 cataract in relation to dysfunction of hypophysis 143
 hypophysis and its relation to eye 141
- Pneumococci, optochiasmatic arachnoiditis due to, 1090
 recurrent pneumococcal dacryodentitis, 112
- Poisons and Poisoning See under names of poisonous substances, as Carbon disulfide, Methyl Sulfate, etc
- Poleff Rickettsia bodies as cause of trachoma 146
- Pope John XXI, our distinguished colleague, 122
- Postgraduate courses Harvard University Medical School 317
 in biomicroscopy, 1073
- Pregnancy, chiasmal syndrome and retrobulbar neuritis in, 672
 obstetric ophthalmology, 340
- Pressure See Tension
- Pringle-Bourneville's Disease See Sclerosis, tuberculous
- Prisons and Prisoners, incidence of unequal pupils in unconvicted prisoners, 513
- Prizes for ophthalmic work, 1073
 Treacher Collins prize, 883
- Proby H Intrasceral grafts with tendon from dead animals after evisceration of globe 134

INDEX TO VOLUME 20

Proptosis See Exophthalmos
 Prowazek Bodies See Trachoma
 Psammoma, general considerations of psammomas apropos of case of cystic psammoma of optic papilla, 518
 Pseudoxanthoma elasticum treatment and development of 2 cases of syndrome of Gronblad and Strandberg (angioid streaks in retina and pseudoxanthoma elasticum), 514
 Pterygium, dominant inheritance of, 108
 Ptosis See Eyelids, ptosis
 Puech, P Opticochiasmatic trachnoiditis, 127
 Punttenney, I Use of sorbitol in glaucoma, *1036
 Pupils, effect of *pu* on miotics and mydriatics, 145
 incidence of unequal pupils (anisocoria) in unconvicted prisoners, 513
 inferior iridotomy in operations for cataract on eyes with posterior synechiae or pupillary membrane, value of operation, *641, 1105
 Tonic See Reflex, pupillary
 Pyrexia See Fever
 Rabies, changes in fundus oculi of rabbits with rabies, 132
 Radium, Therapy See under names of various diseases
 Rand, G Testing fitness for night flying visual acuity, *58
 Rateau, J Sea air and ophthalmology, 135
 Ruh Treatment of tetany cataract, 143
 Reading difficulties in children, *829
 Recklinghausen's Disease See Neurofibromatosis
 Reese, A B Precancerous melanosis of lids and conjunctiva, 683
 Reflex, clinical study of consensual ophthalmotonic reactions, 118
 pupillary, tonic pupil and its relationship to Adie's syndrome, 669
 Refraction See Accommodation and Refraction
 Retina See also Macula Lutea
 anaplastic or differentiated glioblastospongoblastoma of, 863
 angiomatosis retinae, report of case with pathologic study of enucleated eye, *958
 arterial hypertension of, 137
 atrophy, and arterial hypotony, 138
 Blood Supply See also under Thrombosis, Veins
 blood supply, angiosclerotic and angiospastic retinoses, 515
 blood supply, dehydrogenating processes in retina after interruption of circulation, 859
 blood supply, diastolic pressure and glaucoma, 664
 blood supply, examination of retinal vessels by dynamometry, 138
 blood supply, further contribution to knowledge of Gunn's sign, 507
 blood supply in retinal vessels, 131
 blood supply, measurements of caliber of retinal vessels of patients with hypertension and nephritis, 1093
 blood supply, ophthalmodynamometer and its diagnostic value, 140, 667
 blood supply, ophthalmodynamometry and its importance in clinical medicine, 327
 blood supply, relief therapy ("Entlastungstherapie") for tubercle optic atrophy, 865
 blood supply, retinal arterial spasm, fatal forms, lack of acetylcholine, 131
 blood supply, retinal blood pressure in cases of nasal diseases and effects of surgical intervention in nose and nasopharynx, 1092
 blood supply, variations in pressure of retinal arteries and in capillary pressure in relation to compression of globe, 859

Retina—Continued
 changes in fundus oculi of rabbits with rabies, 132
 cholesterol metabolism of patients with retinal lesions, 144
 clinical significance of retinal changes in leukemia, *364
 congestion indicating involvement of retinal vessels in failure of right side of heart, report of cases, 1093
 detachment, anatomicopathologic researches on globe with partial detachment of 20 days' duration, 1092
 detachment and accidents, 860
 detachment, diathermocoagulation (Weve) for, retinal tears without detachment, 132
 detachment, etiology considered from standpoint of surgical correction, *1046
 detachment, idiopathic, alkaline reserve of blood in, 859
 detachment, luxation of lens through retinal tear into subretinal space, 112
 detachment, multiple circumscribed, 673
 detachment, orbital mechanics and limitations and movement of eye, optic nerve, oblique muscle and Müller's muscle with relation to exophthalmos, separation of retina, thrombosis and postoperative malaise, 1111
 detachment, summary of modern opinions, *307
 detachment, temporary artificial indentation of sclera in operations for, 674
 detachment, treatment by means of diathermy and injection of air into vitreous, 1094
 detachment with cyst formation, report of case, 523
 determination and significance of scotopic retinal visibility curve, *713
 diagnostic significance of retinal changes in cases of hypertension, 140
 edema of, 514
 entoptic phenomena—intraretinal fluids, 119
 fusional movements, role of peripheral retinal stimuli, 873
 histologic changes in eyes of patients with hypertension, 138
 importance of ocular examination for understanding of hypertension and nephritis, 139
 Inflammation See Retinitis, Retinochoroiditis
 innervation, retinal myelin fibers, 332
 intraocular mass, 151
 isoperfusion and anisoperfusion of capillaries in cases of retinal hypertension, 140
 juvenile glaucoma with angiomatosis of, 665
 latent processes in visual pathways in cases of hypertension, 139
 measurements in fundus, 327
 ophthalmoscopic study of newborn infants, 144
 pathology, treatment and development of 2 cases of syndrome of Gronblad and Strandberg (angioid streaks in retina and pseudoxanthoma elasticum) 514
 physiology, procedure for measuring threshold sensibility of retina, 857
 recurrent retinal and vitreous hemorrhages in young—Eales' disease, report of 2 cases, *276
 resolving power of human retina, 144
 retinal arterial hypertension in cases of commotio cerebri, 138
 retinitis pigmentosa with "hole" in macula, report of case, *471
 significance of transitory arterial hypertension in genesis of retinal changes, 138
 spontaneous retinal pulsation, 140
 traumatic retinal anglopathy, 1107
 tumors, von Hippel's disease, report of case, 682

- Retina—Continued
 visual cells of Platypus (*Ornithorhynchus*), 661
- Retinitis cerebrospinal pressure in cases of, 140
 exudative, pathologic and clinical reports of 3 cases of, 120
 hereditary choriorretinal disease, 865
 Jensen's See Retinochoroiditis
 Pigmentosa See also Laurence-Biedl Syndrome
 pigmentosa and internal secretion, treatment with estrogen, 112
 pigmentosa, late results from resection of cervical portion of sympathetic trunk in cases of, 155
 pigmentosa, some pathogenic factors in, 120
 pigmentosa, unilateral, report of 2 cases, 680
 pigmentosa with glaucoma, 691
 pigmentosa with 'hole' in macula, report of case, *471
 proliferans, clinical and histologic studies, *427
 pseudo albuminuric, in cases of tumor of brain 332
- Retinochoroiditis radiata, 120
- Riboflavin See Vitamins, B
- Richards, P Nature of filtrable agent of trachoma, *569
- Richman F Acute membranous conjunctivitis in brass worker, 689
 Ophthalmoscopic study of newborn infants, 144
- Rickettsia bodies as cause of trachoma, 146
 problem of rickettsias in trachoma *16
- Roche, M Ophthalmologic aspects of case of chordoma, 127
- Roentgenotherapy See under names of organs regions and diseases
- Von Roth Causation of trachoma by virus 146
- Rosenthal B C von Hippel's disease, report of case, 682
- Rosner, L Biochemistry of lens, effect of galactose on permeability of capsule of lens *80
 Biochemistry of lens, studies on glutathione in crystalline lens *417
- Rosnoblet Blindness in infants, without ophthalmoscopic changes (congenital forms), 133
- Rubegni Blood calcium in cases of juvenile cataract 144
 Use of interferometer for estimation of endocrine function in ocular diseases, 142
- Sabbadini Autonomic hypertension, 138
- Sachs-Tays Disease See Idiocy, amaurotic
- Sadeh Discussion of national policy to be adopted in tropical country for prevention of blindness, 147
- Sallmann Diagnostic significance of retinal changes in cases of hypertension, 140
- Samarin Treatment of trachoma 147
- Samuels, B Panophthalmitis and sympathetic ophthalmia, *804
- Sarcoma See also Cancer, Melanosarcoma, Tumors, and under names of organs and regions
 intraocular tension in cases of sarcoma of choroid and ciliary body *359
 of optic papilla, report of case 677
 unilateral exophthalmos due to sclerosing sarcoma of anterior cranial fossa, 686
 uveal, topography and frequency of complications of, *290
- Schluek, K Suprasellar meningioma 337
- Schools, instruction and professional orientation of amblyopic children, creation of special schools, 1087
- Schools—Continued
 work in schools in Japan for children with defective sight, 147
- Schupfer Resolving power of human retina, 144
- de Schweinitz, George Edmund, 1074
- Sclera, differences in manner of perforation of sclera caused by primary melanosarcoma and metastatic carcinoma of uveal tract, 676
 fibroma of, 1095
 perforating scleromalacia, 109
 scleromalacia perforans, report of case in which eye was examined microscopically, *1013
 senile degeneration of sclera at insertion of ocular muscles, report of case 320
 three cases of anterior sclerouveitis, 2 (1 bilateral) of malignant type (progressive scleroperikeratitis of von Szily), clinical, histopathologic and bacteriologic study of 3 enucleated eyes, 661
- Sclerositis, tuberosus, tumor of lid in case of Bourneville-Pringle's disease, 864
- Scotoma, binocular fixation for scotometry at 1 meter, 1089
 homonymous hemianopic paracentral scotoma report of case, *846
- Scrofula See Tuberculosis
- Sea air and ophthalmology 135
- Secretions, Internal See Endocrine Glands
- Sedan, J Diplopia preceding marked increase of growth in dermolipoma of conjunctiva, 123
- Seech, S G Experimental iontophoresis of rabbits corneas, report of 2 cases of corneal dystrophy with treatment by ionic medication *624
- Senility See Old age
- Serotherapy and Hemotherapy, subconjunctival autohemotherapy in complications of trachoma, 863
 treatment of trachoma by local autoserotherapy, 675
 treatment of trichomatous pinnus by subconjunctival autohemotherapy, 516
- Serr Spontaneous retinal pulsation, 140
- Sezer, F N Accidental vaccination of conjunctiva, *89
- Shiada, D J Binocular vision and orthoptic procedure, *477
- Shannon, C E G Palsy of ocular muscles in case of toxic goiter, 335
- Sharp, G S Treatment of cancer of eyelids, 879
- Shumkin Antepositio conjunctivae fornicis in cases of severe spring catarrh, 143
 Use of auricular cartilage for correction of severe entropion following tarsectomy, 147
- Short Waves See under names of organs, regions and diseases, as Cornea, conical, Eyes diseases, etc
- Sinus, Cavernous See Cavernous Sinus
- Sinuses Nasal See also Sphenoid Sinus
 voluminous chondroma of left ethmoid region with mucocoeles in all sinuses and grave ocular syndrome, 1096
- Skull See Cranium
- Steeple See Acrocephaly
- Slendrets See under Obesity
- Sloan, L L Perimetric studies in syphilitic optic neuropathies, *201
- Smallpox, accidental vaccination of conjunctiva, *89
- Smith F New method for rebuilding lower lid 315

INDEX TO VOLUME 20

- Smith, J W Blepharitis comb, *658
 Subconjunctival cyst simulating prolapse of lacrimal gland, 338
- Smukler, M E Simplification of O'Connor cinch operation, 502
- Snapper Endocrinology and eye, 141
- Snell, A C Visual acuity, its relation to form sense and application of this relationship to medicolegal problems, 871
- Sobby Bey Treatment of trachoma, 147
- Social Service in ophthalmic hospital, 148
 role of social worker in program for prevention of blindness, 148
- Societies, Canadian Ophthalmological Society, meeting, 698
 foreign, directory of, 165, 349, 531, 699, 897, 1119
 formation of American Orthoptic Council, 883
 international, directory of, 165, 349, 531, 699, 897, 1119
 local, directory of, 169, 353, 535, 703, 901, 1123
 national, directory of, 166, 350, 532, 700, 898, 1120
 National Society for Prevention of Blindness, annual meeting, 1071
 ophthalmologic, directory of, 165, 349, 531, 699, 897, 1119
 Ophthalmological Society of Australia, organization of, 689
 sectional, directory of, 167, 351, 533, 701, 899, 1121
 Societe d'ophtalmologie de Paris, annual meeting, 883
 state, directory of, 168, 352, 534, 702, 900, 1122
- SOCIETY TRANSACTIONS
 American Medical Association, Section on Ophthalmology, 867
 American Ophthalmological Society, 1099
 College of Physicians of Philadelphia, Section on Ophthalmology, 152, 334
 French Ophthalmologic Society, 122
 International Association for Prevention of Blindness, 147
 International Congress of Ophthalmology, 135
 International Organization Against Trachoma, 146
 New York Academy of Medicine Section of Ophthalmology, 148, 337, 520, 680
- Sohr Graduation of Baillart and Sobrinski dynamometers on human cadaver, 143
- Sorbitol See under Glaucoma
- Space Perception, does time influence perception of depth? 678
 induced size effect, new phenomenon in binocular space perception associated with relative sizes of images of 2 eyes, *604
 new apparatus for recording depth perception, 678
- Spaeth, E B Etiology of retinal separation considered from standpoint of surgical correction, *1046
 External orbitotomy, 337
 Late results from resection of cervical portion of sympathetic trunk in cases of retinitis pigmentosa, 155
- Spalding, James A, 318
- Specimens, research studies of eye, some technical and practical notes, *395
- Spectacles See Glasses
- Sphenoid Bone, enormous bony tumor of outer wall of orbit, greater wing of sphenoid bone and temporal fossa, 134
- Sphenoid Sinus See also Sinuses, Nasal
 lesion of optic tract probably result of infected sphenoid sinuses, *966
- Spinal Fluid See Cerebrospinal Fluid
- Sporotrichosis, sporothrix granuloma of limbus, 660
- Spotnitz, H Preliminary report of value of functional visual tests for localization of supratentorial tumors of brain, 524
- Squint See Strabismus
- Stains and Staining, research studies of eye, some technical and practical notes, *395
- Staphylococci, mannitol fermentation as indicator of conjunctival pathogenicity of staphylococci, *274
 metastatic septic endophthalmitis with ring abscess of cornea, report of case with description of anatomic changes, 1102
 staphylococcal conjunctivitis, 108
 staphylococcal septicemia following suppurating meibomian cyst, report of case, 508
 treatment of staphylococcal conjunctivitis with staphylococcus toxoid, preliminary note, *271
- Steeple Skull See Acrocephaly
- Stiehler, R D Circulation of aqueous, mechanism of secretion of intraocular fluid, *761
- Stokes, W H Dacryocystitis in lymphatic leukemia, *85
- Strabismus See also Heterophoria
 binocular vision and orthoptic procedure, *477
 simplification of O'Connor cinch operation, 315, 502
 surgical treatment of, 103
 value of orthoptic training, 511
- Strandberg-Gronblad Syndrome See Pseudo-xanthoma elasticum, Retina, pathology
- Streptococci viridans, conjunctivitis associated with infection by, clinical and bacteriologic observations in epidemic, *19
- Sudranski, H F Evaluation of homatropine-benzedrine cycloplegia, *585
- Sulfanilamide, Therapy See under Trachoma
- Sulfides, superficial and deep-seated disciform keratitis in caisson workers after exposure to hydrogen sulfide, report of cases, 323
- Sullivan, N P Intraocular nematode worms, report of case and review of literature, *1006
- Surgery See also Apparatus, Instruments, and under organs and diseases, as Eyes, surgery, Glaucoma, etc
 plastic, advantages of use of coagulants in ocular operations especially in extraction of cataract and in plastic operations, *406
 plastic, rotation of cheek in ophthalmology, *410
- Swindle, P F Events of vascularization and devascularization seen in corneas, *974
- Symblepharon See under Eyelids
- Sympathectomy, late results from resection of cervical portion of sympathetic trunk in cases of retinitis pigmentosa, 155
- Syphilis See also under names of organs, regions and diseases, as Lacrimal Organs, etc
 congenital, traumatic parenchymatous keratitis in, 110
 information based on 349 cases of heredo-syphilitic keratitis, 109
 of lacrimal sac simulating acute dacryocystitis, 1088
 palpebral manifestations in delayed hereditary syphilis, 326
 perimetric studies in syphilitic optic neuropathies, *201
 syphilitic opticochiasmic arachnoiditis and pyretotherapy, 130
 syphilitic opticochiasmic arachnoiditis, 1103

INDEX TO VOLUME 20

- von Szily Dysfunction of thyroid gland and its relation to eye 141
- Szymanski J Selection of contact lenses by means of metallic prostheses, 122
- Tarbes Dorsalis relief therapy ("Entlastungstherapie") for tabetic optic atrophy, 865
- Tainter, M L Influence of vitamins and dinitrophenol on production of experimental cataract, *30
- Tarsectomy See under Eyelids
- Tissman, I S Use of paredrine in cycloplegia, 156, correction, 505
- Taumi Operative treatment of astigmatism, 143
- Try-Sachs' Disease See Idiocy, amaurotic
- Teeth, pathogenesis of ocular diseases of dental origin, 126
- thrombophlebitis of cavernous sinus, 330
- tooth and eye, 663
- Teissler Use of new type of contact glass made of pyroxilin, 145
- Temperature, ocular thermometry, 110
- Tendons, muscle weakening by central tenotomy, 856
- Tension See also Glaucoma
- behavior of intraocular pressure in acute intraocular vascular reactions 670
- clinical study of consensual ophthalmotonic reactions 118
- intraocular, action of yohimbine hydrochloride on, 125
- intraocular experimental study of influence of muscular work on, 119
- intraocular in cases of sarcoma of choroid and ciliary body *359
- Souter tonometer 665
- theory and practice of tonometry, 115
- Teratoma, congenital anophthalmos veiling ophthalmotomoid, report of case, 1082
- Terry, T L Modified corneal clamp to facilitate insertion of stitches, *91
- Tetany, diabetic and tetanic cataract in relation to Krauss theory of muscle function of cortical cells, 145
- latent and cataract 325
- parathyroid glands and eye 666
- treatment of tetany cataract, 143
- Theodore F H Hypersensitivity to Iodochrome *474
- Thiel Importance of ocular examination for understanding of hypertension and nephritis, 139
- Thrombosis orbital mechanics and limitations and movement of eye optic nerve oblique muscle and Mullers muscle with relation to exophthalmos separation of retina thrombosis and postoperative malaise 1111
- oscillometry of arteries of limbs in thrombosis of central retinal vein, 132
- thrombophlebitis of cavernous sinus 330
- Thygeson P Causation of trachoma by virus, 146
- Cultivation of human conjunctival and corneal epithelium in vitro 1107
- Mannitol fermentation as indicator of conjunctival pathogenicity of staphylococci *274
- Nature of filtrable agent of trachoma *569
- Problem of rickettsias in trachoma, *16
- Treatment of staphylococcal conjunctivitis with staphylococcus toxoid, preliminary note, *271
- Thyroid See also Goiter
- dysfunction and its relation to eye 141
- Tille H Histologic and clinical statistics on 80 cases of tumor of bulbar conjunctiva 123
- Tissue, inositol in ocular tissues, *299
- metabolism of lens after parathyroidectomy, 113
- Tomography See under Orbit
- Tonometry See Tension
- Tooke, T T Aleukemic lymphosis involving upper lids, pathologic changes 1110
- Tooker, C W Metastatic septic endophthalmitis with ring abscess of cornea, report of case with description of anatomic changes, 1102
- Toulant Prevention of blindness in tropical country, 147
- Trachoma, acute, 12 cases, 147
- allergic reaction of, 675
- and pyrexia, 676
- causation by virus 146
- development in cornea 147
- formation of follicles in mucous membrane graft applied by Denig technic for pannus, 147
- histologic picture and pathogenesis of 147
- microstructure of epithelial cells and its importance for etiology of 146, 862
- nature of filtrable agent of, *569
- pathologic picture of, 147
- problem of rickettsias in, *16
- rickettsia bodies as cause of, 146
- so-called paratrachoma a virus disease due to Halberstadter-Provazek virus 146
- studies on infectivity of, further observations on filterability of infectious agent 1095
- studies on pathogeny of, 863
- subconjunctival autohemotherapy in complications of 863
- sulfanilamide treatment, preliminary report, 880
- treatment of, 147
- treatment according to recent experimental research 124
- treatment by local autoserotherapy 675
- treatment of trachomatous pannus by subconjunctival autohemotherapy 516
- treatment with trichozid 516
- Trichozid See under Trachoma
- Trantas N Diathermocautulation (Weve) for detachment of retina, retinal tears without detachment 132
- Trauma detachment of retina and accidents 860
- traumatic retinal angiopathy, 1107
- Troncoso Anatomic structure and evolution of iridocorneal angle in mammals 144
- Tropics discussion of national policy to be adopted in tropical country for prevention of blindness, 147
- prevention of blindness in tropical country 147
- Tryparamide pathologic changes in amblyopia following tryparamide therapy, 867
- Tuberculosis See also under special structures of eye and names of diseases
- lymphoid reaction of bulbar conjunctiva reaction to tuberculin, 319
- resistance of capillaries to pressure in ocular tuberculosis 126
- specific diagnosis of tuberculous diseases of eyes, report of cases 1086
- Tularemia from clinical and from ophthalmologic standpoint, 1085
- oculoglandular, *88
- Tumors See also Angioma Chondroma Epithelioma, Fibroma Melanoma Meningioma Sarcoma, Teratoma etc., and under special structures of eye as Brain Conjunctiva Eyelids, Lacrimal Organs, Orbit, Retina etc
- mixed cell tumor of lacrimal sac, 1101
- Twins esophoria and anaphoria in twins of same sex, 330

INDEX TO VOLUME 20

- Ulcers, Mooren's See under Cornea
 Ultrashort Waves See under names of organs, regions and diseases, as Cornea conical, Eyes, diseases, Retina, detachment etc
 Urea, chemical and histochemical investigation of urea in lens and ocular membranes, 1082
 Uvea chronic cellular infiltration of uvea in septic endophthalmitis of ectogenous origin, 865
 circulation of aqueous, mechanism of secretion of intraocular fluid, *761
 differences in manner of perforation of sclera caused by primary melanoma and metastatic carcinoma of uveal tract, 676
 effect of extract of ciliary body on blood pressure, 512
 effect of extract of ciliary body on isolated frog heart, 512
 Hirada's disease, 861
 Inflammation See also Ophthalmia, sympathetic
 inflammation, blood transfusion in uveitis 677
 intraocular tension in cases of sarcoma of choroid and ciliary body, *359
 three cases of anterior sclerouveitis 2 (1 bilateral) of malignant type (progressive scleroperikeratitis of von Szily), clinical histopathologic and bacteriologic study of 3 enucleated eyes, 661
 topography and frequency of complications of uveal sarcoma, *290
 Uveitis See Uvea, inflammation
 Vaccinia See Smallpox
 Vail D Optochiasmic arachnoiditis, importance of mixed type of atrophy of optic nerve as diagnostic sign, *384
 Syphilitic optochiasmic arachnoiditis 1103
 Valeri Cholesterol metabolism of patients with retinal lesions, 144
 Van Lint Resistance of capillaries to pressure in ocular tuberculosis, 126
 Veins See also Thrombosis, etc
 anomalous retinal vein, 683
 Velhagen Examination of eye 143
 Verhoeff, F H Scleromalacia perforans report of case in which eye was examined microscopically, *1013
 Villard, H Our distinguished colleague Pope John XXI, 122
 Viruses, nature of filtrable agent of trachoma, *569
 studies on infectivity of trachoma further observations on filterability of infectious agent, 1095
 Vision See also Accommodation and Refraction Blindness, Eyes, examination etc
 binocular, and orthoptic procedure, *477
 binocular fixation for scotometry at 1 meter 1089
 children with defective vision in need or not of sight-saving class, 1087
 Color See Color Blindness
 condemnation of color indexes in clinical perimetry, 115
 induced size effect, new phenomenon in binocular space perception associated with relative sizes of images of 2 eyes, *604
 light reserve for occupations in sight-saving classes, 1087
 perimetric studies in syphilitic optic neuropathies, *201
 physiologic and clinical ophthalmologic problems in relation to individual variability *541, *726, *913
 physiology, so-called refusion, 1091
 Vision—Continued
 reading difficulties in children, *829
 relation of sensory visual cortex to oculomotor cortex, 510
 studies of visual fields in cases of verified tumor of brain, *437
 subjective study of visual aberrations 1100
 testing fitness for night flying visual acuity, *58
 tests, new test chart to determine visual acuity, 678
 tests, value of functional visual tests for localization of supratentorial tumors of brain preliminary report 524
 visual acuity, its relation to form sense and application of this relationship to medico-legal problems, 871
 visual-acuity survey, 1096
 work in schools in Japan for children with defective sight, 147
 Vitamins, A, study of diet in relation to health, dark adaptation as index of adequate vitamin A intake, 512
 B, arrest of nutritional cataract by use of riboflavin, 507
 C, action on human cataract 142
 C, property of galactose cataracts to protect vitamin C from oxidation, 506
 D complex in keratoconus, etiology, pathology and treatment of conical cornea, preliminary report, 852
 influence of vitamins and dinitrophenol on production of experimental cataract *30
 Vitreous Humor, antigenic properties of cornea and vitreous, 850
 clinical study of, 144
 congenital buphthalmos complicated by dislocation of lens and hemorrhage into vitreous with complete recovery of central vision *757
 delayed removal of magnetic foreign bodies 152
 recurrent retinal and vitreous hemorrhages in young—Eales' disease, report of 2 cases *276
 Volhard Importance of ocular examination for understanding of hypertension and nephritis 139
 Wagener H P Arterial hypertension of retina, 137
 Walker B F Specific treatment for herpes zoster *304
 Walker, C B Orbital mechanics and limitations and movement of eye optic nerve, oblique muscle and Muller's muscle with relation to exophthalmos, separation of retina, thrombosis and postoperative malaise 1111
 Walker, J R Specific treatment for herpes zoster, *304
 Weekers, R Inositol in ocular tissues, *299
 Weeks, W W Ocular hypertelorism, report of case 683
 Weve Spread of blindness in Dutch East Indies, 147
 Wexler, D Coloboma of optic nerve and of macula, microscopic study, *787
 Whirlman, H F Vernal conjunctivitis 877
 Wheeler, John Martin 885
 Correction of entropion by transplantation of orbicularis muscle of lower eyelid 1104
 Implantation of hollow grooved body into orbit for filling late after enucleation of eyeball *709
 Wheeler M C Malignant neoplasm of eyelid, report of 2 cases, 682
 Wibaut Retinitis pigmentosa and internal secretion, treatment with estrogen 142

INDEX TO VOLUME 20

- Wiechmann Effect of pH on miotics and mydriatics, 145
- Wilson Pathologic picture of trachoma, 147
- Wolfe, O Surgical treatment of strabismus 105
- Wood, C George Edmund de Schweinitz 1074
- Woods, A C Perimetric studies in syphilitic optic neuropathies, *201
- Yohimbine hydrochloride, action on intraocular tension, 125
- Zeiss Apparatus for measuring and recording disturbances of ocular motility 145
- Zentmayer, W Association of annular band of pigment on posterior capsule of lens with Krukenberg spindle *52
- Zona See Herpes zoster

